WASHINGTON STATE BOARD OF HEALTH

Notice of Public Meeting

Wednesday, March 13, 2024, 9:00 a.m. – 5:05 p.m.
Physical meeting location:
Swinomish Casino and Lodge
12885 Casino Dr, Anacortes, WA 98221
WA Walton Conference Room
Virtual meeting: ZOOM Webinar
(hyperlink provided below)
Language interpretation available

Final Agenda

Time	Agenda Item	Speaker
9:00 a.m.	Call to Order & Introductions	Patty Hayes, Board Chair
	Swinomish Land Acknowledgement and Welcome	Swinomish Tribal Leader Chairman Edwards or Designee
9:15 a.m.	1. Approval of Agenda – Possible Action	Patty Hayes, Board Chair
9:20 a.m.	2. Approval of January 10, 2024, Minutes – Possible Action	Patty Hayes, Board Chair
9:25 a.m.	3. Public Comment	Please note: Verbal public comment may be limited so that the Board can consider all agenda items. The Chair may limit each speaker's time based on the number people signed up to comment.
9:45 a.m.	4. Announcements and Board Business	Michelle Davis, Board Executive Director
10:05 a.m.	5. Newborn Screening Annual Report	Kelly Oshiro, Board Vice Chair John Thompson, Department of Health Anna Howard, Department of Health
10:55 a.m.	Break	
11:10 a.m.	6. Request for Delegated Rulemaking – On-Site Sewage Systems, 246-272A-110 WAC – Possible Action	Kate Dean, Board Member Andrew Kamali, Board Staff Roger Parker, Department of Health

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11:25 a.m.	7. Swinomish Tribe • Jennifer La Pointe, SITC General Manager • Dr. Rachael Hogan, Swinomish Dental Director • Dr. Cheyanne Warren, dəxʷx̌ayəbus-DT Education Program Director • Beverly Keyes, didgʷálič Wellness Center Director	Mindy Flores, Board Member Swinomish Indian Tribal Community (SITC) Skagit Valley College (SVC) didgʷálič Wellness Center
12:10 p.m.	Lunch	
1:20 p.m.	8. Pro Equity Anti Racism (PEAR) Plan	Paj Nandi, Board Member Ashley Bell, Board Staff
2:05 p.m.	Break	
2:15 p.m.	9. State Health Report Community Panel • Amanda Shi, Manager of Research and Evaluation, Tubman Center for Health and Freedom • Dominique Horn, Community Mobilization Coordinator, Southwest Washington Accountable Community of Health • Molly Parker, Family Health Provider and Chief Medical Officer for Population Health, Jefferson Healthcare • Nyka Osteen, Director of Innovation, North Sound Accountable Community of Health	Mindy Flores, Board Member Molly Dinardo, Board Staff Hannah Haag, Board Staff

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Time	Agenda Item	Speaker
3:45 p.m.	Break	
4:00 p.m.	10. Petition for Rulemaking - WAC 246-290-220, Group A Water Systems – Drinking Water Materials and Additives - Possible Action	Patty Hayes, Board Chair Andrew Kamali, Board Staff Shelley Guinn, Department of Health Mike Means, Department of Health
4:35 p.m.	11. 2024 Board Meeting Schedule Review	Michelle Davis, Board Executive Director
4:45 p.m.	12. Board Member Comments and Updates	
5:05 p.m.	Adjournment	

- **To access the meeting online and to register:**
  https://us02web.zoom.us/webinar/register/WN_Er7t6leERpGyne-2jw4-Ag
  You can also dial-in using your phone for listen-only mode:
  Call in: +1 (253) 215-8782 (not toll-free)
  Webinar ID: 886 3024 9315
  Passcode: 682856

Important Meeting Information to Know:

- Times are estimates only. We reserve the right to alter the order of the agenda.
- Every effort will be made to provide Spanish interpretation, American Sign Language (ASL), and/or Communication Access Real-time Transcription (CART) services. Should you need confirmation of these services, please email wsboh@sboh.wa.gov in advance of the meeting date.
- If you would like meeting materials in an alternate format or a different language, or if you are a person living with a disability and need reasonable modification, please contact the State Board of Health at (360) 236-4110 or by email

wsboh@sboh.wa.gov. Please make your request as soon as possible to help us meet your needs. Some requests may take longer than two weeks to fulfill. TTY users can dial 711.

Information About Giving Verbal Public Comment at Hybrid Meetings:

- For the public attending in-person: If you would like to provide public comment, please write your name on the sign-in sheet before the public comment period begins. We strongly encourage people to sign up with the Board by sending an email by 12:00 Noon the last business day before the meeting to: wsboh@sboh.wa.gov. As this is a business meeting of the Board, time available for public comment is limited (typically 2 to 4 minutes per person). The Chair will call on those who have signed up to speak to the Board, first. The amount of time allotted to each person will depend on the number of speakers present. If time remains, those who have not signed up ahead of time to speak to the Board will be called on to speak until the scheduled time for Public Comment comes to an end.
- For the public attending virtually: If you would like to provide public comment, please sign up through the Zoom webinar link by 12:00 Noon, the last business day before the meeting. Your name will be called when it's your turn to comment.

Information About Giving Written Public Comment:

- Please visit the Board's Meeting Information webpage for details on how to provide written public comment.

WASHINGTON STATE BOARD OF HEALTH

Draft Minutes of the State Board of Health
January 10, 2024
Hybrid Meeting
ASL (or CART) and Spanish interpretation available
Washington State Department of Health
111 Israel Road S.E., Tumwater, WA 98501
Building: Town Center 2, Rooms 166 & 167
Virtual meeting: ZOOM Webinar

State Board of Health members present:
Patty Hayes, RN, MSN, Chair
Kelly Oshiro, JD, Vice Chair
Stephen Kutz, BSN, MPH
Kate Dean, MPA
Socia Love-Thurman, MD
Mindy M. Flores, MBA-HCM
Dimyana Abdelmalek, MD, MPH
Tao Sheng Kwan-Gett, MD, MPH, Secretary's Designee

State Board of Health members absent:
Umair A. Shah, MD, MPH

State Board of Health staff present:
Michelle Davis, Executive Director
Melanie Hisaw, Executive Assistant
Michelle Larson, Communications Manager
Anna Burns, Communications Consultant
Molly Dinardo, Health Policy Advisor
Andrew Kamali, Health Policy Advisor
Jo-Ann Huynh, Administrative Assistant
LinhPhụng Huỳnh, Council Manager
Lilia Lopez, Assistant Attorney General
Ashley Bell, Equity & Engagement Manager
Hannah Haag, Community Engagement Coordinator

Guests and other participants:
Kelly Cooper, Department of Health
Amy Ferris, Department of Health
Vicki Lowe, American Indian Health Commission
Jaime Bodden, Washington Association of Local Public Health Officials
David DeLong, Department of Health
Jeremy Simmons, Department of Health

Patty Hayes, Board Chair, called the public meeting to order at 8:36 a.m. and read from a prepared statement (on file).

1. **APPROVAL OF AGENDA**
   Motion: Approve January 10, 2024, agenda.
   Motion/Second: Vice Chair Oshiro/Member Dean. Approved unanimously.

2. **ADOPTION OF NOVEMBER 8, 2023, MEETING MINUTES**
   Motion: Approve the November 8, 2023, minutes.
   Motion/Second: Member Love-Thurman /Vice Chair Oshiro. Approved unanimously**.**

3. **PUBLIC COMMENT**
   Patty Hayes, Board Chair opened the meeting for public comment and read from a prepared statement (on file).

   Gerald Braude, Jefferson County, commented on the harm from the COVID-19 shots. G. Braude said there are 11 more deaths from COVID-19 shots since the last Board meeting in November, increasing from 222 to 233 deaths. G. Braude gave examples of people from ages 16 to 65 who died from arterial fibrillation and acute aortic dissection after receiving the COVID-19 shot.

   Bill Osmunson, a dentist with a master's in public health, talked about the authority of the Board for dental regulations and the dangers of fluoride. B. Osmunson said the Environment Protection Agency National Toxicology Association states that fluoride lowers infant IQ. B. Osmunson talked about developmental neurotoxicity and mortality issues from fluoride in infants and children.

   Natalie Chavez commented on the harm from the COVID-19 vaccine and asked for experimental vaccines to be put on hold until more research is done. N. Chavez gave several examples of people harmed and said that many have suffered physical, emotional, and financial devastation from the vaccine.

   Lisa Templeton talked about concerns on certain legislative bills, including House Bill (HB) 2157. L. Templeton said there are already dozens of shots on the Centers for Disease Control (CDC) pediatric schedule and talked about the dangers and costs of vaccines.

4. **BOARD ANNOUNCEMENTS AND OTHER BUSINESS**
   Michelle Davis, Board Executive Director welcomed Board Members. Executive Director Davis recognized new Board Member Paj Nandi and Ashley Bell the Board's new Equity and Engagement Manager. Executive Director Davis announced Shay Bauman, the Board's new Policy Advisor, would join the team on February 1.

   Executive Director Davis described the materials in the packet. Executive Director Davis discussed the letter from the Office of Financial Management (OFM) acknowledging the Board's letter of support for the Environmental Justice Council's (EJC) recommendations related to school environmental justice. Executive Director Davis noted that the Governor's budget described additional funding through the Climate Commitment Act that reflects part of the EJCs recommendation.

Executive Director Davis shared additional information related to the Governor's budget, which was released in released in December. It maintained the budget proviso that suspends the Board's school environmental health and safety rules. The Governor's budget provided additional funding for schools:

- Capital, Sec 5007: $20,000,000 (Climate Commitment Account) and $20,000,000 (Common School Construction Fund) for Equitable Access to Clean Air and Improving Classroom Air Quality.

Executive Director Davis noted other investments for local school districts, these investments include:

- Capital Two-Year
    - School Construction Assistance Program (SCAP). Increase in the construction cost allocation from $272/SF to $350/SF for fiscal year (FY) 2025 – funds state matching grants for local school districts.
    - $176,867,000 for the Small District and Tribal Compact Schools Modernization Program.
    - An additional $8,100,000 for School District Health and Safety.
    - An additional $1,500,000 for Healthy Kids-Healthy Schools competitive grant programs.

Executive Director Davis described the remaining materials in the Board packet (materials on file). Executive Director Davis thanked Member Dean for serving as the chair of the Environmental Health Subcommittee and Member Flores for sponsoring the 2024 State Health Report.

Executive Director Davis said the Health Impact Review (HIR) team recently completed two HIRs for Engrossed Substitute House Bill 1589 (Natural Gas) and 1859 (Long-Term Care Residents). Executive Director Davis said the HIR team has started receiving requests for the 2024 legislative session and is currently working on updates to HIRs completed in the 2023 legislative session. These HIRs include Senate House Bill 1368 (Zero-emission school buses) and Senate Bill 5002 (Alcohol concentration). Executive Director Davis reminded Board Members of the short completion deadline for HIRs and of the notification they will receive. Executive Director Davis asked Board Members to reach out with any recommended resources or connections.

Executive Director Davis thanked panelists for participating in the meeting.

Kate Dean, Board Member, asked for clarification on the Climate Commitment Act, and if it was earmarked for a particular use. Executive Director Davis said it pertains to indoor air quality, equitable access to clean air, and improving classroom air quality.

Member Dean asked about the $20 million Common School Construction fund. Executive Director Davis offered to follow up on the types of schools eligible for those funds and the $20 million is part of the Office of Superintendent and Public Instruction (OSPI) request.

Member Dean asked about Capital Gains. Executive Director Davis said there would be more conversations throughout the session. Executive Director Davis reminded the

Board that the budgets begin with the Governor's proposal, then the Senate and House negotiations before a final budget is reached.

Steve Kutz, Board Member, asked about the Governor's budget and schools. Executive Director Davis said the Governor's budget includes the suspension of the school and environmental health and safety rules, but the legislature has had several work sessions and focuses on school infrastructure.

5. 2024 LEGISLATIVE SESSION PREVIEW

Patty Hayes, Board Chair invited the Board's partners at the Department of Health (Department), the Washington State Association of Local Public Health Officials (WSALPHO), and the American Indian Health Commission (AIHC) to share their agency's priorities for the legislative session.

Kelly Cooper, Policy and Legislative Relations Director, Department of Health, shared the Department's legislative priorities. Kelly shared the Department's three pieces of agency request legislation, Senate Bill (SB) 5271 and House Bill (HB) 1434, SB 5982 and HB 2157, and SB 6095.

Amy Ferris, Chief Financial Officer, Department of Health, discussed the Department's budget priorities. Amy said the Department would be focusing on the healthcare workforce, especially for rural and behavioral health, the opioid epidemic, and emergency response management. Amy said the Department would also be supporting public health infrastructure, such as the 988 Suicide and Crisis Lifeline, and internal technology infrastructure supporting public health work.

Jaime Bodden, Managing Director, Washington Association of Local Public Health Officials (WSALPHO), gave a brief introduction of WSALPHO before discussing its priorities for the 2024 legislative session (presentation on file). Jaime said the agency has three policy focuses, SB 5982 and HB 2157, SB 6110, and SB 5983. Jaime then shared WSALPHO's budget priorities around on-site septic systems and school environmental justice.

Vicki Lowe, Executive Director, American Indian Health Commission (AIHC), introduced the AIHC. Vicki discussed AIHC's focus on the opioid and fentanyl epidemic in Indian Country and gave context about the first Washington State Tribal Opioid and Fentanyl Summit in May 2023. Vicki shared AIHC policy priorities, including HB 1877 and 2075. Vicki then spoke about AIHC's budget priorities. Vicki said these priorities are to establish a Tribal Opioid and Fentanyl Response Task Force and to establish Tribally operated facilities addressing opioid and fentanyl use. Vicki said the priorities also include bolstering education and prevention through developing the For Our Native Lives campaign, Tribal prevention models, and school-based prevention programming. Vicki said another priority is to streamline building a Tribally operated crisis stabilization and inpatient facility. Vicki said AIHC was also supporting several Tribal positions as well.

Chair Hayes transitioned the Board to the question-and-answer period.

Chair Hayes thanked Vicki for their presence at the meeting. Chair Hayes said the Board wishes to recognize AIHC's work. Chair Hayes said that the Board will discuss its 2024 Legislative Statement today, in which there are references of support to the AIHC's policy priorities. Chair Hayes also wanted to acknowledge Board Member Steve Kutz's work advising the 2024 Legislative Statement as well. Vicki thanked Chair Hayes for the comments.

Chair Hayes then addressed Jaime. Chair Hayes stated excitement for WSALPHO's work around syphilis treatment and asked Jaime to speak more about SB 5983 and its proposed amendments and funding sources. Jaime shared that the policy recommendation came from a 2022 STI/HPV workgroup which consisted of Department experts, local public health, reproductive healthcare partners, and providers. Jaime said that this group made several policy recommendations, some without budget impacts and that this was one of them. Jaime said this group targeted syphilis because of its significant health impact on adults and infants with congenital syphilis. Jaime said that this legislation sought to amend the revised code of Washington (RCW) regarding the licensing of medical assistants, to allow for the temporary authority to administer treatment under telehealth supervision. Jaime spoke about the need for this treatment pathway due to significant spikes in syphilis cases. Jaime said this legislation would create more efficient avenues for treatment and shared an example from a King County program that performs health outreach at encampments.

Chair Hayes then asked Amy to confirm whether funding for Foundational Public Health Services (FPHS) was maintained in the Governor's Supplemental Budget. Amy confirmed that this funding was maintained.

Kate Dean, Board Member, raised the topic of HB 2070 (integrating environmental justice considerations into certain project decisions). Member Dean said this bill tasks local health jurisdictions to create an environmental justice impact report for any projects associated with the State Environmental Policy Act (SEPA). Member Dean asked the speakers whether they knew about the feasibility of or funding availability for this component of the bill. Jaime said that WSALPHO would be contributing a fiscal note. Jaime said that the assessment components of the bill may be related to FPHS activities. Such as WSALPHO's climate change core group, in which local health jurisdictions and Department staff are working on building a climate and health program. Jaime said these FPHS activities may help mitigate the costs to local health jurisdictions. Jaime said that several local health jurisdictions are leaning into environmental justice work and spoke about Tacoma-Pierce County as an example. Kelly said that in the past, there have been attempts to add environmental health components to SEPA reviews and is glad the conversation is coming up again.

Member Dean asked whether there are efforts to clarify rules around local boards of health aside from HB 2090 and SB 5970 (modifying local board of health county commissioner membership). Member Dean said Jefferson County is still unable to get a Tribal representative on their Board and is concerned with non-compliance. Jaime added context around HB 20290 and SB 5970, which was brought by Thurston County Public Health. Jaime said that WSALPHO is currently gathering information about other needed rule changes, such as how the Public Health Advisory Board currently excludes some of the largest local health jurisdictions. Jaime said that currently, WSALPHO is not

bringing forth any edits but will likely do so in the future. Vicki noted to Member Dean that Clallam County is not out of compliance as the state can't direct Tribal Members to hold seats on local boards of health. Vicki spoke about a recent meeting with Candice Wilson, Tribal Policy Director at the Department, and established a goal of increasing Tribal representation on Local Boards of Health. Vicki hopes to see Tribes receiving funding for the first time to do public health through FPHS. Vicki said that as Tribal public health offices get staffed, hopefully, they will be able to fill empty seats on local boards of health.

Socia Love-Thurman, Board Member, was excited to hear about partners' work regarding behavioral health licensure. Member Love-Thurman spoke about the Seattle Indian Health Board's (SIHB) effort to open a treatment bed facility to address the opioid and fentanyl epidemic in Indian Country. Member Love-Thurman shared their clinic's daily encounters with this issue, such as having to administer CPR on the sidewalk outside of the clinic. Member Love-Thurman asked Vicki if there had been discussion about reimbursements for traditional medicine practitioners in their work at AIHC. Vicki said that this issue is often discussed. Vicki said that there are currently no specific bills regarding this topic. It is a continued topic of discussion with agencies and legislators, and the AIHC is currently focusing on Medicaid transformation with the Health Care Authority (HCA) to tackle this issue. Vicki said there was an effort to pass a compensated care State Plan Amendment in 2012 or 2013, but it didn't go through. Vicki noted that some Tribes are hesitant to participate in the reimbursement mechanism as they view it as capitalistic and culturally inappropriate. Vicki said that the second Washington State Tribal Opioid and Fentanyl Summit would be hosted in spring 2024, focusing on treatment and trauma-informed care. Member Kutz said that the reimbursement issue is at the federal level and that conversations at the state level have been supportive.

Member Love-Thurman asked Jaime whether Tribal Members are a part of childhood mortality review teams. Jaime said it depends on the circumstances. Jaime said that if a child comes from a Tribal or immigrant community, then a representative from that community is involved. Jaime said that the review team coordinator is tasked with identifying the proper participants.

6. WATER RECREATION PETITION WAC 246-260 UPDATE

Patty Hayes, Board Chair, said the Board had received a petition for rulemaking in 2023 relating to barrier latch height for recreation pool facilities (memo on file). Chair Hayes invited presenters to provide an update on how the petition has been incorporated in ongoing rulemaking for Chapter 246-260 Washington administrative code (WAC). Andrew Kamali, Board staff, and David DeLong, Department of Health, provided information on the underlying issue in the rule related to the petition request, actions taken by staff, and proposed rule language (presentation on file).

Chair Hayes asked presenters for a reminder of the timeline for revisions to Chapter 246-260 WAC. Andrew said it is tough to answer since two processes are happening concurrently: (1) agency request legislation to revise the revised code of Washington (RCW) and (2) rulemaking to update chapter 246-260 WAC. Andrew added that processes will likely continue through 2024 and possibly into Summer 2025.

Michelle Davis, Board Executive Director, asked if local health has worked with the specific facility referenced in the rule petition to address the immediate issue noted by the petitioner. David confirmed that this had happened and said the facility needed to install a latch with the original approved condition, which was a lower-height latch to be compliant with the Americans with Disabilities Act (ADA).

Kelly Oshiro, Vice Chair asked if the proposed rule language about latches operated using a key, electronic opener, or combination lock reflects requirements in the Model Aquatic Health Code (MAHC) and the ADA. David confirmed that the proposed rule language would be compliant with the ADA. For latch-height requirements under the MAHC, David said there is a distinction between doors and gates. Vice Chair Oshiro expressed gratitude for the staff's mindfulness. Vice Chair Oshiro said the Board wants to ensure accessibility, safety for children, and compliance with various codes affecting a water recreation facility. Stephen Kutz, Board Member asked whether staff intend to do rulemaking to address these issues in the rule. Andrew confirmed that these issues are being addressed in the ongoing rule making for chapter 246-260 WAC.

Paj Nandi, Board Member asked whether staff are inviting additional disability community partners and advocates to the rule making process, how participants are responding to the efforts, and how this rule making interacts with similar national efforts. David said that a specific constituency is outlined in the rule and staff have invited those groups as well as additional parties. David said rule making is a public process and staff are doing their due diligence to engage communities who may be impacted by changes.

Kate Dean, Board Member asked staff to speak about the rule's alignment with the international building code, which may have different language on the same issue. David said that the Washington State Building Code Council (SBCC) has the authority to develop rules affecting residential swimming pools and the International Swimming Pool and Spa Code applies to residential swimming pools in Washington. David said state law obligates the Board and Department of Health (Department) to look at the MAHC, so staff are modeling proposed rule (chapter 246-260 WAC) on this code. Andrew added that there is SBCC staff serving on the technical advisory committee for this rule making to provide information on how Board rules and SBCC rules interact.

Chair Hayes thanked presenters for the update.

7. 2024 STATE HEALTH REPORT

Patty Hayes, Board Chair, invited Member Flores to introduce the topic. Mindy Flores, Board Member, said Washington law requires the Board to submit a report to the Governor's Office every two years to identify public health priorities and legislative action for the following biennium. Member Flores said the Board's next State Health Report is due by July 2024 and outlined the purpose of the report and introduced staff to discuss the topic further.

Molly Dinardo and Hannah Haag, Board staff, spoke about the report planning and development process, including possible topic areas for the 2024 State Health Report (presentation on file). Molly said the development process will be like the process used

in 2022, it will be iterative and will incorporate more community engagement. Hannah shared the goal and plans for engaging communities. This includes remaining flexible on recommendation areas as the Board actively listens to communities' priorities and listening to voices from communities who are overburdened or disproportionately impacted by health inequities.

Chair Hayes thanked Member Flores for leadership on this project. Chair Hayes asked Jaime Bodden from the Washington Association of Local Public Health Officials (WSALPHO), who was sitting in the audience, to keep the Board's efforts in mind since some local communities have completed community health assessments, which could help inform the Board's report.

Dimyana Abdelmalek, Board Member, expressed gratitude for including community voices in the report development and excitement for the community storytelling panel scheduled for the March Board meeting. Member Abdelmalek offered to share community health assessments from their local jurisdiction.

Paj Nandi, Board Member expressed gratitude and noted the challenges of a short project timeline. Member Nandi asked how staff are prioritizing communities, given the Board's commitment to using an anti-racist lens, as well as their plan for coordinating with partners so as not to duplicate efforts. Hannah said staff have a plan on how to bring folks from overburdened communities into the process. Hannah said staff will engage partners who have an established relationship with the Board, and staff will ask partners whom else staff should speak with. Hannah added that staff are very open to hearing Board Members' ideas about specific groups, sectors, and methods.

Stephen Kutz, Board Member noted some challenges people face when trying to attend a public meeting, such as transportation, weather, and geographic location. Member Kutz asked how the Board can ensure engagement with people in Central and Eastern Washington and said it may take more than one meeting to do this. Molly affirmed Member Kutz's considerations and said staff want to set up a strong foundation for future Board processes and products.

Kelly Oshiro, Vice Chair said it is nice to see continuity between past State Health Report topics and the proposed 2024 topics. Vice Chair Oshiro said it may be beneficial to receive a briefing on the topic of pregnant person health and mortality prevention since it seems the Board is addressing this topic for the first time. Vice Chair Oshiro noted that it would be helpful as a Board Member to get an additional briefing on the accomplishments and achievements on topics the Board is carrying over from past reports.

Kate Dean, Board Member shared an interest in having robust community participation in report development. Member Dean reflected on experience serving on a local board of health and noted that the use of jargon and the creation of an echo chamber can happen when people work in silos. Member Dean said care should be given to how concepts are talked about in community settings, such as buying healthy food, various ways substance use disorder is spoken about, and recognizing that the ability to exercise is a privilege. Molly said the Governor's Office released an executive order on

plain talking, and staff will incorporate that order as well as Board Members' reminders in their efforts.

The Board took a break at 10:45 a.m. and reconvened at 10:55 a.m.

8. INDOOR AIR QUALITY PANEL

Patty Hayes, Board Chair, briefly introduced the topic and the panel. Chair Hayes said that the COVID-19 pandemic highlighted the importance of indoor air quality (IAQ) to reduce the spread of respiratory illness. Chair Hayes said that most exposure to illness happens indoors, as most people in the United States spend 90 percent of their time indoors. Chair Hayes said the Board needs a robust understanding to make decisions impacting people in the state.

Andrew Kamali, Board staff, described materials, discussed the structure of the panel, and shared a brief biography for each panelist (materials on file).

Eric Vander Mey, Delta E Consulting, gave a presentation about IAQ and mechanical heating, ventilation, and air conditioning (HVAC) system design impacts due to the COVID-19 pandemic. Eric also presented on recent changes to the Washington State Energy code, the Washington State Clean Buildings Act and Seattle Building Emissions Performance Standard, and sustainability standards (presentation on file).

Brandon Kemperman, Public Health - Seattle King County, gave a presentation on the importance of IAQ, lessons learned around IAQ in Washington from the COVID-19 pandemic, and topics of importance. Brandon also presented future needs around IAQ work and Public Health – Seattle & King County's IAQ programs (presentation on file).

Nancy Bernard, Department of Health, gave a presentation about the history of the Department of Health (Department) work in school environmental health and safety, its current work, and lessons learned from the COVID-19 pandemic. Nancy also summarized the Department's standards and guidance around ventilation (presentation on file).

Ben Omura, State Building Code Council, gave a presentation about the State Building Code Council (SBCC) and its work. Ben also presented on the 2021 code cycle, which will come into effect on March 15, 2024, and topics of concern for the 2024 code cycle (presentation on file).

Erin McTigue, Environmental Protection Agency Region 10, gave a presentation about the Environmental Protection Agency (EPA) role in indoor air work and its focus areas. Eric discussed issues around housing and health disparities, Tribal and rural communities, children's environmental health, climate change impacts, and infectious disease. Erin noted there are very few regulations related to indoor air quality which means that many of their programs are voluntary and that there are a few grant programs with funding available, including various new funds focused on Environmental Justice (presentation on file).

Chair Hayes transitioned into the discussion.

Stephen Kutz, Board Member asked what the ideal Merv filter is. Member Kutz said there was a lot of conversation about minimum standards, but we should look at ideal as well as minimum. Nancy answered that Merv 13 is ideal. Nancy said the Department tries to provide guidance above minimum standards, and there are some standards focused on the care of machines versus human impact. Member Kutz noted not hearing about the proper need to maintain the system and clarified that there is a difference between filter changes and maintenance. Nancy answered yes, funding for maintenance is always being cut, but if you don't maintain your systems, they won't work.

Michelle Davis, Board Executive Director asked if transient accommodations fall under Merv 13 and if this includes restaurants. Ben answered yes, typically those occupancy types fall under the same Merv 13 requirements, but there are some exceptions for very small systems.

Executive Director Davis asked as the building code adopts its new standards, do those apply just to new construction or to facilities that were constructed before the effective date. Ben answered typically, modifications to current buildings do trigger review and a need to meet current building codes.

Kate Dean, Board Member asked if residential cooking is a new component of the state building code. Ben answered that this is not a new section but adds to it, this new code differentiates space types.

Chair Hayes closed the panel with thanks to all the panelists and a reaffirmation that the Board is taking this issue very seriously.

The Board recessed for lunch at 12:40 p.m. and reconvened at 1:30 p.m.

9. RULES HEARING — ON-SITE SEWAGE SYSTEMS, CHAPTER 246-272 WAC

Kate Dean, Board Member provided a brief introduction to this agenda item. The introduction included the Board's rulemaking authority related to on-site sewage systems, the purpose of the Board's rules, and some background history on this rulemaking work. Member Dean then introduced the Board and Department of Health (Department) staff to provide an overview of the rule revision process, the proposed rule for consideration, and written public comments received on the proposed rule.

Andrew Kamali, Board staff, directed Board Members to the key materials for the hearing in their meeting packet and shared additional background information on the rulemaking. Andrew also introduced the information that would be presented in the presentation leading up to the public hearing and information about how the hearing would be conducted.

Jeremy Simmons, Department of Health, presented on the revision of Chapter 246-272A Washington Administrative Code (WAC). Jeremy started by summarizing the history of this rulemaking, followed by the 2017 rule review process and the changes

proposed based on this work. Jeremy then presented the public comments the Department received on the proposed rules and the adjustments that the Department plans to make based on the comments received to date. Jeremy concluded by providing information on the proposed implementation schedule for the proposed rules if the Board adopts the proposed amendments (see presentation on file).

Patty Hayes, Board Chair, thanked Department staff for their presentation and stated that the Board would open the rules hearing for Chapter 246-272A WAC. Chair Hayes read a statement to provide additional information and instructions for the hearing and then formally opened the hearing for public testimony. Each member of the public was given four minutes for their testimony. Testimonies were provided in person and via Zoom Webinar. Microphones were muted after the allotted time expired.

Eric Long gave testimony based on personal experience with on-site sewage systems (OSS) as a homeowner in Washington. E. Long expressed concern with the proposed rules, noting that the current rule only allows a certified professional, certified by the Department, to install, repair, and construct the design of an on-site sewage system. The current rules do not allow a person to make repairs to their own property. You must hire a licensed contractor, an architect, or another licensed professional, which can be very expensive. E. Long stated that this was wrong, and if a person can make their own repairs and meet the standard, the law allows you to do that, but the current code doesn't permit this. E. Long has requested estimates from different licensed professionals and has been quoted between $100,000 and $200,000 for repairs. E. Long stated that if a homeowner could make their own repairs, even following all the standards and meeting the inspection requirements, in comparison, it would cost more like $15,000. E. Long compared this to an individual unable to file their own tax return because they weren't licensed as a tax professional. E. Long concluded by saying that with the current on-site rules, agencies are not serving the public.

Bill Dewey, Director of Public Affairs for Taylor Shellfish, spoke in support of the proposed rules. B. Dewey shared brief comments regarding the proposed rules to reinforce written comments they already submitted during the public comment period. B. Dewey emphasized how important addressing on-site sewage is for their company and the shellfish industry in Washington. Taylor Shellfish has over 14,000 acres of tidelands that they own or lease and farm in six different counties. As the Director of Public Affairs, B. Dewey's role has been dedicated to addressing water quality issues because it impacts Taylor Shellfish's ability to produce safe shellfish for the public. On-site sewage is one of the primary areas of concern. B. Dewey has been involved in prior on-site sewage system rule updates over the years for both residential and large on-site septic systems. B. Dewey commended Jeremy and Jeremy's staff for the process they follow for the on-site sewage rulemaking, stating that the team takes the time to hear and respond to everyone's comments and that it is a thorough process. B. Dewey concluded by urging Board Member adoption of the rule.

Michael Thomas spoke in opposition to the proposed rules based on their personal experience with on-site sewage systems as a homeowner in King County. M. Thomas expressed several concerns with the rule revisions, first related to per capita water use. From M. Thomas personal experience, they are using only 26 to 30 gallons per day per person. M. Thomas noted they could take more measures, like using a toilet that uses

1.0 gallons per flush or using water recycling technology in their shower like Orbital Systems. M. Thomas stated that this is a key parameter in on-site sewage design. M. Thomas says this propagates to the sizes of things like minimum tank size, field size, and all kinds of very expensive items that are needed for an OSS or even a revised OSS. M. Thomas also shared that they have a 30-year OSS that functions flawlessly. M. Thomas stated that the 45 gallons per capita per day referenced in the proposed rule is ancient and would appreciate it if the Board discussed the last time this requirement was revised. M. Thomas also expressed concern with guidance and clarity around the distance from public sewers, which is 200 feet, questioning the feasibility of this requirement. M. Thomas expressed another concern related to the reduction in minimum surface area in the rule, stating that this would increase the re-permitting costs if ever needed and additional inspection requirements. M. Thomas also stated support for the comments heard earlier in testimony related to the exorbitant costs of professional replacement, saying that well-informed and educated citizens should be able to make their own repairs.

Chair Hayes closed the public testimony portion of the rules hearing and asked if there was a motion and second from Board Members to begin questions and discussion.

Motion: The Board adopts the proposed amendments to chapter 246-272A WAC, On-Site Sewage Systems, as published in WSR 23-22-062 with the revisions agreed upon at today's meeting, if any, and directs staff to file a CR-103, Order of Adoption, and establish an effective date for the rules.

Motion/Second: Member Kutz/Member Kwan-Gett. Member Flores abstained.

Stephen Kutz, Board Member, asked staff to clarify if the rule requires every on-site system to have a plan for review and approval. Member Kutz noted in the Department's presentation, there was mention that the rule requires a review of plans every five years. Jeremy clarified that part of the presentation referred to local management plans that primarily help Puget Sound counties design their inspection programs and inventory septic systems. Jeremey stated that it doesn't refer to individual septic systems, these local management plans are only for counties. Jeremy said septic systems do not need to have plans for review and approval every five years.

Member Kutz inquired about the proposed changes to small lot sizes and whether pre-existing lots will be grandfathered in. Jeremy responded that the minimum lot size is 12,500, and the proposed rules increased that and other lot sizes by their respective soil types in a range of 500 to 1,000. Jeremy stated that for small lot sizes, the requirement changed from 12,500 to 13,000, and asked Member Kutz if this answered the question. Member Kutz confirmed that it did.

Member Kutz asked if all lots require reserved areas that remain unbuildable and unmodified. Jeremy said this was correct and that if a person is going to install a septic system on a new lot, this was a long-standing requirement in the rule. Jeremy clarified that an individual cannot build, subdivide, or pave this area.

Kelly Oshiro, Vice Chair inquired about the language in the definitions section of WAC 246-272A-0010, number 72, Puget Sound County, and where it says King County and

Tacoma-Pierce whether the rule is referring to the City of Seattle or King County as a whole. Jeremy clarified that this language refers to the local health jurisdictions in these counties.

Vice Chair Oshiro noted that this was a bit unclear when reading the rule. Vice Chair Oshiro also commented that in future rulemaking staff should look at removing unnecessary use of acronyms and abbreviations to make the rule easier to read and simplifying language where possible.

Member Kutz asked staff to clarify rule requirements around on-site sewage self-installation, which the first public testifier spoke to during the hearing.

Jeremy shared that, in general, counties can allow owners to do installs on their own properties and that the public testimony was referencing the parts of the rule where it says resident owner and installations and design. Jeremy clarified that the state code says if you own a property and you live there, you can do that install and design, but local health jurisdictions often restrict that further and say not for proprietary products and not for repairs that are close to the shoreline. Jeremy said some local rules are stricter than the state rules.

Kate Dean, Board Member, commented that this is an important issue and that in rural counties like Jefferson County, most residents are on septic systems, and shellfish is their largest farm gate industry. Member Dean asked staff to remind Board Members about the requirements around inspections and whether homeowners can do inspections on some non-proprietary systems.

Jeremy confirmed that homeowners are allowed to do routine inspections. Jeremy also clarified that the proposed rules do not change anything related to routine inspections, which are required every three years for a gravity, low-technology system and every year for a higher-technology septic system, and these requirements have been in place since 2005. Jeremy noted that state rules, allow homeowners to do these inspections themselves, and many counties develop certification or approval processes for homeowners to complete inspections, but counties do not need to require homeowners to do their own inspections.

Member Dean commented on the issues that the first testifier, Eric Long, brought up. Member Dean said these rules are important for public health and environmental public health, but there's also an affordability question, and often a problem where these systems can become unaffordable and make homeownership out of reach for many people, especially in more rural counties. Member Dean stated that the challenge with these rules is that they need to strike a balance and noted appreciation for staff because they have worked with the public to try and find this balance of affordability and safety. Member Dean also inquired if during the rulemaking process if there was a discussion around incentivizing conservation, especially as it relates to water use and the per capita gallon use provision in the rule.

Jeremy said that this topic was briefly discussed during committee meetings. Jeremy noted that the public testifier who spoke about this issue had good points and that this requirement isn't necessarily current. Jeremy stated that, in general, the Department

sizes septic systems based on numbers from the Environmental Protection Agency (EPA) and has been in an adaptive management mode for several years or decades based on these numbers. Jeremy noted that if the rules proposed smaller water use and drain fields, practitioners would say this isn't a good move because drain fields fail at a high rate, about 7 to 20%, depending on the location. Jeremy stated that proposing lower water use rates and drain fields could potentially lead to more failures, and the goal is to lower failure rates for these systems, and this is not where we should try to cut costs. Jeremy also shared that they are working on funding to help people with this infrastructure and that, largely, this issue stems from the U.S. wastewater system infrastructure. Jeremy concluded that as a society, we need to acknowledge this and try to figure out how to make sure things are functioning while also helping people pay for them without putting the total costs of these systems on individuals.

Member Dean added that good policy should incentivize behavior change. Member Dean stated that, for example, if a homeowner were to take their greywater treatment out of their septic system, there are permittable pathways to do that, but it's extremely expensive, and there wouldn't be cost savings. Member Dean said if we always default to the larger, more expensive system, that doesn't necessarily bring about the types of change that we need for overall societal benefits. Member Dean concluded that the Board wouldn't be able to solve this problem today but wanted to raise this issue in the discussion. Member Dean also asked about privies, whether counties still allow them, and if privies are addressed in this rule or perhaps another rule.

Jeremy clarified that privies and other technologies, like composting toilets, are captured in their recommended standards and guidance document.

10. EMERGENCY RULEMAKING — ON-SITE SEWAGE SYSTEMS, WAC 246-272A-0110, PROPRIETARY TREATMENT PRODUCTS AND SUPPLY CHAIN SHORTAGES

Tao Sheng Kwan-Gett, Secretary's Designee, introduced the item. Regardless of today's action of the on-site sewage system (OSS) rules hearing, the following matter requires separate action to maintain continuity of the rule. The fifth emergency rule is set to expire on February 3, 2024. The Department of Health (Department) is requesting a sixth emergency rule to prevent a break in this emergency rule before the completion of the permanent rulemaking. Andrew Kamali, Board Staff, provided additional background on this rule and referred to the meeting materials for more information (materials on file). Andrew introduced Jeremy Simmons, Department of Health, to briefly explain the Department's request.

Patty Hayes, Board Chair, made note of Jeremy doing a stellar job of providing multiple briefings on this rule before. Chair Hayes asked whether Board Members need a briefing or if the Board is ready to make a motion.

Motion: The Board adopts the proposed amendments to chapter 246-272A WAC, On-Site Sewage Systems, as published in WSR 23-22-062 with the revisions agreed upon at today's meeting, if any, and directs staff to file a CR-103, Order of Adoption, and establish an effective date for the rules.

Motion/Second: Member Kwan-Gett/Member Dean. Approved unanimously.

Steve Kutz, Board Member asked whether progress was being made on addressing supply chain issues. Chair Hayes stated the rule that was just passed should take care of these issues, and asked Jeremy to confirm. Jeremy confirmed that this is correct.

The Board took a break at 2:43 p.m. and reconvened at 3:00 p.m.

11. PETITION FOR RULEMAKING FOR CHAPTER 246-760 WAC, VISUAL SCREENING STANDARDS – SCHOOL DISTRICTS

Socia Love-Thurman, Board Member, summarized the Board's petition for rulemaking process, and the statutory requirements the Board must follow when a petition is received. Member Love-Thurman stated that in November, the Board received a petition for rulemaking to amend its school vision screening standards to add screening for color vision deficiency (CVD), also known as color blindness (materials on file).

Molly Dinardo, Board staff, introduced two subject matter experts in school vision screening standards. The first person is Dr. Bruce Moore, New England College of Optometry, National Center for Children Vision and Eye Health. The second is Annie Hetzel, Office of Superintendent Public Instruction. Molly provided more information on the petition and CVD, an overview of the National Childhood Vision Screening guidelines, and the Board's options for responding to the petition (materials on file).

Patty Hayes, Board Chair, opened the topic for further discussion.

Steve Kutz, Board Member, inquired about the rationale for periodic screening for color vision deficiency at prescribed intervals, as you're either born with color vision deficiency or not. Dr. Moore responded that Member Kutz was correct, and in almost all cases, it is a genetic condition, with only some rare diseases affecting color vision over time. Dr. Moore also added several comments in addition to the presentation from staff. Dr. Moore shared that they have color vision deficiency, and it wasn't until they had to take Organic Chemistry at University that it became an issue. Dr. Moore also provided more details on why color vision deficiency screening in schools isn't recommended, including that if schools do not have a precise, correct, and expensive light source, they cannot conduct accurate and reliable testing. Dr. Moore also noted that the color plates are expensive and sensitive. If fingerprints get on the plates, the accuracy of the test can be destroyed. Dr. Moore concluded that implementing this screening in all school buildings across all districts wouldn't be feasible and that color vision deficiency isn't as big of a problem as people without the condition think it is.

Paj Nandi, Board Member, thanked Molly for the strong background and context, and Dr. Moore for the additional comments. Member Nandi asked what we know about the states currently testing for color vision deficiency and the costs of screening. Molly

responded that some states have color vision deficiency screening as a requirement in their state law, while others are doing targeted screening in kindergarten or screening by referral from teachers to the school nurses. Molly stated that it varies, and they would need to follow up with additional information and invited Dr. Moore and Annie to chime in if they had more insight to share.

Dr. Moore added that if you do not have the precise light source or the precision of plates, screening results will be off, which is a particular issue in a school setting. Dr. Moore stated that in school screening programs, there are a lot of people handling the plates and tests, which can leave more room for error. Dr. Moore said the bottom line is that testing for color vision deficiency should be done at an eye doctor's office with proper materials and equipment, and there is little value in doing it statewide through mass screening. Molly stated that there are research documents that outline which states conduct testing for color vision deficiency and can forward these materials to Member Nandi.

Tao Kwan-Gett, Board Member, thanked Molly and the subject matter experts for their presentation. Member Kwan-Gett inquired about the administrative and personnel burden of adding this testing and whether the type of color vision deficiency that is caused by certain eye conditions can be modified by early detection of color vision deficiency. Annie asked to respond to some of the topics previously discussed. Annie commented on the challenge of tracking students that have been tested, and said it is very difficult, logistically, during annual school screenings to know who has been screened for color vision or any other kind of vision screening. Regarding the administrative burden, Annie stated that not all schools have vision and hearing screening equipment on site, and many school nurses need to be able to pack up the equipment and travel with it from school to school. Annie also mentioned that recent changes to the vision screening rules five or six years ago increased the time that students are out of class for vision screenings, and several school nurses have experienced pushback from school administrators who are upset about students missing out on educational time. Annie concluded that adding another test would complicate this further and would put school nurses in a challenging position.

Kate Dean, Board Member, stated that Member Kwan-Gett's second question wasn't answered. Member Kwan-Gett repeated the question of whether early detection of conditions can be modified by testing for color vision deficiency. Dr. Moore answered no, not really. Dr. Moore said that an individual who has an ocular disease condition with color vision defects as a component is almost always going to have more significant visual acuity deficits that would become apparent or picked up on a screener. Dr. Moore added that there is nothing specific about color vision deficiency testing that would improve the ability to detect other eye conditions at an earlier time.

Motion: The Board declines the petition for rulemaking to revise applicable sections of chapter 246-760 WAC to include screening for color vision deficiency in the Washington State school vision screening standards and procedures under RCW 28A.210.020 for the reasons articulated by Board Members. The Board directs staff to notify the petitioner of the Board's decision.

Motion/Second: Member Kutz/Member Nandi. Approved unanimously.

Member Love-Thurman thanked the subject matter experts and Molly for their time. Member Love-Thurman said that given the high prevalence of color vision deficiency in boys, it sounds like we need to adapt school teaching materials and classrooms to better suit folks, knowing that there are mostly boys out there who don't see red and green very well.

Kate Dean, Board Member thanked the petitioner and their efforts. Member Dean stated that if implementing this test didn't come with so many challenges, then it would have maybe been a good idea. Member Dean added that they hope there is more of an effort to identify kids with color vision deficiency.

12. 2024 LEGISLATIVE STATEMENT

Michelle Davis, Board Executive Director, reminded Board Members that she had shared the 2023 Legislative Statement at the November Board meeting, and directed members to an updated 2024 draft for their consideration.

Executive Director Davis said the legislative document is intended to guide staff during the 2024 legislative session. Executive Director Davis said during legislative sessions, Board staff identify, review, and analyze bills that align with the Board's legislative statement. Executive Director Davis shared that the team may just monitor a bill's progress through the legislative session or submit written comments to the sponsor or a committee in support of or against the legislation, or may provide testimony at hearings on behalf of the Board. Board staff often suggest technical changes to improve legislation. Executive Director Davis shared the draft and asked the Board to help take action to finalize the statement (materials on file).

Paj Nandi, Board Member asked if the Board previously adopted a statement of racism as a public health crisis. Executive Director Davis replied yes. Member Nandi asked if there's a way to recognize the Board standing behind that statement, saying this aligns with Chair Hayes's recommendation that the crisis was a call to action around the pandemic and that the work is not done.

Patty Hayes, Board Chair concurred with Member Nandi, saying the intent was to make the statement stronger by calling it out and making it clear.

Steve Kutz, Board Member, said any stresses in the system exacerbate the problem.

Executive Director Davis talked about the data desegregation change, saying we added the national academies' recommendation and clarified data collection. Member Oshiro's suggestion included desegregation for policymakers for better informed decisions regarding disparities in communities.

Executive Director Davis described additional updates, including maternal mortality, newborn screening, Health Impact Reviews (HIR), school environment and safety, shellfish sanitation, drinking water, oral health, opioids, mental health, and other Board work.

Member Kutz said this is an incredible amount of work between meetings. Executive Director Davis complimented the Board staff.

Motion: The Board adopts the Statement of Policy on the 2024 Legislative Issues as discussed on January 10, 2024, including Board Member Nandi's suggestion to make strong and clear the piece on racism as a public health crisis.

Motion/Second: Vice Chair Oshiro/Member Kutz. Approved unanimously.

13. SNOHOMISH COUNTY HEALTH DEPARTMENT COMPLAINT

Kelly Oshiro, Vice Chair, introduced the complaint filed on November 28, 2023, against the Snohomish County Health Department (SCHD) Director and Local Health Officer. Vice Chair Oshiro provided background on the Board's authority related to complaints against local health officials and stated that Washington law allows anyone to file a complaint and that Board authority allows the Board to authorize an investigation if the complaint is warranted. Vice Chair Oshiro provided some additional details on the complaint and asked if there were any Board Members who needed to recuse themselves from this discussion.

Patty Hayes, Board Chair, stated they were turning the gavel over to Vice Chair Oshiro and would be recusing themself from this discussion. Paj Nandi, Board Member, also recused themself.

Vice Chair Oshiro had the Board Members who recused themselves step away from the discussion table.

Molly Dinardo, Board staff provided additional information on the complaint and directed Board Members to the materials for this agenda item. Molly mentioned that Snohomish County provided a response to the initial complaint and that per the Board's policy for responding to complaints, the Board sends a copy of the complaint to the subject local health officials, and they are permitted to respond if they choose to. Molly then outlined the Board's options for possible action on the complaint.

Vice Chair Oshiro opened the discussion and asked if a Board Member wanted to make a motion before the discussion.

Steve Kutz, Board Member, said before making a motion, they wanted to acknowledge that health officers have had an incredibly hard book of business in Washington during the pandemic. Member Kutz stated that many health officers have been pulled in multiple directions during the pandemic, but the pandemic has been declared as over.

Vice Chair Oshiro agreed and stated Governor Inslee has rescinded all emergency orders, so there are not currently any masking guidelines statewide, except for certain facilities.

Member Kutz added that this guidance is recommended for people to follow, but they are not required.

Kate Dean, Board Member, made a motion.

Motion: The Board determines that the complaint does not merit an investigation because, for the reasons articulated by the Board, it does not indicate a possible violation of public health law and that the Board directs staff to notify the complainant of the Board's decision.

Motion/Second: Member Dean/Member Kutz**.** Approved unanimously

Vice Chair Oshiro asked if there were any further comments or discussions.

Dimyana Abdelmalek, Board Member, said they wanted to echo what has already been shared, and that at this point, masking is largely voluntary.

Tao Kwan-Gett, Secretary's Designee, added that Board Members were correct in saying that there are no current statewide requirements for masking. Member Kwan-Gett said that state guidance is aligned with guidance from the Centers for Disease Control (CDC), and guidance cannot be enforced. Member Kwan-Gett shared that the Board needs to consider these complaints when they are submitted because it is possible there could be situations where local health officials are not serving the needs of their community. Member Kwan-Gett said that in this case, if there is a standard of practice for public health, the health officer and administrator in Snohomish County exceed that standard. Member Kwan-Gett agreed with the motion that this complaint should be denied as it does not merit an investigation.

Member Kutz commented on the complainant's request for mandatory isolation and quarantine. Member Kutz said during the pandemic, in the United States, mandatory or enforced quarantine did not occur, only recommendations for people to voluntarily quarantine themselves.

Mindy Flores, Board Member, stated they could not determine where there was substantial evidence that a violation occurred and agreed with Member Kwan-Gett that none of these complaints should be taken lightly.

Chair Hayes and Member Nandi returned.

14. BOARD MEMBER COMMENTS

Tao Kwan-Gett, Secretary's Designee, said a media release went out today that the Department of Health (Department) will supply naloxone to schools that want it. Member Kwan-Gett shared this was done in collaboration with the Office of Superintendent Public Instruction (OSPI) and is meaningful and potentially lifesaving. Member Kwan-Gett said hopefully no school will use it, but they will have it if they need it, and this shows how seriously we take it.

Stephen Kutz, Board Member, said it appears that the federal government is looking again at the issue around Kratom and asked Board staff to research. Member Kutz said the items are coming into the United States without knowledge of ingredients and people are dying from using them.

Michelle Davis, Board Executive Director, recommends having the April 10 Board meeting in Eastern Washington, based Board Member requests to hear from the community regarding the State Health Report. Executive Director Davis said the March meeting is packed and that an April 10 meeting will give staff additional time to engage with the community in the process.

Patty Hayes, Board Chair thanked the staff and Board Members for a full meeting.

ADJOURNMENT

Patty Hayes, Board Chair, adjourned the meeting at 3:59 p.m.

WASHINGTON STATE BOARD OF HEALTH

WASHINGTON STATE BOARD OF HEALTH

Public Comment

Added three business days prior to meeting

There are two sections.
The first contains timely materials sent during the open public comment time period.
The second section contains materials sent within 15 minutes of the deadline. Included as they are relevant to a topic under consideration by the Board at the March 13 meeting.

From: Christi Ellefson
Sent: 1/17/2024 11:59:52 AM
To: DOH WSBOH
Cc:
Subject: Important vaccine information


attachments\55A4B10E46FE4306_20240103-halt-use-covid19-mrna-vaccines.pr.pdf

External Email

https://www.floridahealth.gov/_documents/newsroom/press-releases/2024/01/20240103-halt-use-covid19-mrna-vaccines.pr.pdf <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.floridahealth.gov%2F_docum
releases%2F2024%2F01%2F20240103-halt-use-covid19-mrna-vaccines.pr.pdf&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Cfabdfcb1158547af321008dc17965613%7C

From: Scott Shock
Sent: 1/7/2024 2:07:20 AM
To: DOH Secretary's Office,DOH Office of the Chief of Staff,DOH Office of Innovation and Technology,DOH Office of Prevention Safety and Health,DOH Office of Strategic Partnerships,DOH Office of Health and Science,DOH Office of Public Affairs and Equity,DOH OS Executive Office of Policy Planning and Evaluation,DOH Office of Resilience and Health Security,DOH WSBOH,AGOOmbuds@atg.wa.gov,Ferguson, Bob (ATG)
Cc:
Subject: Call for a Halt to the Use of COVID-19 mRNA Vaccines

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External Email

I'm still looking forward to responses on what actions the WSDOH, WSBOH, and WA AG are taking to protect the people of Washington State against these unsafe products, and to gain justice for those injured by these products. Here is more for your consideration.

<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fopen.substack.com%2Fpub%2Fst
summary-of-the-evidence-
against%3Fr%3Dtaogl%26utm_campaign%3Dpost%26utm_medium%3Demail&data=05%7C02%7Cwsboh

A summary of the evidence against the COVID vaccines
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fopen.substack.com%2Fpub%2Fst
summary-of-the-evidence-
against%3Fr%3Dtaogl%26utm_campaign%3Dpost%26utm_medium%3Demail&data=05%7C02%7Cwsboh

open.substack.com
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fopen.substack.com%2Fpub%2Fst
summary-of-the-evidence-
against%3Fr%3Dtaogl%26utm_campaign%3Dpost%26utm_medium%3Demail&data=05%7C02%7Cwsboh

Here is a short list of reasons that everyone should be concerned about the COVID vaccine. This is not an exhaustive list.

1. Doctors are told to trust the FDA and CDC, but not verify, when prescribing vaccines. All the post-marketing safety data is kept hidden by health authorities so not even doctors can look at the data themselves to find out if any vaccine is safe. Doctors

have to trust the authorities. They are essentially told: “trust, do not verify.”

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6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7

2. The CDC itself doesn’t have the data to make a post-marketing independent vaccine safety assessment and they are not interested in obtaining the data either!The CDC relies on the FDA who relies on the manufacturer to test the product. The CDC could ask states for vaccination records tied to death records, but they don’t want to even ask because if they did an analysis, it could be discovered in a FOIA request. The CDC basically has no interest whatsoever in verifying what the actual safety data is.

3. Lack of transparency by health authorities. Not a single health authority anywhere in the world has ever released anonymized record-level patient data for independent researchers to assess the safety of any vaccine. There isn’t any paper in a peer-reviewed journal showing that health outcomes are improved if public health data is kept secret.

4. Lack of interest in data transparency by the medical community. Can you name a single high-profile pro-vaccine member of the medical community who has called for data transparency of public health data? Time-series cohort analyses can be easily produced by health authorities and published for everyone to see. These would show safety signals and do not jeopardize patient privacy. These are all kept hidden.

5. We aren’t allowed to see even the simplest of charts. Wouldn’t it be great to define two cohorts on July 1, 2021: COVID vaccinated vs. COVID unvaccinated. Then you simply record the deaths from that point forward and plot them. Why isn’t this being published?

6. Misinformation is deemed to be a problem, but the people making these statements are unwilling to take any steps to stop the so-called misinformation. These steps include: open public discussion to resolve differences of opinion and making public health data available/public in a way that preserves privacy. For example, HHS (as well as every state health department) should welcome all of us with open arms and invite us to query their databases (such as VSD and Medicare in the case of HHS) and publish whatever we find. Why does this information need to be hidden? The numbers tell the story, not the individual records.

7. No response from health authorities to reasonable requests. I’ve sent emails to Sarah Caul of the UK ONS on four ways the ONS can increase data transparency. There was no response.

8. No response when asked to explain damaging evidence. When credible scientists receive government data that shows very troubling safety signals, there is a total unwillingness of any health authority to discuss the matter and resolve it.

9. The US Medicare data clearly shows mortality increases after people take the jab. Is there any epidemiologist who can explain why deaths rose during a period in time when they should have been falling (per the Medicare death data)?

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1f9b1b3fd807%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7

For the first 120 days after the shots given in March 2021, death rates overall were falling. But if you got the vaccine, your death rates went up. We know from data from other vaccines that the baseline death rate of 81-year olds in Medicare is 3.85%, so the baseline death rate of this group is <800 deaths a day. These deaths climb far above baseline after you took the COVID shot.

10. The patient-level data released from NZ data confirms that mortality increases after the shots are given despite the fact that most of the shots were given during time periods when deaths were falling

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6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7

NZ data: Doses 2 and 4 were given while background mortality was falling, dose 3 while rising. So we'd expect the slope to fall in the first 6 months after vaccination. It does the opposite.

11. Anecdotes such as the one from Jay Bonnar
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2F33a
c373-4b28-b55c-
d90cae2b6e57%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
who lost 15 of his DIRECT friends unexpectedly since the shots rolled out. Four of the 15 died on the same day as that vaccine was given. Before the shots rolled out, Jay had lost only one friend unexpectedly. The probability this happened by chance is given by poisson.sf(14, .25) which is 5.6e-22. So this can't happen by chance. SOMETHING killed Jay's friends and 4 of the 15 died on the same day as they were vaccinated. Is there a more plausible explanation for what killed Jay's friends? All of them who died were vaccinated with the COVID vaccines.

12. Well done studies like the one done by Denis Rancourt
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2F8fb2
dde4-4eec-bce1-
99237cda9de5%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
showing 1 death per 800 shots on average. Jay Bonnar estimates he has around 14,000 friends so Jay's numbers are consistent with Rancourt's results.

13. Survey data like Skidmore

<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2Fb2c
6f7a-420b-a525-
325379d1e6da%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
and Rasmussen Reports
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2F485
67aa-4d85-818e-
422a5362a138%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
showing that hundreds of thousands of Americans have been killed by the COVID shots. There have never been any counter surveys published showing this not to be the case.

14. The lack of any success stories. It appears that "vaccine success stories" where COVID infection fatality ratios dropped or that myocarditis cases plummeted do not exist. The US Nursing home data
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2F6fa3
b881-48d4-ab92-
b38a07406cd8%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
shows that the infection fatality rate (IFR) increased after the vaccine rolled out. There is nobody using that data making the claim it reduced the IFR.

15. Anecdotes from healthcare are extremely troubling. One nurse
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2Fe9b
6f4c-4e32-b913-
8a8057b3f865%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
reported a hospital admission rate that was 3X higher than anything in the 33-year history of the hospital after the COVID vaccines rolled out. Symptoms rarely ever seen were common after vaccines rolled out in that age group.

16. Lack of autopsies in clinical trials and post-marketing. The CDC doesn't request anyone to do autopsies even for people who die on the same day as they got the vaccine. Don't they want to know what killed those people… just to be sure?

17. Young people dying in sleep. There are way too many cases of young people who die in their sleep after being vaccinated. Doctors say this is a rare event. Now it is much more common. If the shots are safe, why is this happening?

18. I have direct personal experience with the vaccine: two people I know were killed by the vaccine, none from COVID. I know many people who are vaccine injured from the COVID vaccine.

19. Ed Dowd's book statistics. This very popular book ("Cause Unknown"
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2F15c
c0d0-49b8-b054-
b3c76b9b9d2b%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
) listed 500 who died unexpectedly. Ed didn't know how many were unvaccinated. Only one person has come forward saying that one of the people in the book who died after the vaccines rolled out was unvaccinated.

20. Prominent doctor/scientists switching sides. Paul Marik is one of the top intensivists in the world. After seeing many COVID vaccine injured patients, he changed his mind about the safety of vaccines. When he was not allowed to practice medicine consistent with his Hippocratic Oath, he resigned his position.

21. The corruption with COVID protocols. The COVID hospital protocols likely caused

90% of the COVID deaths in hospitals. This led to Paul Marik resigning. See details in this article <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2Fe9b6f4c-4e32-b913-8a8057b3f865%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7. Why are doctors forced to use hospital protocols that kill a huge percentage of patients instead of using their best judgment to save patients?

22. This JAMA paper <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2Fb692677-4b5b-a000-97b2419f3cd4%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7 shows that COVID and influenza vaccines don't work. Why are we pushing a vaccine where the statistics clearly show the vaccines don't work?

23. The consistency of the data. There have been no counter-anecdotes showing the vaccines are safe. I keep looking for one and come up empty.

24. No debates with anyone prominent promoting the government narrative.Those who promote the narrative refuse to engage in any scientific discussions to resolve differences of opinion. This is similar to the question of whether vaccines cause autism: nobody who thinks it doesn't is willing to engage in a public discussion about it to discuss the evidence. Why not resolve the issue through dialog? It isn't resolved in the peer-review literature where half the papers say vaccines cause autism and the other half don't. Why can't we talk about it?

25. Fear and intimidation tactics are used to silence dissent. Open debate would be more productive. But people are not allowed to hold or discuss views that go against the "consensus" or they will lose their jobs, their certifications, or their medical licenses. Health care workers are told they will be fired if they report an adverse event to VAERS, there are nurses who won't talk about anaphylaxis after getting the vaccine for fear of being fired, vaccine injuries are covered up, hospital workers are afraid to talk about it at work.

26. The cognitive dissonance is very disturbing. When healthcare workers bring up the topic of mortality and morbidity due to the vaccine, their peers say nothing and walk away.

27. Censorship tactics employed by the US government to silence dissent instead of public recorded open debates. History has shown that purveyors of censorship are always on the wrong side of the issue.

Scott

On Jan 4, 2024, at 1:11 AM, SCOTT SHOCK <ssshock@comcast.net> wrote:

☐
Dear WSDOH and WSBOH Members, and Attorney General's Office,

The Florida State Surgeon General has been a leader in protecting the people of his state against the unsafe mRNA COVID vaccinations. What actions are the WSDOH, WSBOH, and WA AG taking to protect the people of Washington State against these unsafe products, and to gain justice for those injured by these products (including members of

my family)? I look forward to your responses.

Scott Shock
Seattle

Joseph A. Ladapo, MD, PhD on X: "I am calling for a halt to the use of mRNA COVID-19 vaccines. https://t.co/olg8VTh6gB" / X (twitter.com)
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Ftwitter.com%2FFLSurgeonGen%2

Florida State Surgeon General

Calls for Halt in the Use of

COVID-19 mRNA Vaccines

Tallahassee, Fla. – On December 6, 2023, State Surgeon General Dr. Joseph A. Ladapo sent a letter <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.floridahealth.gov%2Fabout%
06-2023-DOH-Letter-to-FDA-RFI-on-COVID-19-Vaccines.pdf%3Futm_medium%3Demail%26utm_source%3Dgovdelivery&data=05%7C02%7Cwsboh%40s to the United States Food and Drug Administration (FDA) Commissioner Dr. Robert M. Califf and Center for Disease Control and Prevention (CDC) Director Dr. Mandy Cohen regarding questions pertaining to the safety assessments and the discovery <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fosf.io%2Fpreprints%2Fosf%2Fmjc of billions of DNA fragments per dose of the Pfizer and Moderna COVID-19 mRNA vaccines.

The Surgeon General outlined concerns regarding nucleic acid contaminants in the approved Pfizer and Moderna COVID-19 mRNA vaccines, particularly in the presence of lipid nanoparticle complexes, and Simian Virus 40 (SV40) promoter/enhancer DNA. Lipid nanoparticles are an efficient vehicle for delivery of the mRNA in the COVID-19 vaccines into human cells and may therefore be an equally efficient vehicle for delivering contaminant DNA into human cells. The presence of SV40 promoter/enhancer DNA may also pose a unique and heightened risk of DNA integration into human cells.

In 2007, the FDA published guidance on regulatory limits for DNA vaccines in the Guidance for Industry: Considerations for Plasmid DNA Vaccines for Infectious Disease Indications (Guidance for Industry) <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.fda.gov%2Fmedia%2F7366 . In this Guidance for Industry, the FDA outlines important considerations for vaccines that use novel methods of delivery regarding DNA integration, specifically:

* DNA integration could theoretically impact a human's oncogenes – the genes which can transform a healthy cell into a cancerous cell.
* DNA integration may result in chromosomal instability.
* The Guidance for Industry discusses biodistribution of DNA vaccines and how such integration could affect unintended parts of the body including blood, heart, brain, liver, kidney, bone marrow, ovaries/testes, lung, draining lymph nodes, spleen, the site of administration and subcutis at injection site.

On December 14, 2023, the FDA provided a written response providing no evidence that DNA integration assessments have been conducted to address risks outlined by the FDA <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.fda.gov%2Fmedia%2F7366 themselves in 2007. Based on the FDA's recognition of unique risks posed by DNA

integration, the efficacy of the COVID-19 mRNA vaccine's lipid nanoparticle delivery system, and the presence of DNA fragments in these vaccines, it is essential to human health to assess the risks of contaminant DNA integration into human DNA. The FDA has provided no evidence that these risks have been assessed to ensure safety. As such, Florida State Surgeon General Dr. Joseph A. Ladapo has released the following statement:

"The FDA's response does not provide data or evidence that the DNA integration assessments they recommended themselves have been performed. Instead, they pointed to genotoxicity studies – which are inadequate assessments for DNA integration risk. In addition, they obfuscated the difference between the SV40 promoter/enhancer and SV40 proteins, two elements that are distinct.

DNA integration poses a unique and elevated risk to human health and to the integrity of the human genome, including the risk that DNA integrated into sperm or egg gametes could be passed onto offspring of mRNA COVID-19 vaccine recipients. If the risks of DNA integration have not been assessed for mRNA COVID-19 vaccines, these vaccines are not appropriate for use in human beings.

Providers concerned about patient health risks associated with COVID-19 should prioritize patient access to non-mRNA COVID-19 vaccines and treatment. It is my hope that, in regard to COVID-19, the FDA will one day seriously consider its regulatory responsibility to protect human health, including the integrity of the human genome."

In the spirit of transparency and scientific integrity, State Surgeon General Dr. Joseph A. Ladapo will continue to assess research surrounding these risks and provide updates to Floridians.

On September 13, 2023, State Surgeon General Dr. Joseph A. Ladapo provided guidance <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Ffloridahealthcovid19.gov%2Fwp-content%2Fuploads%2F2023%2F09%2F20230913-booster-guidance-final.pdf%3Futm_medium%3Demail%26utm_source%3Dgovdelivery&data=05%7C02%7Cwsboh%40sboh.
against COVID-19 boosters for individuals under 65 and younger. In addition to aforementioned concerns, providers and patients should be aware of outstanding safety and efficacy concerns outlined in the State Surgeon General's previous booster guidance released in September.

From: Arne Christensen
Sent: 1/17/2024 11:09:22 AM
To: DOH WSBOH
Cc:
Subject: lonely people walking in the rain wearing face masks

External Email

The health department needs to stop lying to us about the effectiveness of face masks, vaccines, and social distancing for protecting people against covid. I just saw a man with a flimsy blue plastic mask walking outdoors, by himself, in the cold rain. He is only doing this because public health agencies have lied about masks for 4 years, and have inexplicably failed to advise people that masks don't work when wet.

From: bill teachingsmiles.com
Sent: 1/8/2024 8:32:17 AM
To: DOH WSBOH
Cc:
Subject: Public Comment 1/10/2024 Osmunson

External Email

Dear Washington State Board of Health,

I am requesting to provide public comment for the January 10, 2024 Board of Health Meeting.

My comments:

The Board of Health is the highest health authority in Washington State. Overhearing one Board member say, "but we are not supposed to have to look at the science." My jaw dropped almost to the floor. If the Board does not read science, what does the Board use to determine "health" policy such as fluoridation? Gossip? Rummers? Industry? The Dental Lobby?

In effect, the Board trusts the dental lobby and disregards inconvenient empirical factual evidence, laws and authorities such as:

I. The Washington State Board of Pharmacy, who determined that fluoride is a "legend drug." However, the Board of Health disagrees and trusts the dental lobby. The Board of Pharmacy was disbanded in part because they agreed with the law and science that fluoride ingested with intent to prevent disease is a prescription drug. Are you Board of Health doctors willing to put your license on the line prescribing the drug for everyone in Washington State without their consent or being patients of record? That would be unethical. Pharmacists have more training and expertise with toxins, dosage, adverse reactions and inter reactions of toxins than any other licensed profession. What empirical evidence does the Board of Health have which disagrees with the Board of Pharmacy? None. The Board of Health is violating science and laws of health.

See: Krzeczkowski JE, et al. Prenatal fluoride exposure, offspring visual acuity and autonomic nervous system function in 6-month-old infants. <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.sciencedirect.com%2Fscienc
Environment International. 2023

II. U.S. Congress which has authorized the Food and Drug Administration Center for Drug Evaluation and Research (FDA CDER) to determine the efficacy, dosage, safety and label of substances used to prevent disease. No, the Board trusts the dental lobby.

III. FDA CDER warns, "Do Not Swallow". Instead, the Board trusts the dental lobby and promotes mandated fluoride ingestion for everyone without patient consent, without patient dosage control, without the Doctor as legal intermediary, without regard for age or health of the patient. FDA CDER has determined fluoride ingestion lacks evidence of efficacy. And the FDA has given warnings to bottled water manufacturers (not FDA CDER approved) the fluoridated water must not be marketed to those under two years of age. The Board of Health is harming the public by disagreeing with authorized regulatory agencies.

IV. The Environmental Protection Agency scientists finding over two decades ago that fluoridation borders on a criminal Act because of toxicity and lack of current benefit. The Safe Drinking Water prohibits the EPA from adding anything to water to treat humans, so the Board trusts the dental lobby. And the EPA Dose Response Analysis and Relative Source Contribution of 2010 reporting that most or all infants and toddlers are ingesting too much fluoride.

V. The National Research Council 2006 report for the EPA that EPA's Maximum Contaminant Level for fluoride was not protective and harms most if not all cells and systems of the body. Instead, the Board of Health trusts the dental lobby. Fluoride is a contaminant the Board recommends adding to water.

VI. The National Toxicology Program reporting fluoride is a presumed developmental neurotoxin with 55 human studies, 52 reported IQ loss a 95% consistency. And their meta-analysis reports IQ loss. But no. The Board would rather trust the dental lobby rather than toxicologists for toxicity. Not everyone has the same sensitivity to drugs/toxins or the same health or the same ability to handle drugs/toxins. Some individuals had much more IQ loss and some were probably unaffected. The mean is not protective or representative of each individual. The Board must protect everyone, not just the healthiest and wealthiest.

"This January, Birnbaum <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fen.wikipedia.org%2Fwiki%2FLinda issued a scathing legal declaration as part of the lawsuit, writing, 'The decision to set aside the results of an external peer review process based on concerns expressed by agencies with strong policy interests on fluoride suggests the presence of political interference in what should be a strictly scientific endeavor.' Birnbaum said she issued the legal declaration in part over concerns the report might never be publicly released… the science proves there is 'no real benefit' from ingesting fluoride. 'The benefit from fluoride is from topical applications,' she said." - Capital and Main (March 14, 2023)

VII. Only one RCT (randomized controlled trial, the highest quality of research) of fluoride ingestion has been published and it report no statistical benefit from ingesting the fluoride. That's right. NO, NONE, ZERO quality studies reporting dental benefit of fluoride ingestion. No wonder the FDA said the evidence of efficacy is incomplete.

VIII. The lack of mechanism of action. Fluoride cannot go from the blood to the tooth pulp chamber through the calcium rich dentin and enamel to the outside of the tooth where the dental caries are forming and active. Fluoride during swallowing of water is short term and little gets to the lower teeth and the theoretical slight increase of fluoride in saliva with water at 0.7 ppm is too dilute to have an effect. Research has not reported a benefit at 700 ppm let alone 0.7 ppm.

IX. 97% of Europe does not fluoridate their water. And their dental caries are a similar rate as fluoridated communities and states not fluoridated.

X. CDC has known since the publication of the 2006 National Research Council (NRC) report to the EPA, that there is no safety data for susceptible sub-populations and significant scientific evidence of probable harm. In 2018, Mr. Casey Hannan of the CDC admitted under oath in a deposition for the trial in federal court expected to wrap up in February 2024 that the CDC accepts the 2006 NRC conclusions. Mr. Hannan also admitted that the CDC has no safety data specific to pre- and post-natal exposure. We understand Mr. Hannan decided to retire before commencement of that trial.

XI. Public Health Service (PHS) researchers advised the PHS in 1956 and 1961 that a portion of the allergic population would experience significant and acute ill effects from fluoridation programs with no pragmatic recourse to avoid the irritant. Other researchers in that decade advised that the placentas of women living in 'optimally' fluoridated

communities were saturated with fluoride at twice the concentration of the water they drank. They opined that although they didn't know the fetal impact, the mothers would probably be fine. (Feltman 1956; Feltman & Kosel 1961; Gardner et al. 1952

PHS lowered fluoridation concentration recommendation from 0.7-1.2 mg/L to 0.7 mg/L. However, no studies on efficacy have been done at current lower concentrations.

Once again, I am calling for the Board to remove their endorsement of fluoridation from your web site and protect the fetus and infants from known harm.

Current evidence is alarming on fluoride's contribution not just to lower IQ, but also to preterm birth and infant mortality.

See also https://www.fluoridelawsuit.com/science <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.fluoridelawsuit.com%2Fscier

Once again, I am calling for the Board to remove their endorsement of fluoridation from your web site and protect the fetus and infants from known harm.

Bill Osmunson DDS MPH

From: Arne Christensen
Sent: 2/6/2024 1:21:14 PM
To: DOH WSBOH
Cc:
Subject: alleged Taiwan face mask death

External Email

You need to read this article from January, "Infant dies after allegedly suffocating on mask at New Taipei daycare":
https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Ffocustaiwan.tw%2Fsociety%2F2024

It begins: "Authorities in New Taipei on Wednesday said they are investigating the death of an 11-month-old boy at a public daycare center, which the child's family allege happened when he suffocated on a mask a teacher forced him to wear."

After reading it, do you still think face masks are just an inconvenience? I don't accept the reply that public health authorities never said infants should have to wear masks. Normalizing and requiring masks on toddlers was going to lead to requiring masks on infants somewhere in the world.

From: Garry Blankenship
Sent: 2/5/2024 8:15:15 AM
To: hcinfo.infosc@canada.ca,DOH WSBOH,dhsmoh@yahoo.com,secretary@health.gov.bz,Van De Wege, Kevin,Chapman, Mike (LEG),sheriff@co.clallam.wa.us,mozias@co.clallam.wa.us,rjohnson@co.clallam.wa.us,shahidafatin@gmail.c
Allison 2 (DOHi)
Cc:
Subject: The NOP BOH Needs Introspection

External Email

I do not doubt the BOH intentions, but recommending, promoting and mandating these mRNA injections was and remains a colossal mistake. Denying the naturally immune public access was worse. The Federal, State and local pandemic management record is without exception an abject failure. I request the Board make the effort to insure mistakes like this never repeat.

https://www.theepochtimes.com/health/for-every-life-saved-mrna-vaccines-caused-nearly-14-times-more-deaths-study-5579794?utm_source=Ccpv&src_src=Ccpv&utm_campaign=2024-02-05&src_cmp=2024-02-05&utm_medium=email&est=0Y%2F9GSyc74a%2FdwbERhO%2FTk2D8BeBhXgQlredhB%2Fte85A4PYzcUdI

<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.theepochtimes.com%2Fheal every-life-saved-mrna-vaccines-caused-nearly-14-times-more-deaths-study-5579794%3Futm_source%3DCcpv%26src_src%3DCcpv%26utm_campaign%3D2024-02-05%26src_cmp%3D2024-02-05%26utm_medium%3Demail%26est%3D0Y%252F9GSyc74a%252FdwbERhO%252FTk2D8BeBhXgQlredh

Sincerely,

Garry Blankenship

From: patrice tullai
Sent: 1/5/2024 6:34:20 PM
To: DOH WSBOH
Cc:
Subject: Racism is a public health crisis

External Email

Hello, and good day to you,
When I was a child all children played together no matter race or color or religion, the policies that are being inflamed are creating more division among people, not less. I see division and victim mentality being pushed to the forefront, this does not help our children, youth, or society, this is dividing people. We need to come together. The problems come from class ….the poor suffer. I would like to encourage you to not act under the idea, or create policies that racism is a public health problem ,
Thank you
I hope you and 2024 work to bring humanity together not divided,
Patrice Tullai
PateiceTullai@gmail.com

_____

From: DOH WSBOH
Sent: 3/8/2024 11:51:33 AM
To: DOH WSBOH
Cc:
Subject: FW: My Public Comments

Forwarding as this email has the same subject line as her email from 3/7 and the system would not accept a duplicate.

From: Melissa Leady <melleady@yahoo.com>
Sent: Friday, March 8, 2024 11:11 AM
To: DOH WSBOH <WSBOH@SBOH.WA.GOV>
Subject: My Public Comments

External Email

As part of the PEAR Plan Development, will the Department of Health (DOH) be conducting a pandemic policy review, looking at some of the unintended negative impacts of covid policies? Pandemic policy in Washington state disproportionately impacted lower-income families and people of color.

Loss of in-person learning at schools resulted in lower test scores. In Vancouver, for example, the city is providing $500,000 to the Vancouver Public School District to address covid learning loss at elementary schools in the Fourth Plain corridor. These are among the most ethnically diverse and economically challenged schools in the district. For the students in these schools, the cost of covid learning loss could be felt for their lifetimes, according to a UN study on children living in learning poverty.

Covid job loss also disproportionately impacted low wage jobs, as the "laptop class" quickly transitioned to working from home. At my last county board of health meeting, my local health director mentioned that the covid job loss often resulted in loss of health insurance. Has there been any assessment of the effects of pandemic policy-related job loss on access to healthcare?

During the pandemic, the public was told to isolate and parks and outdoor recreation were closed. The obesity rate in Washington state increased 2%. Obesity is closely linked to a wide variety of negative health outcomes, including diabetes, heart disease, cancer, and covid death. According to the CDC, the current obesity rates in Washington state by race are: 10% Asian, 30% white, 36% Black, 36% Hispanic, and 43% Native American. Will the PEAR Plan Development be looking at differing rates of obesity by race as part of their efforts to understand differing rate of covid deaths by race?

These are just a few examples. Other areas to explore include : impacts on small businesses and restaurants, school enrollment, mental health, anxiety, depression,

substance abuse, drug overdoses, domestic violence, housing and housing affordability, food insecurity, and loss of cultural events and religion gatherings.

In addition, has there been an assessment of the impacts of the Washington state vaccine mandate? A recent study comparing states with vaccine mandates and states banning vaccines mandates showed 1) no comparable difference in vaccine uptake; and 2) reduced rates of flu and booster uptake in states that imposed mandates.

Does DOH attribute the low 2023-2024 rates for flu vaccination (30%) and covid vaccination (18%) to "blow back" from the vaccine mandates? What was the impact of the mandates of jobs and healthcare? In Clark County, for example, there was a 10% drop in hospital beds after the mandate took effect, when some hospital staff chose to quit instead of getting vaccinated. Eventually that difference was made up by employing traveling nurses at an increased cost, driving up costs locally.

I hope that DOH will take the time to assess the "collateral damage" of covid policy decisions, as former NIH director Francis Collins recently termed it. Perhaps this could be done in conjunction with the PEAR Impact Assessment.

Sincerely,

Melissa Leady

Clark County Resident

From: Garry Blankenship
Sent: 3/2/2024 8:22:24 AM
To: Van De Wege, Kevin,Chapman, Mike (LEG),DOH WSBOH,sheriff@co.clallam.wa.us,mozias@co.clallam.wa.us,rjohnson@co.clallam.wa.us,shahidafatin@gmail Allison 2 (DOHi),Tharinger, Steve
Cc:
Subject: Higher Mortality In Vaxed Vs Unvaxed

External Email

Good Day All,

I have found any contra "vaccine" information, regardless of documentable verification, to be summarily dismissed by most medical practitioners, particularly those holding any authoritative position, with no effort to independently vet that information. No objectivity in vetting drug safety is a huge looming problem that will not go away. Confidence in our health care system has been critically damaged by a lack of acknowledging mistakes made in the "pandemic". It is clear that the medical community was given false information on the COVID "vaccines", treatment protocols and repurposed drugs, but the absence of acknowledging that will self destruct the medical complex. I implore you to stop pretending that promoting these mRNA platform injectable products was or is health positive. These drugs are killing the young and working aged disproportionately.

https://www.theepochtimes.com/health/study-finds-higher-mortality-among-vaccinated-patients-hospitalized-for-covid-19-post-5597490?utm_source=Ccpv&src_src=Ccpv&utm_campaign=2024-03-02&src_cmp=2024-03-02&utm_medium=email&est=AAAAAAAAAAAAAAAAaes4chIex8Tb%2F5MNsWhaCqduhU0LM8%2FAPpFNrub

<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.theepochtimes.com%2Fheal finds-higher-mortality-among-vaccinated-patients-hospitalized-for-covid-19-post-5597490%3Futm_source%3DCcpv%26src_src%3DCcpv%26utm_campaign%3D2024-03-02%26src_cmp%3D2024-03-02%26utm_medium%3Demail%26est%3DAAAAAAAAAAAAAAAAaes4chIex8Tb%252F5MNsWhaCqduhU0LM

Not seeking anonymity,

Garry Blankenship

From: Michelle Anderson
Sent: 2/1/2024 5:10:05 PM
To: DOH WSBOH
Cc:
Subject: Public Comments for the Environmental Health Committee

External Email

Dear Board.
I would just like to remind you that Mandatory COVID shots or testing is unacceptable!
It is now just another virus that we must all deal with!
Just like the FLU, Common Cold or any other Corona Virus (there are a bunch and tests don't tell you WHICH one it is)
We are ADULTS and we can make decisions for our own children!
Government mandates are unnecessary!
Thank you very much for all you do!

From: Garry Blankenship
Sent: 2/24/2024 7:40:04 AM
To: Van De Wege, Kevin,Chapman, Mike (LEG),DOH WSBOH,sheriff@co.clallam.wa.us,mozias@co.clallam.wa.us,rjohnson@co.clallam.wa.us,shahidafatin@gmail Allison 2 (DOHi)
Cc:
Subject: "Vaccine" Adverse Events

External Email

I can only hope those responsible for promoting and particularly mandating these toxins are held accountable. These injections violate informed consent and the Hippocratic Oath.

https://www.theepochtimes.com/health/a-host-of-notable-covid-19-vaccine-adverse-events-those-backed-by-evidence-5590525?utm_source=Health&src_src=Health&utm_campaign=health-2024-02-24&src_cmp=health-2024-02-24&utm_medium=email&est=AAAAAAAAAAAAAAAAaes4chIex8Tb%2F5MNsWhaCqduhU0LM8%2FAPpFNrub

_____

From: Stuart Halsan
Sent: 2/6/2024 8:07:49 PM
To: DOH WSBOH
Cc:
Subject: Communicating With Board Members

External Email

For Patty Hayes

I have some genealogical info for you.. You can reach out to me at this email.
Hope all is well.
Stuart Halsan
Sent via the Samsung Galaxy S9+, an AT&T 5G Evolution capable smartphone

_____

From: Karen Spencer
Sent: 3/8/2024 10:05:47 AM
To: DOH WSBOH
Cc:
Subject: Comment: Fluoridation Poisoning

External Email

"Fluoride is capable of producing any number of symptoms. They include drowsiness, profound desire to sleep, dizziness, nasal congestion, sneezing, runny nose, sore throat, coughing, wheezing (asthma), chest pain, hives, and various intestinal symptoms. Most of the information concerning specific reactions to fluoride, as seen in private practice, never reach publication." - Hobart Feldman, MD, American Board of Allergy and Immunology (1979)

Board of Health -

I signed up to make a comment on Wednesday March 13th, but may be unavailable at that time. Therefore, I am sending a written comment for your consideration:

MY PERSONAL STORY:

My name is Karen Spencer. I am a retired analyst and project management consultant who has worked with all levels of Corporate America.

I am angry about what happened to me and my children. I was poisoned by fluoridated water while pregnant in 1981. My normal pregnancy turned difficult overnight. I was ill with chronic dizziness, nausea, bloody stools and rashes beginning the first week of July. I didn't make the connection to water until much later. Fluoridation began on July 1st.

I did not recover after giving birth. Worse, both my children shared my symptoms. It took me until late 1982 to realize tap water was causing our rashes and gastrointestinal problems. My primary care physician who was the Chair of the Board of Health yelled me out of his office in November when I asked if the water could be making us sick. In January '83, an allergist specializing in environmental health recommended I only use spring water in glass bottles for all of our water needs, which alleviated our symptoms.

Since bottled water is expensive, I installed a high-quality under the sink filter in '91. I was diagnosed with Lyme disease about the same time, so I accepted my doctors attribution of my emerging and ongoing arthritis and neurological symptoms to Chronic Lyme. They also diagnosed me with irritable bowel syndrome. I was in my 30s. I developed kidney and liver problems in my 50s.

I switched back to bottled water in 2014 to see if it would have a positive effect on my declining health. It did— within days. My multi-stage system wasn't adequate and never had been. Can you imagine my outrage when I realized, in my 60s, that decades of arthritis, gastrointestinal illness, neurological issues and even concerns over organ failure had been fluoride poisoning?

There is no happy ending for me. The damage to my bones and spinal discs from decades of fluoride poisoning cannot be undone, and neither can the damage to my son who has learning disabilities consistent with what has been validated by developmental neurotoxicity studies.

The chair of my local board of health, a doctor, told me in 2014 that "they" knew some

people would have problematic symptoms from fluoridation, but it was a "greater good" to prevent a cavity or two in poor children. Please don't tell me that my life and the lives of my children are collateral damage. I suggest that ending fluoridation not only provides health equity for susceptible sub-populations, but also serves justice to the grandchildren of my baby-boomer generation who were poisoned by an ill-conceived, immoral medical mandate.

* CAPE ANN STORY WITH REFERENCES: https://fluoridealert.org/wp-content/uploads/SalemState2016.09.07.pdf <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Ffluoridealert.org%2Fwp-content%2Fuploads%2FSalemState2016.09.07.pdf&data=05%7C02%7Cwsboh%40sboh.wa.gov%7C684f7

* ANNOTATED SCIENCE BIBLIOGRAPHY: https://www.fluoridelawsuit.com/science <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.fluoridelawsuit.com%2Fscien

For more about me, see my signature.

Regards,

_____

Karen Favazza Spencer
Leominster, MA 01453
978.283.4606
Subscribe on YouTube <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.youtube.com%2Fchannel%2vZ55u7oKUchQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7C684f7f0a9660495d62fd08dc3f99f847%7C

See the Call to Action <https://gcc02.safelinks.protection.outlook.com/?url=http%3A%2F%2Fwww.greenmedinfo.com%2Fblog%letter-nutritionists-about-fluoride-deception&data=05%7C02%7Cwsboh%40sboh.wa.gov%7C684f7f0a9660495d62fd08dc3f99f847%7C11d0e

More power to you if fluoridation doesn't bother you, but not the power to assume it's safe for your neighbor with kidney disease, his pregnant wife or their diabetic daughter!

About Karen: Currently a semi-retired consultant working with software development teams, Karen Spencer is a former analyst and project leader. She is adept at conducting research and analyzing trends. Her special interests include critical thinking, data-driven decision making, and organizational theory. She and others in her family are among the 15% of Americans with chemical sensitivities triggered by exposure to fluoridated food and drink. Karen's publications were featured in:
Medical Hypotheses (2018): https://pubmed.ncbi.nlm.nih.gov/30396472/ <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fpubmed.ncbi.nlm.nih.gov%2F303

GreenMed (2019): https://www.greenmedinfo.com/blog/wetoo-medical-assault-and-battery <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.greenmedinfo.com%2Fblog%medical-assault-and-battery&data=05%7C02%7Cwsboh%40sboh.wa.gov%7C684f7f0a9660495d62fd08dc3f99f847%7C11d0e2

Gloucester Times (2022): https://www.gloucestertimes.com/opinion/column-stop-poisoning-gloucester/article_0089c49c-1278-11ed-8a42-fb294218a4fe.html <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.gloucestertimes.com%2Fopistop-poisoning-gloucester%2Farticle_0089c49c-1278-11ed-8a42-fb294218a4fe.html&data=05%7C02%7Cwsboh%40sboh.wa.gov%7C684f7f0a9660495d62fd08dc3f99f847

Message to CDC (2022): https://www.youtube.com/watch?v=PzviupO1cDQ
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.youtube.com%2Fwatch%3Fv

Collaborative Activism (2022-current): https://www.fluoridelawsuit.com/actions
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.fluoridelawsuit.com%2Factio

Bill in MA Legislature (2023): https://malegislature.gov/Bills/193/S460
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fmalegislature.gov%2FBills%2F19

Document Fraud at CDC (2024):
https://www.researchgate.net/publication/377152337_Document_Fraud_at_CDC
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.researchgate.net%2Fpublica

From: Garry Blankenship
Sent: 2/17/2024 10:31:07 AM
To: hcinfo.infosc@canada.ca,DOH WSBOH,OADS@cdc.gov,sheriff@co.clallam.wa.us,Van De Wege, Kevin,Chapman, Mike (LEG),mozias@co.clallam.wa.us,rjohnson@co.clallam.wa.us,shahidafatin@gmail.com,gbsjrmd@sisna.com,n
Allison 2 (DOHi)
Cc:
Subject: Vaccine Shedding

External Email

Fascinating article and video on shedding. The probability of vaccinated people shedding spike proteins on other people is very real.

https://www.theepochtimes.com/health/covid-vaccine-shedding-is-real-fda-and-pfizer-documents-are-proof-clinicians-5588819?utm_source=Health&src_src=Health&utm_campaign=health-2024-02-17&src_cmp=health-2024-02-17&utm_medium=email&est=AAAAAAAAAAAAAAAAaes4chIex8Tb%2F5MNsWhaCqduhU0LM8%2FAPpFNrub

_____
From: Cheryl Lewis
Sent: 1/23/2024 7:57:12 AM
To: DOH WSBOH
Cc:
Subject: Communicating With Board Members

attachments\B7B9A7277A0E4E2B_Sledge - BOH Strategies.pdfDept o_PRDTOOL_NAMETOOLONG.pdf

External Email

Microsoft Edge - ready to share - Presentation and 6 more pages - Personal - Microsoft Edge - 15 January 2024 - Watch Video
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.loom.com%2Fshare%2F8bc0

<https://cdn.loom.com/sessions/thumbnails/8bc09cd7d30146e6a46991886f25c8c8-00001.jpg>
healthcare hygienist!

Hello All

I am a dental hygienist who would love to see an improvement in oral care for our community. I believe there are many ways to improve this and ran across this publication on your site (it is at the bottom of the page). It seems to be dated 2013. I am wondering how far we have come since then? I have created a presentation that I would like to share with you. It is about 30 minutes long and I feel it promotes your cause in a different light. I would be honored if you would review it and allow me to be a resource to you in this arena. I have a deep desire to improve the oral care of our facility residents, from the hospital to the long term care facilities. I believe dental hygienist's should be employed as a member of each of these facilities as oral care specialists, not to perform traditional dental cleanings but to improve daily oral care which will improve quality of life. Having a hygienist visit a facility every 3-6 months isn't helping people keep their mouths healthy. Please watch my presentation to gain insight on this. I think we should at the very least, create a certification for caregivers, one that specializes in oral care. Maybe they could have increased training on oral diseases to look for (cancer, gum disease, cavities, dry mouth sores, abscesses). Special training on treatment and prevention of caries and gum disease. This distinction could create value of the caregiver and maybe that could translate to an increase in their wage, which may lead to retention, maybe decrease turnover? If there was a team or even an individual in charge of oral care and only oral care, our dependents would not suffer with dry mouth sores and bleeding gums. Oral care is often the first area to be neglected and a visit from the hygienist 2 times a year is not the way to maintain oral health. We are learning more and more about the bacteria's role in our health and allowing plaque (bacteria) and food to linger for days, weeks, months is not promoting health. Often oral care is left to the resident, unless it is noted on the residents care plan to brush for them.

I know you are busy, but please take a moment (30 minutes or so ☐☐) and consider the change that could be made. It's like a child who drowns in the swimming pool, when everyone is watching, no one is watching. We need a go to, a someone in charge of daily oral hygiene to ensure people are receiving the care they need and deserve. This would not only reduce risk of cavities and gum disease, but aspirated pneumonia, sepsis, and death as well.

I am trying to make change starting at the top (you).

I look forward to hearing from you and thank you sincerely for taking the time to consider this.

Sent from Mail <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fgo.microsoft.com%2Ffwlink%2F% for Windows

From: Cheryl
Sent: 1/30/2024 6:08:46 AM
To: DOH WSBOH
Cc:
Subject: Communicating With Board Members

attachments\88C9EC27E025473E_my presentation (1).htm

attachments\5B71ED4076E84D7A_Sledge - BOH Strategies.pdfDept o_PRDTOOL_NAMETOOLONG.pdf

External Email

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Best

Cheryl lewis RDH

Sent from Mail
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fgo.microsoft.com%2Ffwlink%2F%
for Windows

_____

From: bill teachingsmiles.com
Sent: 2/29/2024 7:31:08 AM
To: DOH WSBOH
Cc:
Subject: March 13 Public Comment

External Email

Dear Washington State Board of Health,

The Legislature has made one of the duties of the Board of Health to assure drinking water is safe, because water is essential for life.

The Legislature does not say the duty is to assure efficacy, because that’s the duty of the FDA.

Fluoridation of public water is not safe because, not once did the EPA expert scientists during the two-week trial before the Superior Court of California (January and February 2024) testify that fluoridation was safe, or effective.

Fluoridation of public water is not safe because, it is a highly toxic contaminated scrubbings of manufacturing, a poison, a prescription drug, not FDA approved, misbranded and adulterated.

Fluoridation is not safe because, it violates an individual’s consent, freedom to choose, and their doctor's oversight.

Fluoridation is not safe because, fluoride causes dental fluorosis. I, and most dentists, each made and make hundreds of thousands of dollars treating cosmetic and functional dental fluorosis, harm.

Fluoridation is not safe because, fluoride ingestion increases developmental neurotoxicity as measured with lower IQ. Lower IQ increases the rate of special education in schools, lower wage jobs, more unemployment, more divorce, more incarceration, more grief, fewer gifted, and is bad for America, especially minorities.

Fluoridation is not safe because, fluoride ingestion harms the developing fetus, infant and child as measured with increased miscarriage, increased premature birth, and increased infant mortality.

Fluoridation is not safe because, fluoride ingestion is stored in the bones and as the bones remodel the fluoride is given off. Mother's blood concentration of fluoride in the third trimester increases when she has inadequate intake of calcium for her fetus's needs.

Fluoridation is not safe because, fluoride ingestion harms the joints causing rheumatoid and osteoarthritic-like pain.

Fluoridation is not safe because fluoride ingestion harms the thyroid and is an endocrine disruptor, increasing diabetes, obesity and ADHD.

Fluoridation is not safe because fluoride ingestion increases osteosarcoma a rare but lethal bone cancer, mostly in boys drinking fluoridated water during growth spurts.

Fluoridation is not safe because fluoride ingestion harms the kidneys and GI disorders.

Do not let the fluoridation lobby confuse you. The Board's job is to assure safety. The dental lobby's job is to gain FDA CDER approval. They have failed, but you must not.

We look forward to participating in a forum on fluoride ingestion because we and many are being harmed.

Bill Osmunson DDS MPH

Washington Action for Safe Water

_____

From: Melissa Leady
Sent: 3/7/2024 6:13:04 PM
To: DOH WSBOH
Cc:
Subject: My Public Comments

External Email

IS THE CURRENTLY AUTHORIZED COVID-19 VACCINE EFFECTIVE?

During a recent county board of health meeting, the health director for my county made the claim that there is state data showing that the updated covid-19 vaccine is effective at preventing severe illness, hospitalizations, and deaths; and that it is effective at preventing infection and thereby transmission.

It seems my local health director is out on a limb in making this claim. If DOH has such data on the updated covid-19 vaccine, they have never publicly shared it.

The DOH report on Hospitalizations and Deaths by Vaccination Status (#421-010), which hasn't updated in three months, begins by stating, "PLEASE NOTE: Information about bivalent booster doses (authorized in the fall of 2022) or the updated monovalent booster doses (authorized in September of 2023) is not included in this report."

Is the board recommending the currently authorized updated covid-19 vaccine? If so, do you have Washington state data showing the vaccine's effectiveness? Please share it with the public.

Florida State Surgeon General Calls for Halt in the Use of COVID-19 mRNA Vaccines

Contact:
Communications Office
***NewsMedia@flhealth.gov**,*
850-245-4111

Tallahassee, Fla.— On December 6, 2023, State Surgeon General Dr. Joseph A. Ladapo sent a letter to the United States Food and Drug Administration (FDA) Commissioner Dr. Robert M. Califf and Center for Disease Control and Prevention (CDC) Director Dr. Mandy Cohen regarding questions pertaining to the safety assessments and the discovery of billions of DNA fragments per dose of the Pfizer and Moderna COVID-19 mRNA vaccines.

The Surgeon General outlined concerns regarding nucleic acid contaminants in the approved Pfizer and Moderna COVID-19 mRNA vaccines, particularly in the presence of lipid nanoparticle complexes, and Simian Virus 40 (SV40) promoter/enhancer DNA. Lipid nanoparticles are an efficient vehicle for delivery of the mRNA in the COVID-19 vaccines into human cells and may therefore be an equally efficient vehicle for delivering contaminant DNA into human cells. The presence of SV40 promoter/enhancer DNA may also pose a unique and heightened risk of DNA integration into human cells.

In 2007, the FDA published guidance on regulatory limits for DNA vaccines in the Guidance for Industry: Considerations for Plasmid DNA Vaccines for Infectious Disease Indications (Guidance for Industry). In this Guidance for Industry, the FDA outlines important considerations for vaccines that use novel methods of delivery regarding DNA integration, specifically:

- DNA integration could theoretically impact a human's oncogenes – the genes which can transform a healthy cell into a cancerous cell.
- DNA integration may result in chromosomal instability.
- The Guidance for Industry discusses biodistribution of DNA vaccines and how such integration could affect unintended parts of the body including blood, heart, brain, liver, kidney, bone marrow, ovaries/testes, lung, draining lymph nodes, spleen, the site of administration and subcutis at injection site.

On December 14, 2023, the FDA provided a written response providing no evidence that DNA integration assessments have been conducted to address risks outlined by the FDA themselves in 2007. Based on the FDA's recognition of unique risks posed by DNA integration, the efficacy of the COVID-19 mRNA vaccine's lipid

nanoparticle delivery system, and the presence of DNA fragments in these vaccines, it is essential to human health to assess the risks of contaminant DNA integration into human DNA. The FDA has provided no evidence that these risks have been assessed to ensure safety. **As such, Florida State Surgeon General Dr. Joseph A. Ladapo has released the following statement:**

“The FDA’s response does not provide data or evidence that the DNA integration assessments they recommended themselves have been performed. Instead, they pointed to genotoxicity studies – which are inadequate assessments for DNA integration risk. In addition, they obfuscated the difference between the SV40 promoter/enhancer and SV40 proteins, two elements that are distinct.

DNA integration poses a unique and elevated risk to human health and to the integrity of the human genome, including the risk that DNA integrated into sperm or egg gametes could be passed onto offspring of mRNA COVID-19 vaccine recipients. If the risks of DNA integration have not been assessed for mRNA COVID-19 vaccines, these vaccines are not appropriate for use in human beings.

Providers concerned about patient health risks associated with COVID-19 should prioritize patient access to non-mRNA COVID-19 vaccines and treatment. It is my hope that, in regard to COVID-19, the FDA will one day seriously consider its regulatory responsibility to protect human health, including the integrity of the human genome.”

In the spirit of transparency and scientific integrity, State Surgeon General Dr. Joseph A. Ladapo will continue to assess research surrounding these risks and provide updates to Floridians.

About the Florida Department of Health

The Florida Department of Health, nationally accredited by the Public Health Accreditation Board, works to protect, promote and improve the health of all people in Florida through integrated state, county and community efforts.

Follow us on Twitter at @HealthyFla and on Facebook. For more information about the Florida Department of Health please visit www.FloridaHealth.gov.

WASHINGTON STATE BOARD OF HEALTH

Working for a safer and healthier Washington since 1889

Recommended Strategies to Improve the Oral Health of Washington Residents

Presented by
Dr. Jim Sledge

Background

Board of Health Addresses Oral Health

- June 2012 – Briefing – Oral Health Risk Factors and Systemic Connections
- October 2012 – Briefing – Oral Health in Washington State
- March 2013 – Board approves implementation of the Oral Health Project
- November 2013 – Board approves the Oral Health Strategy
- Summer 2014 – Board held interagency Oral Health workshop
- April 2015 – Board approves the workshop final report

WASHINGTON STATE BOARD OF HEALTH
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Oral Health Project

- **Goal**
  - Create a Washington State Board of Health set of strategies to improve the oral health of Washington State residents

- **Purpose:**
  - To promote strategies that improve the oral health of Washington residents
  - To guide Washington State Board of Health (SBOH) rule and policy development activity

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Rationale

Oral diseases are costly, painful, debilitating, and widespread in Washington State

- Dental disease is the most common chronic disease of childhood (NHNES)
  - Nearly 40% of kindergarteners in WA have had tooth decay
  - 77% of WA's Native American kindergarteners have had tooth decay – Washington State Smile Survey 2010
- Nationally:
  - More than 51 million school hours are lost each year to dental-related issues.
  - Adults lose more than 164 million hours of work due to dental health issues – Report of the Surgeon General, 2000

WASHINGTON STATE BOARD OF HEALTH
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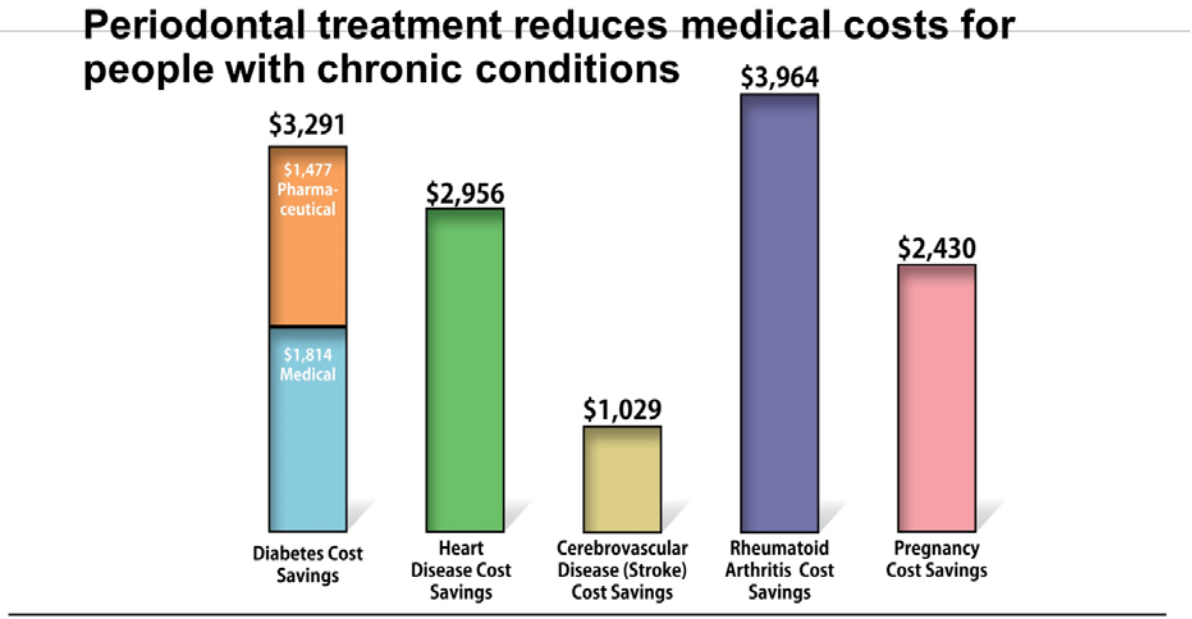
Rationale

- Poor oral health is costly for Washington residents:
  - Dental pain is the number one reason uninsured adults visited Washington state emergency rooms
  - Dental-related Emergency room charges were over $36 million in an 18 month period – Washington State Hospital Association, 2010
- Oral infections are also associated with systemic conditions such as diabetes, heart disease, and aspiration pneumonia

Rationale

- Strategies that prevent and treat dental disease improve oral health and save money



Study Conducted by University of Pennsylvania, School of Dental Medicine for United Concordia Dental

Rationale

- In Washington, adults aged 55 years and older rank higher than the national average when it comes to dental insurance
  - However, 20% of adults ages 55 and older reported having a dental issue that needed to be addressed in the next month
  - Nearly 24% of seniors with an annual income under $25,000 have not seen a dentist in five years or more –

    2012 Older Adult Oral Health Survey, Washington Dental Service Foundation

- Older adults are particularly at-risk due to taking multiple medications that cause dry mouth and can lead to tooth decay

Oral Health Project –Methods

- Reviewed literature
- Drafted strategic recommendations
- Shared proposed strategies with State expert review panel – updated recommendations
- Consulted with National oral health expert advisory group – updated recommendations

Strategic Recommendations

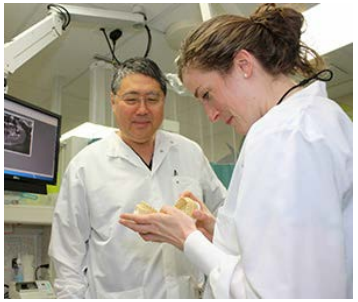
Topic Areas

- Health Systems
- Community Water Fluoridation
- Sealant Programs
- Interprofessional Collaboration
- Oral Health Literacy
- Surveillance
- Work Force

(not ranked in order of importance)

Health Systems

- Cost-effective programs allow more people to get the services they need at affordable rates


- **Recommendation**: Support policies and programs that improve oral health for Washington state residents

Health Systems

Programs working for Washington:

- Adult Medicaid coverage restored
  - Over 750,000 adults will gain dental coverage in 2014
- Access to Baby and Child Dentistry (ABCD)
  - Connects Medicaid enrolled children with dental services
  - The program now operates in all 39 Washington counties
  - Percentage of low-income children accessing dental care has more than doubled since 1997- to 51%
- University of Washington Regional Initiatives in Dental Education (RIDE)
  - Over half of the graduates now work in dental underserved regions of the state

Health Systems

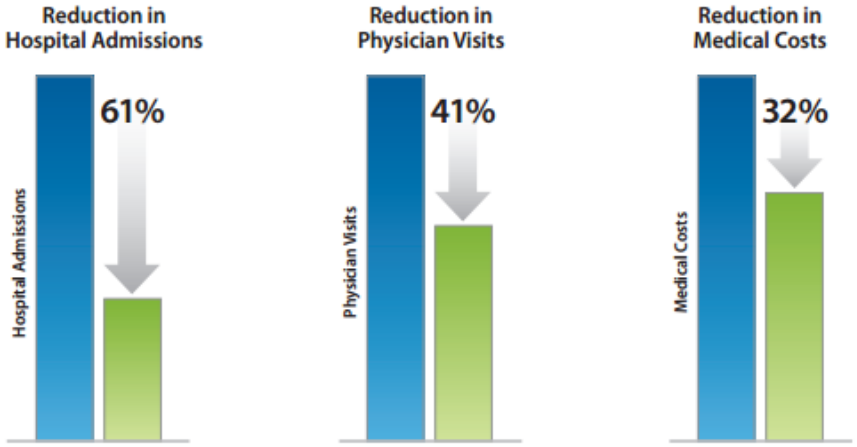
Opportunities remain:

- Evaluate methods to ensure adequate access to treatment and prevention services with particular attention to:
  - Pregnant women
    - Over 50% of women born in Washington state 2010 were on Medicaid
    - Mothers with healthy teeth are less likely to pass cavity causing bacteria to their children
  - Low-income populations
    - With new coverage available to adults there will be an increasing demand for services

Health Systems

Opportunities remain:

- Diabetes and oral health
  - Collaborate to improve outcomes for people with diabetes



Jeffcoat M., et. al, Periodontal Therapy Reduces Hospitalization and Medical Costs in Diabetes, Abstract, American Association of Dental Research, March 23, 2012

Community Water Fluoridation

- Access to community water fluoridation benefits the health of everyone: children, adults, and seniors



- **Recommendation:** Expand and maintain access to community water fluoridation

Community Water Fluoridation

- CDC has recognized water fluoridation as one of 10 great public health achievements of the 20th century
- 65% of Washington's residents on public water supplies receive optimally fluoridated water as compared to 74% nationally
- Washington ranks 35th in the nation for communities receiving fluoridated water

Community Water Fluoridation

- For water systems serving 20,000 people or more, every $1 invested in fluoridation saves $38 in dental treatment costs
- Water fluoridation reduces tooth decay by about 25 percent over a person's lifetime
- Community water fluoridation is safe. After 65 years in service and hundreds of studies it continues show its safety
- Water fluoridation reduces the disparities in tooth decay rates that exist by race, ethnicity and income

Sealant Programs

- Children with fewer cavities are healthier and better able to learn, grow, and thrive

- **Recommendation**: Provide school-age children with access to dental sealants to prevent cavities

Sealant Programs

- Dental sealants are placed on chewing surfaces to create a barrier between teeth and decay-causing bacteria
- The CDC's Task Force on Community Preventive Services (2002) found that school sealant programs are highly effective at preventing tooth decay
- According to the Surgeon General's Report on Oral Health (2000), sealants have been shown to reduce decay by more than 70% and are most cost-effective when provided to children who are at highest risk for tooth decay
- In Washington, the Smile Survey found that 51 percent of third grade children have received sealants

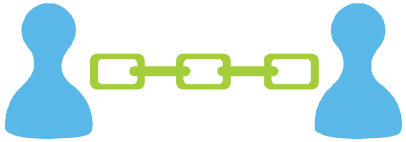
Interprofessional Collaboration:

- Collaboration between health professions and systems improves patient care



- **Recommendation**: Incorporate oral health improvement strategies across healthcare professions and systems to improve oral health knowledge and patient care

Interprofessional Collaboration:



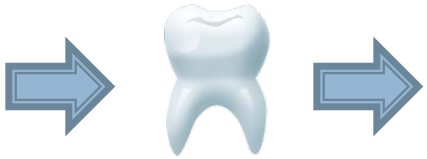
- Dental diseases are highly prevalent, yet largely preventable
- Clear links exist between oral health and chronic conditions, including diabetes and cardiovascular disease
- Interprofessional Collaboration is supported by research from the Institute of Medicine to improve patient care

Interprofessional Collaboration:

- Develops professionals who work together towards a common goal of optimizing patient care
- Fosters structures that support collaboration

Trained collaborative Dental, Medical, & Allied Professionals

Improved Understanding



A focus on high risk groups

- Diabetes
- Pregnancy
- HIV/AIDS
- Pneumonia

Improved outcomes & reduced treatment costs for Washington residents

Interprofessional Collaboration:

- Medical providers have regular consistent contact with patients
  - Already doing prevention and looking in the mouth
  - Well-positioned to address oral health
- The National Interprofessional Initiative on Oral Health 2012 Report compared 4 states
  - Two-thirds of Washington programs included some oral health material

Physician Curriculum by State	Includes Oral Health
Colorado	40%
New York	29%
Virginia	62%
Washington	67%

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Oral Health Literacy

- Clear and accessible oral health information empowers people to make good choices for themselves and their families

- **Recommendation**: Improve the capacity of people to obtain, understand, and use health information in order to increase their acceptance and adoption of effective oral health focused preventive practices

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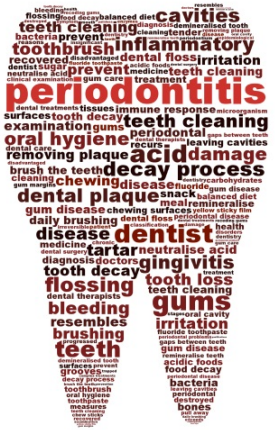
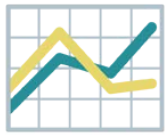
Oral Health Literacy

- Oral health literacy represents the capacity of people (individuals and policy makers) to obtain, understand, and use health information in order to make correct decisions – US National Oral Health Alliance
  - In 2006, only 12% of the US population had proficient health literacy
  - People with low health literacy have adverse health outcomes
  - Parental health literacy impacts the health outcomes of their children



Surveillance

- Understanding the burden of oral disease for Washington residents allows programs to identify needs, work to achieve the biggest impact and measure progress and success



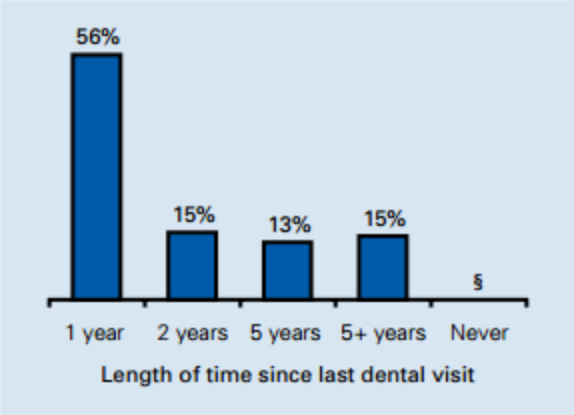
- **Recommendation**: Monitor trends in oral health indicators to ensure policies and programs are advancing the oral health of Washington residents, including those most at risk for poor oral health outcomes

Surveillance

Sustain Data-based monitoring and decision making tools, like:

- The Impact of Oral Disease on the Lives of Washingtonians
  - Report provides an overview of the burden of dental diseases on all Washingtonians
  - Compares WA to nationally comparable objectives
  - Includes data from the Behavioral Risk Factor Surveillance System (BRFSS) and the Washington State Oral Disease Surveillance System
  - Published by the Department of Health, Oral Health Program

Figure 30: Dental visits among adult smokers, 2004 WA BRFSS.



Note: § – Numbers too small to report.

Surveillance

Maintain the Washington State Smile Survey for pre-school and elementary school children

- Washington State Smile Survey:

- Assesses the oral health of school children every five years
- Provides benchmarks to compare with the Centers for Disease Control's Health People 2020 goals for oral health
- Completed in partnership with:
  - Washington State Department of Health
  - Washington Dental Service Foundation
  - Washington State Department of Early Learning
  - Superintendent of Public Instruction

Surveillance

- Incorporate oral health measures in surveillance tools, BRFSS, Pregnancy Risk Assessment Management (PRAMS), etc.
- Develop and implement surveillance systems for vulnerable populations, e.g. Medicaid
- Maximize community data sources:
  - Dental Workforce Report, Washington State Dental Association, 2012
  - Oral Health Senior Survey, Washington Dental Services Foundation, 2012
  - Emergency Room Use Report, Washington State Hospital Association, 2010

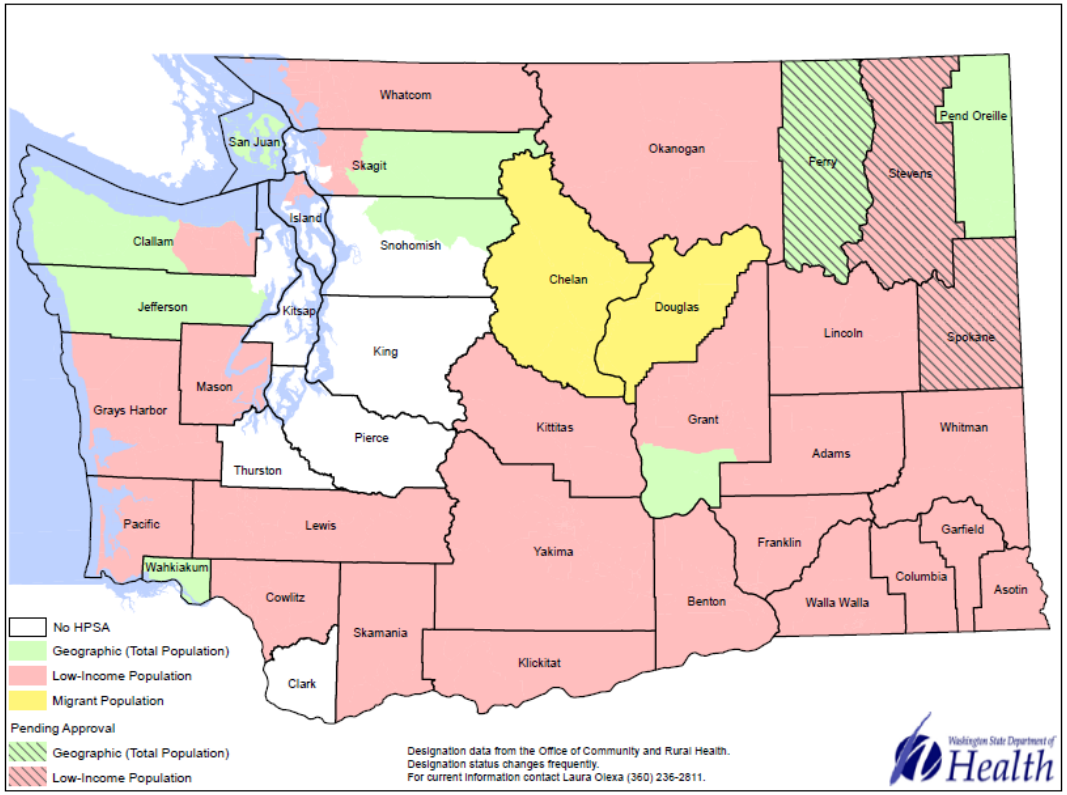
Work Force

- Health disparities decrease when all Washington residents are able to access dental care

- **Recommendation**: Develop health professional policies and programs which better serve the dental needs of underserved populations

Work Force

- Federally Designated Health Professional Shortage Areas for Dental Care, July 2013



Work Force

- Find Opportunities to develop a workforce that provides care to the dental underserved regions in our state
  - Partner with academic institutions
  - Recruit professionals:
    - From communities that face the highest incidence of tooth decay
    - To serve populations that currently lack access to dental services, including:
      - Rural communities
      - Low-Income families
      - Communities of color

Summary

SBOH Strategic Recommendations on Oral Health:

- Improve Health Systems
- Expand Community Water Fluoridation
- Promote Sealant Programs
- Build Interprofessional Collaboration
- Improve Oral Health Literacy
- Sustain Surveillance programs
- Develop Work Force

Questions?

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From: Christi Ellefson
Sent: 1/17/2024 11:59:52 AM
To: DOH WSBOH
Cc:
Subject: Important vaccine information


attachments\55A4B10E46FE4306_20240103-halt-use-covid19-mrna-vaccines.pr.pdf

External Email

https://www.floridahealth.gov/_documents/newsroom/press-releases/2024/01/20240103-halt-use-covid19-mrna-vaccines.pr.pdf <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.floridahealth.gov%2F_docum
releases%2F2024%2F01%2F20240103-halt-use-covid19-mrna-vaccines.pr.pdf&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Cfabdfcb1158547af321008dc17965613%7C

From: Scott Shock
Sent: 1/7/2024 2:07:20 AM
To: DOH Secretary's Office,DOH Office of the Chief of Staff,DOH Office of Innovation and Technology,DOH Office of Prevention Safety and Health,DOH Office of Strategic Partnerships,DOH Office of Health and Science,DOH Office of Public Affairs and Equity,DOH OS Executive Office of Policy Planning and Evaluation,DOH Office of Resilience and Health Security,DOH WSBOH,AGOOmbuds@atg.wa.gov,Ferguson, Bob (ATG)
Cc:
Subject: Call for a Halt to the Use of COVID-19 mRNA Vaccines

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External Email

I'm still looking forward to responses on what actions the WSDOH, WSBOH, and WA AG are taking to protect the people of Washington State against these unsafe products, and to gain justice for those injured by these products. Here is more for your consideration.

<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fopen.substack.com%2Fpub%2Fst
summary-of-the-evidence-
against%3Fr%3Dtaogl%26utm_campaign%3Dpost%26utm_medium%3Demail&data=05%7C02%7Cwsboh

A summary of the evidence against the COVID vaccines
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fopen.substack.com%2Fpub%2Fst
summary-of-the-evidence-
against%3Fr%3Dtaogl%26utm_campaign%3Dpost%26utm_medium%3Demail&data=05%7C02%7Cwsboh

open.substack.com
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summary-of-the-evidence-
against%3Fr%3Dtaogl%26utm_campaign%3Dpost%26utm_medium%3Demail&data=05%7C02%7Cwsboh

Here is a short list of reasons that everyone should be concerned about the COVID vaccine. This is not an exhaustive list.

1. Doctors are told to trust the FDA and CDC, but not verify, when prescribing vaccines. All the post-marketing safety data is kept hidden by health authorities so not even doctors can look at the data themselves to find out if any vaccine is safe. Doctors

have to trust the authorities. They are essentially told: “trust, do not verify.”

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9bd9-48d4-b525-
567127205c25%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7

2. The CDC itself doesn’t have the data to make a post-marketing independent vaccine safety assessment and they are not interested in obtaining the data either!The CDC relies on the FDA who relies on the manufacturer to test the product. The CDC could ask states for vaccination records tied to death records, but they don’t want to even ask because if they did an analysis, it could be discovered in a FOIA request. The CDC basically has no interest whatsoever in verifying what the actual safety data is.

3. Lack of transparency by health authorities. Not a single health authority anywhere in the world has ever released anonymized record-level patient data for independent researchers to assess the safety of any vaccine. There isn’t any paper in a peer-reviewed journal showing that health outcomes are improved if public health data is kept secret.

4. Lack of interest in data transparency by the medical community. Can you name a single high-profile pro-vaccine member of the medical community who has called for data transparency of public health data? Time-series cohort analyses can be easily produced by health authorities and published for everyone to see. These would show safety signals and do not jeopardize patient privacy. These are all kept hidden.

5. We aren’t allowed to see even the simplest of charts. Wouldn’t it be great to define two cohorts on July 1, 2021: COVID vaccinated vs. COVID unvaccinated. Then you simply record the deaths from that point forward and plot them. Why isn’t this being published?

6. Misinformation is deemed to be a problem, but the people making these statements are unwilling to take any steps to stop the so-called misinformation. These steps include: open public discussion to resolve differences of opinion and making public health data available/public in a way that preserves privacy. For example, HHS (as well as every state health department) should welcome all of us with open arms and invite us to query their databases (such as VSD and Medicare in the case of HHS) and publish whatever we find. Why does this information need to be hidden? The numbers tell the story, not the individual records.

7. No response from health authorities to reasonable requests. I’ve sent emails to Sarah Caul of the UK ONS on four ways the ONS can increase data transparency. There was no response.

8. No response when asked to explain damaging evidence. When credible scientists receive government data that shows very troubling safety signals, there is a total unwillingness of any health authority to discuss the matter and resolve it.

9. The US Medicare data clearly shows mortality increases after people take the jab. Is there any epidemiologist who can explain why deaths rose during a period in time when they should have been falling (per the Medicare death data)?

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1f9b1b3fd807%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7

For the first 120 days after the shots given in March 2021, death rates overall were falling. But if you got the vaccine, your death rates went up. We know from data from other vaccines that the baseline death rate of 81-year olds in Medicare is 3.85%, so the baseline death rate of this group is <800 deaths a day. These deaths climb far above baseline after you took the COVID shot.

10. The patient-level data released from NZ data confirms that mortality increases after the shots are given despite the fact that most of the shots were given during time periods when deaths were falling

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6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7

NZ data: Doses 2 and 4 were given while background mortality was falling, dose 3 while rising. So we'd expect the slope to fall in the first 6 months after vaccination. It does the opposite.

11. Anecdotes such as the one from Jay Bonnar
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6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
who lost 15 of his DIRECT friends unexpectedly since the shots rolled out. Four of the 15 died on the same day as that vaccine was given. Before the shots rolled out, Jay had lost only one friend unexpectedly. The probability this happened by chance is given by poisson.sf(14, .25) which is 5.6e-22. So this can't happen by chance. SOMETHING killed Jay's friends and 4 of the 15 died on the same day as they were vaccinated. Is there a more plausible explanation for what killed Jay's friends? All of them who died were vaccinated with the COVID vaccines.

12. Well done studies like the one done by Denis Rancourt
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99237cda9de5%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
showing 1 death per 800 shots on average. Jay Bonnar estimates he has around 14,000 friends so Jay's numbers are consistent with Rancourt's results.

13. Survey data like Skidmore

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6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
and Rasmussen Reports
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422a5362a138%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
showing that hundreds of thousands of Americans have been killed by the COVID shots. There have never been any counter surveys published showing this not to be the case.

14. The lack of any success stories. It appears that "vaccine success stories" where COVID infection fatality ratios dropped or that myocarditis cases plummeted do not exist. The US Nursing home data
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2F6fa3
b881-48d4-ab92-
b38a07406cd8%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
shows that the infection fatality rate (IFR) increased after the vaccine rolled out. There is nobody using that data making the claim it reduced the IFR.

15. Anecdotes from healthcare are extremely troubling. One nurse
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2Fe9b
6f4c-4e32-b913-
8a8057b3f865%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
reported a hospital admission rate that was 3X higher than anything in the 33-year history of the hospital after the COVID vaccines rolled out. Symptoms rarely ever seen were common after vaccines rolled out in that age group.

16. Lack of autopsies in clinical trials and post-marketing. The CDC doesn't request anyone to do autopsies even for people who die on the same day as they got the vaccine. Don't they want to know what killed those people… just to be sure?

17. Young people dying in sleep. There are way too many cases of young people who die in their sleep after being vaccinated. Doctors say this is a rare event. Now it is much more common. If the shots are safe, why is this happening?

18. I have direct personal experience with the vaccine: two people I know were killed by the vaccine, none from COVID. I know many people who are vaccine injured from the COVID vaccine.

19. Ed Dowd's book statistics. This very popular book ("Cause Unknown"
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2F15c
c0d0-49b8-b054-
b3c76b9b9d2b%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-
6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7
) listed 500 who died unexpectedly. Ed didn't know how many were unvaccinated. Only one person has come forward saying that one of the people in the book who died after the vaccines rolled out was unvaccinated.

20. Prominent doctor/scientists switching sides. Paul Marik is one of the top intensivists in the world. After seeing many COVID vaccine injured patients, he changed his mind about the safety of vaccines. When he was not allowed to practice medicine consistent with his Hippocratic Oath, he resigned his position.

21. The corruption with COVID protocols. The COVID hospital protocols likely caused

90% of the COVID deaths in hospitals. This led to Paul Marik resigning. See details in this article <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2Fe9b6f4c-4e32-b913-8a8057b3f865%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7. Why are doctors forced to use hospital protocols that kill a huge percentage of patients instead of using their best judgment to save patients?

22. This JAMA paper <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fsubstack.com%2Fredirect%2Fb692677-4b5b-a000-97b2419f3cd4%3Fj%3DeyJ1IjoidGFvZ2wifQ.6dRNrWa0LFC4PLtNGoJqvfmMd1pcH-6zh1fnBKsOnmQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7Caacf7d1c246240cc3f3408dc0f6811e4%7 shows that COVID and influenza vaccines don't work. Why are we pushing a vaccine where the statistics clearly show the vaccines don't work?

23. The consistency of the data. There have been no counter-anecdotes showing the vaccines are safe. I keep looking for one and come up empty.

24. No debates with anyone prominent promoting the government narrative.Those who promote the narrative refuse to engage in any scientific discussions to resolve differences of opinion. This is similar to the question of whether vaccines cause autism: nobody who thinks it doesn't is willing to engage in a public discussion about it to discuss the evidence. Why not resolve the issue through dialog? It isn't resolved in the peer-review literature where half the papers say vaccines cause autism and the other half don't. Why can't we talk about it?

25. Fear and intimidation tactics are used to silence dissent. Open debate would be more productive. But people are not allowed to hold or discuss views that go against the "consensus" or they will lose their jobs, their certifications, or their medical licenses. Health care workers are told they will be fired if they report an adverse event to VAERS, there are nurses who won't talk about anaphylaxis after getting the vaccine for fear of being fired, vaccine injuries are covered up, hospital workers are afraid to talk about it at work.

26. The cognitive dissonance is very disturbing. When healthcare workers bring up the topic of mortality and morbidity due to the vaccine, their peers say nothing and walk away.

27. Censorship tactics employed by the US government to silence dissent instead of public recorded open debates. History has shown that purveyors of censorship are always on the wrong side of the issue.

Scott

On Jan 4, 2024, at 1:11 AM, SCOTT SHOCK <ssshock@comcast.net> wrote:

☐
Dear WSDOH and WSBOH Members, and Attorney General's Office,

The Florida State Surgeon General has been a leader in protecting the people of his state against the unsafe mRNA COVID vaccinations. What actions are the WSDOH, WSBOH, and WA AG taking to protect the people of Washington State against these unsafe products, and to gain justice for those injured by these products (including members of

my family)? I look forward to your responses.

Scott Shock
Seattle

Joseph A. Ladapo, MD, PhD on X: "I am calling for a halt to the use of mRNA COVID-19 vaccines. https://t.co/olg8VTh6gB" / X (twitter.com)
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Ftwitter.com%2FFLSurgeonGen%2

Florida State Surgeon General

Calls for Halt in the Use of

COVID-19 mRNA Vaccines

Tallahassee, Fla. – On December 6, 2023, State Surgeon General Dr. Joseph A. Ladapo sent a letter <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.floridahealth.gov%2Fabout%
06-2023-DOH-Letter-to-FDA-RFI-on-COVID-19-Vaccines.pdf%3Futm_medium%3Demail%26utm_source%3Dgovdelivery&data=05%7C02%7Cwsboh%40s to the United States Food and Drug Administration (FDA) Commissioner Dr. Robert M. Califf and Center for Disease Control and Prevention (CDC) Director Dr. Mandy Cohen regarding questions pertaining to the safety assessments and the discovery <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fosf.io%2Fpreprints%2Fosf%2Fmjc of billions of DNA fragments per dose of the Pfizer and Moderna COVID-19 mRNA vaccines.

The Surgeon General outlined concerns regarding nucleic acid contaminants in the approved Pfizer and Moderna COVID-19 mRNA vaccines, particularly in the presence of lipid nanoparticle complexes, and Simian Virus 40 (SV40) promoter/enhancer DNA. Lipid nanoparticles are an efficient vehicle for delivery of the mRNA in the COVID-19 vaccines into human cells and may therefore be an equally efficient vehicle for delivering contaminant DNA into human cells. The presence of SV40 promoter/enhancer DNA may also pose a unique and heightened risk of DNA integration into human cells.

In 2007, the FDA published guidance on regulatory limits for DNA vaccines in the Guidance for Industry: Considerations for Plasmid DNA Vaccines for Infectious Disease Indications (Guidance for Industry) <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.fda.gov%2Fmedia%2F7366 . In this Guidance for Industry, the FDA outlines important considerations for vaccines that use novel methods of delivery regarding DNA integration, specifically:

* DNA integration could theoretically impact a human's oncogenes – the genes which can transform a healthy cell into a cancerous cell.
* DNA integration may result in chromosomal instability.
* The Guidance for Industry discusses biodistribution of DNA vaccines and how such integration could affect unintended parts of the body including blood, heart, brain, liver, kidney, bone marrow, ovaries/testes, lung, draining lymph nodes, spleen, the site of administration and subcutis at injection site.

On December 14, 2023, the FDA provided a written response providing no evidence that DNA integration assessments have been conducted to address risks outlined by the FDA <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.fda.gov%2Fmedia%2F7366 themselves in 2007. Based on the FDA's recognition of unique risks posed by DNA

integration, the efficacy of the COVID-19 mRNA vaccine's lipid nanoparticle delivery system, and the presence of DNA fragments in these vaccines, it is essential to human health to assess the risks of contaminant DNA integration into human DNA. The FDA has provided no evidence that these risks have been assessed to ensure safety. As such, Florida State Surgeon General Dr. Joseph A. Ladapo has released the following statement:

"The FDA's response does not provide data or evidence that the DNA integration assessments they recommended themselves have been performed. Instead, they pointed to genotoxicity studies – which are inadequate assessments for DNA integration risk. In addition, they obfuscated the difference between the SV40 promoter/enhancer and SV40 proteins, two elements that are distinct.

DNA integration poses a unique and elevated risk to human health and to the integrity of the human genome, including the risk that DNA integrated into sperm or egg gametes could be passed onto offspring of mRNA COVID-19 vaccine recipients. If the risks of DNA integration have not been assessed for mRNA COVID-19 vaccines, these vaccines are not appropriate for use in human beings.

Providers concerned about patient health risks associated with COVID-19 should prioritize patient access to non-mRNA COVID-19 vaccines and treatment. It is my hope that, in regard to COVID-19, the FDA will one day seriously consider its regulatory responsibility to protect human health, including the integrity of the human genome."

In the spirit of transparency and scientific integrity, State Surgeon General Dr. Joseph A. Ladapo will continue to assess research surrounding these risks and provide updates to Floridians.

On September 13, 2023, State Surgeon General Dr. Joseph A. Ladapo provided guidance <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Ffloridahealthcovid19.gov%2Fwp-content%2Fuploads%2F2023%2F09%2F20230913-booster-guidance-final.pdf%3Futm_medium%3Demail%26utm_source%3Dgovdelivery&data=05%7C02%7Cwsboh%40sboh.
against COVID-19 boosters for individuals under 65 and younger. In addition to aforementioned concerns, providers and patients should be aware of outstanding safety and efficacy concerns outlined in the State Surgeon General's previous booster guidance released in September.

From: Arne Christensen
Sent: 1/17/2024 11:09:22 AM
To: DOH WSBOH
Cc:
Subject: lonely people walking in the rain wearing face masks

External Email

The health department needs to stop lying to us about the effectiveness of face masks, vaccines, and social distancing for protecting people against covid. I just saw a man with a flimsy blue plastic mask walking outdoors, by himself, in the cold rain. He is only doing this because public health agencies have lied about masks for 4 years, and have inexplicably failed to advise people that masks don't work when wet.

From: bill teachingsmiles.com
Sent: 1/8/2024 8:32:17 AM
To: DOH WSBOH
Cc:
Subject: Public Comment 1/10/2024 Osmunson

External Email

Dear Washington State Board of Health,

I am requesting to provide public comment for the January 10, 2024 Board of Health Meeting.

My comments:

The Board of Health is the highest health authority in Washington State. Overhearing one Board member say, "but we are not supposed to have to look at the science." My jaw dropped almost to the floor. If the Board does not read science, what does the Board use to determine "health" policy such as fluoridation? Gossip? Rummers? Industry? The Dental Lobby?

In effect, the Board trusts the dental lobby and disregards inconvenient empirical factual evidence, laws and authorities such as:

I. The Washington State Board of Pharmacy, who determined that fluoride is a "legend drug." However, the Board of Health disagrees and trusts the dental lobby. The Board of Pharmacy was disbanded in part because they agreed with the law and science that fluoride ingested with intent to prevent disease is a prescription drug. Are you Board of Health doctors willing to put your license on the line prescribing the drug for everyone in Washington State without their consent or being patients of record? That would be unethical. Pharmacists have more training and expertise with toxins, dosage, adverse reactions and inter reactions of toxins than any other licensed profession. What empirical evidence does the Board of Health have which disagrees with the Board of Pharmacy? None. The Board of Health is violating science and laws of health.

See: Krzeczkowski JE, et al. Prenatal fluoride exposure, offspring visual acuity and autonomic nervous system function in 6-month-old infants. <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.sciencedirect.com%2Fscienc
Environment International. 2023

II. U.S. Congress which has authorized the Food and Drug Administration Center for Drug Evaluation and Research (FDA CDER) to determine the efficacy, dosage, safety and label of substances used to prevent disease. No, the Board trusts the dental lobby.

III. FDA CDER warns, "Do Not Swallow". Instead, the Board trusts the dental lobby and promotes mandated fluoride ingestion for everyone without patient consent, without patient dosage control, without the Doctor as legal intermediary, without regard for age or health of the patient. FDA CDER has determined fluoride ingestion lacks evidence of efficacy. And the FDA has given warnings to bottled water manufacturers (not FDA CDER approved) the fluoridated water must not be marketed to those under two years of age. The Board of Health is harming the public by disagreeing with authorized regulatory agencies.

IV. The Environmental Protection Agency scientists finding over two decades ago that fluoridation borders on a criminal Act because of toxicity and lack of current benefit. The Safe Drinking Water prohibits the EPA from adding anything to water to treat humans, so the Board trusts the dental lobby. And the EPA Dose Response Analysis and Relative Source Contribution of 2010 reporting that most or all infants and toddlers are ingesting too much fluoride.

V. The National Research Council 2006 report for the EPA that EPA's Maximum Contaminant Level for fluoride was not protective and harms most if not all cells and systems of the body. Instead, the Board of Health trusts the dental lobby. Fluoride is a contaminant the Board recommends adding to water.

VI. The National Toxicology Program reporting fluoride is a presumed developmental neurotoxin with 55 human studies, 52 reported IQ loss a 95% consistency. And their meta-analysis reports IQ loss. But no. The Board would rather trust the dental lobby rather than toxicologists for toxicity. Not everyone has the same sensitivity to drugs/toxins or the same health or the same ability to handle drugs/toxins. Some individuals had much more IQ loss and some were probably unaffected. The mean is not protective or representative of each individual. The Board must protect everyone, not just the healthiest and wealthiest.

"This January, Birnbaum <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fen.wikipedia.org%2Fwiki%2FLinda issued a scathing legal declaration as part of the lawsuit, writing, 'The decision to set aside the results of an external peer review process based on concerns expressed by agencies with strong policy interests on fluoride suggests the presence of political interference in what should be a strictly scientific endeavor.' Birnbaum said she issued the legal declaration in part over concerns the report might never be publicly released… the science proves there is 'no real benefit' from ingesting fluoride. 'The benefit from fluoride is from topical applications,' she said." - Capital and Main (March 14, 2023)

VII. Only one RCT (randomized controlled trial, the highest quality of research) of fluoride ingestion has been published and it report no statistical benefit from ingesting the fluoride. That's right. NO, NONE, ZERO quality studies reporting dental benefit of fluoride ingestion. No wonder the FDA said the evidence of efficacy is incomplete.

VIII. The lack of mechanism of action. Fluoride cannot go from the blood to the tooth pulp chamber through the calcium rich dentin and enamel to the outside of the tooth where the dental caries are forming and active. Fluoride during swallowing of water is short term and little gets to the lower teeth and the theoretical slight increase of fluoride in saliva with water at 0.7 ppm is too dilute to have an effect. Research has not reported a benefit at 700 ppm let alone 0.7 ppm.

IX. 97% of Europe does not fluoridate their water. And their dental caries are a similar rate as fluoridated communities and states not fluoridated.

X. CDC has known since the publication of the 2006 National Research Council (NRC) report to the EPA, that there is no safety data for susceptible sub-populations and significant scientific evidence of probable harm. In 2018, Mr. Casey Hannan of the CDC admitted under oath in a deposition for the trial in federal court expected to wrap up in February 2024 that the CDC accepts the 2006 NRC conclusions. Mr. Hannan also admitted that the CDC has no safety data specific to pre- and post-natal exposure. We understand Mr. Hannan decided to retire before commencement of that trial.

XI. Public Health Service (PHS) researchers advised the PHS in 1956 and 1961 that a portion of the allergic population would experience significant and acute ill effects from fluoridation programs with no pragmatic recourse to avoid the irritant. Other researchers in that decade advised that the placentas of women living in 'optimally' fluoridated

communities were saturated with fluoride at twice the concentration of the water they drank. They opined that although they didn't know the fetal impact, the mothers would probably be fine. (Feltman 1956; Feltman & Kosel 1961; Gardner et al. 1952

PHS lowered fluoridation concentration recommendation from 0.7-1.2 mg/L to 0.7 mg/L. However, no studies on efficacy have been done at current lower concentrations.

Once again, I am calling for the Board to remove their endorsement of fluoridation from your web site and protect the fetus and infants from known harm.

Current evidence is alarming on fluoride's contribution not just to lower IQ, but also to preterm birth and infant mortality.

See also https://www.fluoridelawsuit.com/science <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.fluoridelawsuit.com%2Fscier

Once again, I am calling for the Board to remove their endorsement of fluoridation from your web site and protect the fetus and infants from known harm.

Bill Osmunson DDS MPH

From: Arne Christensen
Sent: 2/6/2024 1:21:14 PM
To: DOH WSBOH
Cc:
Subject: alleged Taiwan face mask death

External Email

You need to read this article from January, "Infant dies after allegedly suffocating on mask at New Taipei daycare":
https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Ffocustaiwan.tw%2Fsociety%2F2024

It begins: "Authorities in New Taipei on Wednesday said they are investigating the death of an 11-month-old boy at a public daycare center, which the child's family allege happened when he suffocated on a mask a teacher forced him to wear."

After reading it, do you still think face masks are just an inconvenience? I don't accept the reply that public health authorities never said infants should have to wear masks. Normalizing and requiring masks on toddlers was going to lead to requiring masks on infants somewhere in the world.

From: Garry Blankenship
Sent: 2/5/2024 8:15:15 AM
To: hcinfo.infosc@canada.ca,DOH WSBOH,dhsmoh@yahoo.com,secretary@health.gov.bz,Van De Wege, Kevin,Chapman, Mike (LEG),sheriff@co.clallam.wa.us,mozias@co.clallam.wa.us,rjohnson@co.clallam.wa.us,shahidafatin@gmail.c
Allison 2 (DOHi)
Cc:
Subject: The NOP BOH Needs Introspection

External Email

I do not doubt the BOH intentions, but recommending, promoting and mandating these mRNA injections was and remains a colossal mistake. Denying the naturally immune public access was worse. The Federal, State and local pandemic management record is without exception an abject failure. I request the Board make the effort to insure mistakes like this never repeat.

https://www.theepochtimes.com/health/for-every-life-saved-mrna-vaccines-caused-nearly-14-times-more-deaths-study-5579794?utm_source=Ccpv&src_src=Ccpv&utm_campaign=2024-02-05&src_cmp=2024-02-05&utm_medium=email&est=0Y%2F9GSyc74a%2FdwbERhO%2FTk2D8BeBhXgQlredhB%2Fte85A4PYzcUdI

<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.theepochtimes.com%2Fheal every-life-saved-mrna-vaccines-caused-nearly-14-times-more-deaths-study-5579794%3Futm_source%3DCcpv%26src_src%3DCcpv%26utm_campaign%3D2024-02-05%26src_cmp%3D2024-02-05%26utm_medium%3Demail%26est%3D0Y%252F9GSyc74a%252FdwbERhO%252FTk2D8BeBhXgQlredh

Sincerely,

Garry Blankenship

From: patrice tullai
Sent: 1/5/2024 6:34:20 PM
To: DOH WSBOH
Cc:
Subject: Racism is a public health crisis

External Email

Hello, and good day to you,
When I was a child all children played together no matter race or color or religion, the policies that are being inflamed are creating more division among people, not less. I see division and victim mentality being pushed to the forefront, this does not help our children, youth, or society, this is dividing people. We need to come together. The problems come from class ….the poor suffer. I would like to encourage you to not act under the idea, or create policies that racism is a public health problem ,
Thank you
I hope you and 2024 work to bring humanity together not divided,
Patrice Tullai
PateiceTullai@gmail.com

_____

From: DOH WSBOH
Sent: 3/8/2024 11:51:33 AM
To: DOH WSBOH
Cc:
Subject: FW: My Public Comments

Forwarding as this email has the same subject line as her email from 3/7 and the system would not accept a duplicate.

From: Melissa Leady <melleady@yahoo.com>
Sent: Friday, March 8, 2024 11:11 AM
To: DOH WSBOH <WSBOH@SBOH.WA.GOV>
Subject: My Public Comments

External Email

As part of the PEAR Plan Development, will the Department of Health (DOH) be conducting a pandemic policy review, looking at some of the unintended negative impacts of covid policies? Pandemic policy in Washington state disproportionately impacted lower-income families and people of color.

Loss of in-person learning at schools resulted in lower test scores. In Vancouver, for example, the city is providing $500,000 to the Vancouver Public School District to address covid learning loss at elementary schools in the Fourth Plain corridor. These are among the most ethnically diverse and economically challenged schools in the district. For the students in these schools, the cost of covid learning loss could be felt for their lifetimes, according to a UN study on children living in learning poverty.

Covid job loss also disproportionately impacted low wage jobs, as the "laptop class" quickly transitioned to working from home. At my last county board of health meeting, my local health director mentioned that the covid job loss often resulted in loss of health insurance. Has there been any assessment of the effects of pandemic policy-related job loss on access to healthcare?

During the pandemic, the public was told to isolate and parks and outdoor recreation were closed. The obesity rate in Washington state increased 2%. Obesity is closely linked to a wide variety of negative health outcomes, including diabetes, heart disease, cancer, and covid death. According to the CDC, the current obesity rates in Washington state by race are: 10% Asian, 30% white, 36% Black, 36% Hispanic, and 43% Native American. Will the PEAR Plan Development be looking at differing rates of obesity by race as part of their efforts to understand differing rate of covid deaths by race?

These are just a few examples. Other areas to explore include : impacts on small businesses and restaurants, school enrollment, mental health, anxiety, depression,

substance abuse, drug overdoses, domestic violence, housing and housing affordability, food insecurity, and loss of cultural events and religion gatherings.

In addition, has there been an assessment of the impacts of the Washington state vaccine mandate? A recent study comparing states with vaccine mandates and states banning vaccines mandates showed 1) no comparable difference in vaccine uptake; and 2) reduced rates of flu and booster uptake in states that imposed mandates.

Does DOH attribute the low 2023-2024 rates for flu vaccination (30%) and covid vaccination (18%) to "blow back" from the vaccine mandates? What was the impact of the mandates of jobs and healthcare? In Clark County, for example, there was a 10% drop in hospital beds after the mandate took effect, when some hospital staff chose to quit instead of getting vaccinated. Eventually that difference was made up by employing traveling nurses at an increased cost, driving up costs locally.

I hope that DOH will take the time to assess the "collateral damage" of covid policy decisions, as former NIH director Francis Collins recently termed it. Perhaps this could be done in conjunction with the PEAR Impact Assessment.

Sincerely,

Melissa Leady

Clark County Resident

From: Garry Blankenship
Sent: 3/2/2024 8:22:24 AM
To: Van De Wege, Kevin,Chapman, Mike (LEG),DOH WSBOH,sheriff@co.clallam.wa.us,mozias@co.clallam.wa.us,rjohnson@co.clallam.wa.us,shahidafatin@gmail Allison 2 (DOHi),Tharinger, Steve
Cc:
Subject: Higher Mortality In Vaxed Vs Unvaxed

External Email

Good Day All,

I have found any contra "vaccine" information, regardless of documentable verification, to be summarily dismissed by most medical practitioners, particularly those holding any authoritative position, with no effort to independently vet that information. No objectivity in vetting drug safety is a huge looming problem that will not go away. Confidence in our health care system has been critically damaged by a lack of acknowledging mistakes made in the "pandemic". It is clear that the medical community was given false information on the COVID "vaccines", treatment protocols and repurposed drugs, but the absence of acknowledging that will self destruct the medical complex. I implore you to stop pretending that promoting these mRNA platform injectable products was or is health positive. These drugs are killing the young and working aged disproportionately.

https://www.theepochtimes.com/health/study-finds-higher-mortality-among-vaccinated-patients-hospitalized-for-covid-19-post-5597490?utm_source=Ccpv&src_src=Ccpv&utm_campaign=2024-03-02&src_cmp=2024-03-02&utm_medium=email&est=AAAAAAAAAAAAAAAAaes4chIex8Tb%2F5MNsWhaCqduhU0LM8%2FAPpFNrub

<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.theepochtimes.com%2Fheal finds-higher-mortality-among-vaccinated-patients-hospitalized-for-covid-19-post-5597490%3Futm_source%3DCcpv%26src_src%3DCcpv%26utm_campaign%3D2024-03-02%26src_cmp%3D2024-03-02%26utm_medium%3Demail%26est%3DAAAAAAAAAAAAAAAAaes4chIex8Tb%252F5MNsWhaCqduhU0LM

Not seeking anonymity,

Garry Blankenship

From: Michelle Anderson
Sent: 2/1/2024 5:10:05 PM
To: DOH WSBOH
Cc:
Subject: Public Comments for the Environmental Health Committee

External Email

Dear Board.
I would just like to remind you that Mandatory COVID shots or testing is unacceptable!
It is now just another virus that we must all deal with!
Just like the FLU, Common Cold or any other Corona Virus (there are a bunch and tests don't tell you WHICH one it is)
We are ADULTS and we can make decisions for our own children!
Government mandates are unnecessary!
Thank you very much for all you do!

From: Garry Blankenship
Sent: 2/24/2024 7:40:04 AM
To: Van De Wege, Kevin,Chapman, Mike (LEG),DOH WSBOH,sheriff@co.clallam.wa.us,mozias@co.clallam.wa.us,rjohnson@co.clallam.wa.us,shahidafatin@gmail Allison 2 (DOHi)
Cc:
Subject: "Vaccine" Adverse Events

External Email

I can only hope those responsible for promoting and particularly mandating these toxins are held accountable. These injections violate informed consent and the Hippocratic Oath.

https://www.theepochtimes.com/health/a-host-of-notable-covid-19-vaccine-adverse-events-those-backed-by-evidence-5590525?utm_source=Health&src_src=Health&utm_campaign=health-2024-02-24&src_cmp=health-2024-02-24&utm_medium=email&est=AAAAAAAAAAAAAAAAaes4chIex8Tb%2F5MNsWhaCqduhU0LM8%2FAPpFNrub

_____

From: Stuart Halsan
Sent: 2/6/2024 8:07:49 PM
To: DOH WSBOH
Cc:
Subject: Communicating With Board Members

External Email

For Patty Hayes

I have some genealogical info for you.. You can reach out to me at this email.
Hope all is well.
Stuart Halsan
Sent via the Samsung Galaxy S9+, an AT&T 5G Evolution capable smartphone

_____

From: Karen Spencer
Sent: 3/8/2024 10:05:47 AM
To: DOH WSBOH
Cc:
Subject: Comment: Fluoridation Poisoning

External Email

"Fluoride is capable of producing any number of symptoms. They include drowsiness, profound desire to sleep, dizziness, nasal congestion, sneezing, runny nose, sore throat, coughing, wheezing (asthma), chest pain, hives, and various intestinal symptoms. Most of the information concerning specific reactions to fluoride, as seen in private practice, never reach publication." - Hobart Feldman, MD, American Board of Allergy and Immunology (1979)

Board of Health -

I signed up to make a comment on Wednesday March 13th, but may be unavailable at that time. Therefore, I am sending a written comment for your consideration:

MY PERSONAL STORY:

My name is Karen Spencer. I am a retired analyst and project management consultant who has worked with all levels of Corporate America.

I am angry about what happened to me and my children. I was poisoned by fluoridated water while pregnant in 1981. My normal pregnancy turned difficult overnight. I was ill with chronic dizziness, nausea, bloody stools and rashes beginning the first week of July. I didn't make the connection to water until much later. Fluoridation began on July 1st.

I did not recover after giving birth. Worse, both my children shared my symptoms. It took me until late 1982 to realize tap water was causing our rashes and gastrointestinal problems. My primary care physician who was the Chair of the Board of Health yelled me out of his office in November when I asked if the water could be making us sick. In January '83, an allergist specializing in environmental health recommended I only use spring water in glass bottles for all of our water needs, which alleviated our symptoms.

Since bottled water is expensive, I installed a high-quality under the sink filter in '91. I was diagnosed with Lyme disease about the same time, so I accepted my doctors attribution of my emerging and ongoing arthritis and neurological symptoms to Chronic Lyme. They also diagnosed me with irritable bowel syndrome. I was in my 30s. I developed kidney and liver problems in my 50s.

I switched back to bottled water in 2014 to see if it would have a positive effect on my declining health. It did— within days. My multi-stage system wasn't adequate and never had been. Can you imagine my outrage when I realized, in my 60s, that decades of arthritis, gastrointestinal illness, neurological issues and even concerns over organ failure had been fluoride poisoning?

There is no happy ending for me. The damage to my bones and spinal discs from decades of fluoride poisoning cannot be undone, and neither can the damage to my son who has learning disabilities consistent with what has been validated by developmental neurotoxicity studies.

The chair of my local board of health, a doctor, told me in 2014 that "they" knew some

people would have problematic symptoms from fluoridation, but it was a "greater good" to prevent a cavity or two in poor children. Please don't tell me that my life and the lives of my children are collateral damage. I suggest that ending fluoridation not only provides health equity for susceptible sub-populations, but also serves justice to the grandchildren of my baby-boomer generation who were poisoned by an ill-conceived, immoral medical mandate.

* CAPE ANN STORY WITH REFERENCES: https://fluoridealert.org/wp-content/uploads/SalemState2016.09.07.pdf <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Ffluoridealert.org%2Fwp-content%2Fuploads%2FSalemState2016.09.07.pdf&data=05%7C02%7Cwsboh%40sboh.wa.gov%7C684f7

* ANNOTATED SCIENCE BIBLIOGRAPHY: https://www.fluoridelawsuit.com/science <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.fluoridelawsuit.com%2Fscien

For more about me, see my signature.

Regards,

_____

Karen Favazza Spencer
Leominster, MA 01453
978.283.4606
Subscribe on YouTube <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.youtube.com%2Fchannel%2vZ55u7oKUchQ&data=05%7C02%7Cwsboh%40sboh.wa.gov%7C684f7f0a9660495d62fd08dc3f99f847%7C

See the Call to Action <https://gcc02.safelinks.protection.outlook.com/?url=http%3A%2F%2Fwww.greenmedinfo.com%2Fblog%letter-nutritionists-about-fluoride-deception&data=05%7C02%7Cwsboh%40sboh.wa.gov%7C684f7f0a9660495d62fd08dc3f99f847%7C11d0e

More power to you if fluoridation doesn't bother you, but not the power to assume it's safe for your neighbor with kidney disease, his pregnant wife or their diabetic daughter!

About Karen: Currently a semi-retired consultant working with software development teams, Karen Spencer is a former analyst and project leader. She is adept at conducting research and analyzing trends. Her special interests include critical thinking, data-driven decision making, and organizational theory. She and others in her family are among the 15% of Americans with chemical sensitivities triggered by exposure to fluoridated food and drink. Karen's publications were featured in:
Medical Hypotheses (2018): https://pubmed.ncbi.nlm.nih.gov/30396472/ <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fpubmed.ncbi.nlm.nih.gov%2F303

GreenMed (2019): https://www.greenmedinfo.com/blog/wetoo-medical-assault-and-battery <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.greenmedinfo.com%2Fblog%medical-assault-and-battery&data=05%7C02%7Cwsboh%40sboh.wa.gov%7C684f7f0a9660495d62fd08dc3f99f847%7C11d0e2

Gloucester Times (2022): https://www.gloucestertimes.com/opinion/column-stop-poisoning-gloucester/article_0089c49c-1278-11ed-8a42-fb294218a4fe.html <https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.gloucestertimes.com%2Fopistop-poisoning-gloucester%2Farticle_0089c49c-1278-11ed-8a42-fb294218a4fe.html&data=05%7C02%7Cwsboh%40sboh.wa.gov%7C684f7f0a9660495d62fd08dc3f99f847

Message to CDC (2022): https://www.youtube.com/watch?v=PzviupO1cDQ
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.youtube.com%2Fwatch%3Fv

Collaborative Activism (2022-current): https://www.fluoridelawsuit.com/actions
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.fluoridelawsuit.com%2Factio

Bill in MA Legislature (2023): https://malegislature.gov/Bills/193/S460
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fmalegislature.gov%2FBills%2F19

Document Fraud at CDC (2024):
https://www.researchgate.net/publication/377152337_Document_Fraud_at_CDC
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.researchgate.net%2Fpublica

From: Garry Blankenship
Sent: 2/17/2024 10:31:07 AM
To: hcinfo.infosc@canada.ca,DOH WSBOH,OADS@cdc.gov,sheriff@co.clallam.wa.us,Van De Wege, Kevin,Chapman, Mike (LEG),mozias@co.clallam.wa.us,rjohnson@co.clallam.wa.us,shahidafatin@gmail.com,gbsjrmd@sisna.com,n
Allison 2 (DOHi)
Cc:
Subject: Vaccine Shedding

External Email

Fascinating article and video on shedding. The probability of vaccinated people shedding spike proteins on other people is very real.

https://www.theepochtimes.com/health/covid-vaccine-shedding-is-real-fda-and-pfizer-documents-are-proof-clinicians-5588819?utm_source=Health&src_src=Health&utm_campaign=health-2024-02-17&src_cmp=health-2024-02-17&utm_medium=email&est=AAAAAAAAAAAAAAAAaes4chIex8Tb%2F5MNsWhaCqduhU0LM8%2FAPpFNrub

_____
From: Cheryl Lewis
Sent: 1/23/2024 7:57:12 AM
To: DOH WSBOH
Cc:
Subject: Communicating With Board Members

attachments\B7B9A7277A0E4E2B_Sledge - BOH Strategies.pdfDept o_PRDTOOL_NAMETOOLONG.pdf

External Email

Microsoft Edge - ready to share - Presentation and 6 more pages - Personal - Microsoft Edge - 15 January 2024 - Watch Video
<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.loom.com%2Fshare%2F8bc0

<https://cdn.loom.com/sessions/thumbnails/8bc09cd7d30146e6a46991886f25c8c8-00001.jpg>
healthcare hygienist!

Hello All

I am a dental hygienist who would love to see an improvement in oral care for our community. I believe there are many ways to improve this and ran across this publication on your site (it is at the bottom of the page). It seems to be dated 2013. I am wondering how far we have come since then? I have created a presentation that I would like to share with you. It is about 30 minutes long and I feel it promotes your cause in a different light. I would be honored if you would review it and allow me to be a resource to you in this arena. I have a deep desire to improve the oral care of our facility residents, from the hospital to the long term care facilities. I believe dental hygienist's should be employed as a member of each of these facilities as oral care specialists, not to perform traditional dental cleanings but to improve daily oral care which will improve quality of life. Having a hygienist visit a facility every 3-6 months isn't helping people keep their mouths healthy. Please watch my presentation to gain insight on this. I think we should at the very least, create a certification for caregivers, one that specializes in oral care. Maybe they could have increased training on oral diseases to look for (cancer, gum disease, cavities, dry mouth sores, abscesses). Special training on treatment and prevention of caries and gum disease. This distinction could create value of the caregiver and maybe that could translate to an increase in their wage, which may lead to retention, maybe decrease turnover? If there was a team or even an individual in charge of oral care and only oral care, our dependents would not suffer with dry mouth sores and bleeding gums. Oral care is often the first area to be neglected and a visit from the hygienist 2 times a year is not the way to maintain oral health. We are learning more and more about the bacteria's role in our health and allowing plaque (bacteria) and food to linger for days, weeks, months is not promoting health. Often oral care is left to the resident, unless it is noted on the residents care plan to brush for them.

I know you are busy, but please take a moment (30 minutes or so ☐☐) and consider the change that could be made. It's like a child who drowns in the swimming pool, when everyone is watching, no one is watching. We need a go to, a someone in charge of daily oral hygiene to ensure people are receiving the care they need and deserve. This would not only reduce risk of cavities and gum disease, but aspirated pneumonia, sepsis, and death as well.

I am trying to make change starting at the top (you).

I look forward to hearing from you and thank you sincerely for taking the time to consider this.

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From: Cheryl
Sent: 1/30/2024 6:08:46 AM
To: DOH WSBOH
Cc:
Subject: Communicating With Board Members

attachments\88C9EC27E025473E_my presentation (1).htm

attachments\5B71ED4076E84D7A_Sledge - BOH Strategies.pdfDept o_PRDTOOL_NAMETOOLONG.pdf

External Email

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I am trying to make change starting at the top (you).

I look forward to hearing from you and thank you sincerely for taking the time to consider this.

Best

Cheryl lewis RDH

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<https://gcc02.safelinks.protection.outlook.com/?url=https%3A%2F%2Fgo.microsoft.com%2Ffwlink%2F%
for Windows

_____

From: bill teachingsmiles.com
Sent: 2/29/2024 7:31:08 AM
To: DOH WSBOH
Cc:
Subject: March 13 Public Comment

External Email

Dear Washington State Board of Health,

The Legislature has made one of the duties of the Board of Health to assure drinking water is safe, because water is essential for life.

The Legislature does not say the duty is to assure efficacy, because that’s the duty of the FDA.

Fluoridation of public water is not safe because, not once did the EPA expert scientists during the two-week trial before the Superior Court of California (January and February 2024) testify that fluoridation was safe, or effective.

Fluoridation of public water is not safe because, it is a highly toxic contaminated scrubbings of manufacturing, a poison, a prescription drug, not FDA approved, misbranded and adulterated.

Fluoridation is not safe because, it violates an individual’s consent, freedom to choose, and their doctor's oversight.

Fluoridation is not safe because, fluoride causes dental fluorosis. I, and most dentists, each made and make hundreds of thousands of dollars treating cosmetic and functional dental fluorosis, harm.

Fluoridation is not safe because, fluoride ingestion increases developmental neurotoxicity as measured with lower IQ. Lower IQ increases the rate of special education in schools, lower wage jobs, more unemployment, more divorce, more incarceration, more grief, fewer gifted, and is bad for America, especially minorities.

Fluoridation is not safe because, fluoride ingestion harms the developing fetus, infant and child as measured with increased miscarriage, increased premature birth, and increased infant mortality.

Fluoridation is not safe because, fluoride ingestion is stored in the bones and as the bones remodel the fluoride is given off. Mother's blood concentration of fluoride in the third trimester increases when she has inadequate intake of calcium for her fetus's needs.

Fluoridation is not safe because, fluoride ingestion harms the joints causing rheumatoid and osteoarthritic-like pain.

Fluoridation is not safe because fluoride ingestion harms the thyroid and is an endocrine disruptor, increasing diabetes, obesity and ADHD.

Fluoridation is not safe because fluoride ingestion increases osteosarcoma a rare but lethal bone cancer, mostly in boys drinking fluoridated water during growth spurts.

Fluoridation is not safe because fluoride ingestion harms the kidneys and GI disorders.

Do not let the fluoridation lobby confuse you. The Board's job is to assure safety. The dental lobby's job is to gain FDA CDER approval. They have failed, but you must not.

We look forward to participating in a forum on fluoride ingestion because we and many are being harmed.

Bill Osmunson DDS MPH

Washington Action for Safe Water

_____

From: Melissa Leady
Sent: 3/7/2024 6:13:04 PM
To: DOH WSBOH
Cc:
Subject: My Public Comments

External Email

IS THE CURRENTLY AUTHORIZED COVID-19 VACCINE EFFECTIVE?

During a recent county board of health meeting, the health director for my county made the claim that there is state data showing that the updated covid-19 vaccine is effective at preventing severe illness, hospitalizations, and deaths; and that it is effective at preventing infection and thereby transmission.

It seems my local health director is out on a limb in making this claim. If DOH has such data on the updated covid-19 vaccine, they have never publicly shared it.

The DOH report on Hospitalizations and Deaths by Vaccination Status (#421-010), which hasn't updated in three months, begins by stating, "PLEASE NOTE: Information about bivalent booster doses (authorized in the fall of 2022) or the updated monovalent booster doses (authorized in September of 2023) is not included in this report."

Is the board recommending the currently authorized updated covid-19 vaccine? If so, do you have Washington state data showing the vaccine's effectiveness? Please share it with the public.

From: Stuart Cooper
Sent: 3/8/2024 12:13:53 PM
To: DOH WSBOH
Cc:
Subject: Public Comment - Fluoridation Petition

attachments\7605F4B7B7C3499D_word - Letter to WA BOH.docx

External Email

Please see my public comment attached in the word document.

Thank you,

Stuart Cooper
Executive Director
Fluoride Action Network


EMERGENCY
EYE WASH
EMERGENCY
SHOWER

From: John Mueller
Sent: 3/8/2024 12:07:57 PM
To: DOH WSBOH
Cc:
Subject: My Public Comments

attachments\6C7FF512628C4B63_30427CA8A0374B29BF1125260FD59D80.jpg

External Email

Please consider the hazardous work conditions and equipment maintenance expenses with the operation of a water fluoridation program. Fluorosilicic acid is highly corrosive, with vapors combining with ambient air moisture to form hydrofluoric acid. The attached photo shows the corrosive effects on safety equipment in a fluorosilicic acid storage room at a large municipal treatment plant. Obviously the equipment in the photo needed frequent and regular maintenance.

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for Windows

Submission to WA Board of Health

The State Board of Health Ought to Act to Protect Residents From the Unnecessary Risks Posed By Water Fluoridation

Dear Members of the State Board of Health:

I'm urging the State Board of Health to respond appropriately to the CDC's data showing that millions of U.S. residents have visible signs of overexposure to fluoride, as well as government-funded research linking fluoride in water to impaired brain development in children.

The CDC's National Health and Nutrition Examination Survey (NHANES) has consistently found skyrocketing rates of dental fluorosis. The agency reported that 41% of adolescents (12 to 15) had dental fluorosis in 2004, an increase of over 400% from the rates found 60 years prior. The CDC's 2012 survey found that the rate jumped significantly to 65+% of adolescents with dental fluorosis. Now, according to a recent study (Yang, June 2021) published in the journal Ecotoxicology and Environmental Safety using the data from the NHANES 2015-16 survey, the "prevalence of dental fluorosis was 70% in U.S. children."

Dental fluorosis is a permanent tooth enamel defect caused by excessive fluoride intake during childhood. It appears as white spots or lines in milder cases and pitted and stained enamel in more severe cases, weakening the teeth and resulting in increased decay. More importantly, fluorosis is a biomarker of overexposure to fluoride during childhood development.

Ingesting fluoridated water, particularly in reconstituted infant formula, and processed foods made with fluoridated water are recognized as the primary sources of exposure, though swallowing toothpaste and fluoride prescriptions also contribute.

The teeth of millions of children, teens, and adults have already been permanently damaged by overexposure to fluoride during their first 8 years of life, and the CDC, along with the other promoters of fluoridation, are fully aware. And yet, the public health officials have not only failed to warn consumers about this side-effect, but have continued to push for the expansion of the practice in Washington, with a recent attempt to

initiate fluoridation in Spokane and throughout the state with legislation that fortunately failed in 2022.

Teeth are obviously not the only tissues in the body that are harmed by, or accumulate, fluoride. The CDC's NHANES data has been used in recent published peer-reviewed studies to link fluoridated water with a number of additional side effects, including earlier onset of menstruation for black teens, sleep disorders in adolescents, increased uric acid levels in the blood, 2.5 times greater risk of pediatric fractures, and kidney and liver impairment in adolescents.

Additional studies on fluoridation have also recently found higher rates of hip fractures, disruption of the endocrine system, and increased rates of hypothyroidism.

There is also now a large body of government-funded studies linking early life exposure to neurotoxicity, including during infancy. The Board of health must take action to warn both pregnant women and parents of formula-fed infants about the recent NIH-sponsored research indicating that fluoride in drinking water poses a risk to the developing brain at the exposure levels experienced in fluoridated communities, both in utero and during early infancy. The lack of appropriate action to protect children by our federal agencies like the CDC and HHS is an alarming disregard for science and disrespect for the welfare of U.S. citizens, but that doesn't mean that the Washington Board of Health must also fail to act. Our federal agencies leave fluoridation decisions up to state and local policymakers. This includes you. You're in a position to act and ought to.

It has now been six years since the first high quality US-government funded study (Bashash et al., 2017) found an association between fetal exposure to fluoride and lowered IQ, five years since a government-funded study found an increase in ADHD symptoms associated with in utero exposure to fluoride (Bashash et al., 2018), four years since the findings in Bashash's study were repeated by another US-government funded study (Green et al., 2019), and 3 years since a third US-government-funded study (Till et al., 2020) found that bottle-fed infants in fluoridated communities in Canada had a significantly lowered IQ compared to bottle-fed infants in non-fluoridated communities.

You should also be made aware of the following:

- A study from Canada published last summer in the journal Nutrients found that pregnant women who had low iodine levels and elevated fluoride had boys who suffered an average IQ loss of 9.3 IQ points [Goodman 2022]. Artificially fluoridated drinking water was the main source of the fluoride. To put this huge 9-point IQ loss from fluoride into perspective, studies show that a pregnant woman smoking 20+ cigarettes each and every day during their pregnancy can cause less of an IQ loss for the child than fluoride, averaging about 6.2-points lost.

- Experts in environmental toxins, including the former Director of the National Toxicology Program, Dr. Linda Birnbaum, published an op-ed calling for policy makers to look at the science and take action to protect pregnant women and their children.

- Famed Harvard researcher Phillippe Grandjean, known for helping warn the world about the effects of arsenic, mercury, and PFOAs, conducted the first benchmark dose analysis in 2020 on maternal fluoride exposure and neurotoxicity to the fetus, which was published in the journal Risk Analysis (Grandjean, 2021). Benchmark dose analyses are used by the Environmental Protection Agency (EPA) and toxicologists to determine at what level a substance starts to cause harm. The analysis confirmed that extremely low fluoride exposure during pregnancy impairs fetal brain development, finding that a maternal urine fluoride concentration of only 0.2mg/L — which coincides with the level in the water (0.2ppm) — was enough to lower IQ by at least 1 point.

  This is 3.5 times lower than the current government "recommended" level of 0.7ppm in fluoridated communities. For perspective, a urinary fluoride (UF) concentration of 0.2mg/L is far below what a pregnant woman in a fluoridated community would have, as confirmed by two recent studies.

The authors of the benchmark dose analysis stated: “These findings suggest that fetal brain development is highly vulnerable to fluoride exposure … and provide additional evidence that fluoride is a developmental neurotoxicant (i.e., causing adverse effects on brain development in early life). Given the ubiquity of fluoride exposure, the population impact of adverse effects from fluoride may be even greater than for other toxic elements like lead, mercury, and arsenic … and the benchmark results should inspire a revision of water fluoride recommendations aimed at protecting pregnant women and young children.”

- After conducting a 7-year systematic review of fluoride’s neurotoxicity, the National Toxicology Program reported that 52 of 55 fluoride brain studies found decreases in child IQ associated with an increase in fluoride, a remarkable 95% consistency. Of the 19 studies rated higher quality, 18 found a lowering of IQ. The meta-analysis could not detect any safe exposure, including at levels common from drinking artificially fluoridated water.

  The NTP’s report says: “Our meta-analysis confirms results of previous meta-analyses and extends them by including newer, more precise studies with individual-level exposure measures. The data support a consistent inverse association between fluoride exposure and children’s IQ.”

Meanwhile, more and more studies are being published on this issue around the world. See the list of 23 human studies that have been published in the four years since the Bashash, 2017 study was published. which have found a lowering of IQ associated with fluoride exposure at modest levels and in the case of the US-government funded studies at the levels experienced in artificially fluoridated communities.

It is an embarrassment for the USA to be perceived by the rest of the world as being willing to risk our children’s brains for anything, let alone a highly questionable benefit to their teeth that could easily be replaced with alternative oral health strategies. The longer you delay, the more citizens will be harmed.

Sincerely,

Stuart Cooper
Executive Director
Fluoride Action Network

www.FluorideAlert.org

WASHINGTON STATE BOARD OF HEALTH

Shay Bauman
Policy Advisor

Shay Bauman joined the Washington State Board of Health (Board) as a Policy Advisor for the Board's environmental public health/natural environment portfolio on February 1, 2024. Before joining the Board, Shay worked for the Washington State Office of the Attorney General, where she served as a Regulatory Analyst for the Public Counsel Unit. In this role, she represented residential electric, natural gas, solid waste, and water customers in the rulemakings and processes of the Washington Utilities and Transportation Commission (UTC). She has testified before the UTC on the impacts of various infrastructure investments, the Clean Energy Transformation Act, the Climate Commitment Act, and the equitable distribution of energy and non-energy benefits, among other topics. Her work has increased energy efficiency program uptake, reduced negative health impacts from energy production, and reduced energy burdens for Highly Impacted Communities and Vulnerable Populations. She also served on advisory boards to Washington's investor-owned utilities on topics related to conservation, low-income assistance, resource planning, and equity.

Shay was born and raised in Cedar City, Utah, a small town about 40 minutes north of Zion National Park. She received her Bachelor of Science in Economics with a minor in Political Science through Southern Utah University. She later received her Master of Public Administration through the University of Washington with emphases in public financial management and policy analysis.

STATE OF WASHINGTON
WASHINGTON STATE BOARD OF HEALTH
PO Box 47990 • Olympia, Washington 98504-7990

January 12, 2024

Sent via email

Dear John Gehman:

This letter is to inform you that on January 10, 2024, the Washington State Board of Health (Board) reviewed your complaint against the Snohomish County Health Department (SCHD) Director and Local Health Officer. Your complaint alleged that these health officials had violated RCW 70.05.070(3) and refused or neglected to obey and enforce the Board's rules related to communicable disease control under WAC 246-100-036 and 040.

The Board determined that the complaint did not warrant further investigation and dismissed the complaint for the following reasons. When reviewing your complaint and the supplemental statement of authorities you provided, Board Members did not find a violation of RCW 70.05.070(3), or WAC 246-100-036 or -040 by the Local Health Officer or Director at SCHD. The Board stated that there are currently no statewide requirements for masking and that current guidance is aligned with the Centers for Disease Control. Board Members also clarified that guidance is not enforceable.

The Board also discussed the topic of quarantine and how WAC 246-100-040, procedures of isolation and quarantine, is only used under certain circumstances. Board Members also mentioned that during the pandemic, mandatory isolation and quarantine did not occur in Washington or elsewhere in the United States. Recommendations around isolation and quarantine for COVID-19 in the U.S. remain voluntary.

Board Members also expressed the importance of reviewing these complaints, as there may be situations where local health officials are not serving the needs of their communities. However, Board Members noted that in this case, the Local Health Officer and Director at SCHD have appropriately protected the public's health with respect to COVID-19 and remain involved in statewide discussions related to masking policies and other public health topics.

The Board now considers your complaint closed and will take no further action. Materials related to this matter are available on the Board's website.

Sincerely,

Michelle A. Davis
Executive Director

cc: John G. Gehman

STATE OF WASHINGTON
WASHINGTON STATE BOARD OF HEALTH
PO Box 47990 • Olympia, Washington 98504-7990

January 26, 2024

Sent via email.

Dear John Gehman:

This letter is in response to your most recent submittal, received on Monday, January 22, 2024, alleging that the Snohomish County Health Department (SCHD) Director and Health Officer have failed to comply with provisions of Chapter 70.05 RCW in relation to COVID-19. It is substantially similar to the complaint you submitted on November 28, 2023, concerning alleged failure by the SCHD Director and Local Health Officer to control and prevent COVID-19 pursuant to public health statutes and rules. It asserts that the Board must conduct a preliminary investigation whenever it receives a complaint.

The Washington State Board of Health (Board) reviewed and discussed your previous, substantially similar complaint against the SCHD Director and Local Health Officer at its regular meeting on January 10, 2024. Consistent with the Board's procedure regarding complaints under RCW 70.05.120(1), the Board determined that your complaint did not merit a preliminary investigation. Materials related to this matter are available on the Board's website.

The Board may dismiss a complaint without a preliminary investigation if it lacks sufficient information to support a preliminary investigation or is frivolous— Washington State Board of Health Policy and Procedure, *Responding to Complaints Against a Local Health Officer or Administrative Officer Under RCW 70.05.120,* November 2022.

Your previous complaint was dismissed without a preliminary investigation for the reasons outlined in the letter sent to you on January 16, 2024. As noted in the letter, the Board considers your complaint closed and will take no further action.

Sincerely,

Michelle A. Davis
Executive Director

STATE OF WASHINGTON
WASHINGTON STATE BOARD OF HEALTH
PO Box 47990 • Olympia, Washington 98504-7990

January 16, 2024

John Strick
7331 17th Ave NE
Seattle, WA, 98115

Sent Via Email

Dear John Strick,

Thank you for the rulemaking petition you submitted to the State Board of Health (Board) on November 8, 2023. In your petition, you requested that the Board amend WAC 246-760-070 to add testing for color vision deficiency, also known as colorblindness, as part of the vision screening required for all students in Washington.

The Board met on January 10, 2024, and after reviewing and discussing your petition, voted to deny your request at this time. The Board determined that adding testing for color vision deficiency to chapter 246-760 WAC does not align with current national recommendations for school vision screenings, and implementing this test across schools in Washington would have significant financial costs and administrative challenges.

In its deliberations, however, the Board did acknowledge that color vision deficiency is a prevalent condition, especially in boys. Board Members stated that as a society, we need to adapt our teaching materials and classrooms to better support children with the condition.

Under RCW 34.05.330, a petitioner may appeal an agency's decision to deny a petition to repeal or amend a rule. An appeal must be made to the Governor within 30 days of denial.

If you require further assistance, please don't hesitate to contact Molly Dinardo, Health Policy Advisor in our office, at 564-669-3455 or at Molly.Dinardo@sboh.wa.gov.

Sincerely,

Patty Hayes

Patty Hayes, Chair

RULE-MAKING ORDER
EMERGENCY RULE ONLY

CR-103E (December 2017)
(Implements RCW 34.05.350 and 34.05.360)

CODE REVISER USE ONLY

OFFICE OF THE CODE REVISER
STATE OF WASHINGTON
FILED

DATE: February 02, 2024
TIME: 10:30 AM

WSR 24-04-071

Agency: State Board of Health

Effective date of rule:

Emergency Rules

☒ Immediately upon filing.

☐ Later (specify)

Any other findings required by other provisions of law as precondition to adoption or effectiveness of rule?

☐ Yes ☒ No If Yes, explain:

Purpose: On-Site sewage system substitute proprietary treatment product components.

The State Board of Health (board) adopted an emergency rule regarding substitute components of registered products as part of the certification and registration of proprietary treatment products used in on-site sewage systems. The original emergency rule was filed on June 15, 2022 (WSR 22-13-101). Emergency rules have been filed continuously thereafter with the most recent filing on October 6, 2023 (WSR 23-21-061). Only one change has been made to the amendments since the filing of the original emergency rule. This emergency rule is being adopted with a slight change to the previous emergency rule language. The rule language changes "written application" to "written request" to maintain consistent terminology with chapter 246-272A WAC.

This sixth emergency rule amends WAC 246-272A-0110 to allow manufacturers to make a written request to the Department of Health (department) to substitute components of a registered product's construction in cases of a demonstrated supply chain shortage or similar manufacturing disruptions that may impact installations, operation, or maintenance. The request must include information that demonstrates the substituted component will not negatively impact performance or diminish the effect of the treatment, operation, and maintenance of the original registered product. The emergency rule will also allow manufacturers of registered proprietary treatment products to replace components of their products that are not available due to supply chain shortages or similar manufacturing disruptions with like components, as long as the components will not negatively impact performance, treatment, operation, or maintenance of the original registered product.

The current rule requires manufacturers of proprietary treatment products used in on-site sewage systems to test their products with the NSF and register their products with the department based on NSF test results before the product is allowed to be permitted or installed in Washington. Without the emergency rule, the current rule would impede home sales when maintenance of proprietary products has not been completed as noted on home inspections for property transfers because replacement parts with NSF registration are unavailable. New construction is likewise impacted as many active or pending permits include on-site sewage systems using Salcor products. Salcor manufactures a disinfecting ultraviolet (UV) light system incorporated into several proprietary treatment products used in Washington State. There are other manufacturers of disinfecting UV light systems that can be substituted into proprietary treatment products in place of Salcor products. Salcor was sold and the new owner is working with NSF to get their products approved but this process will take several months. In order to continue to protect the public's health, safety, and welfare, it is necessary to adopt a sixth emergency rule to allow the department to consider written requests from manufacturers of proprietary treatment products for substitutes to proprietary treatment product components so their systems will be able to function properly without negatively impacting treatment, operation or maintenance during supply chain shortages. To date, four manufacturers have received department approval to substitute the Salcor 3G UV lamp with an alternate UV lamp.

In 2018, the board filed a CR-101, Preproposal Statement of Inquiry (WSR 18-06-082), to initiate permanent rulemaking and update the on-site sewage system rules. That rulemaking is still underway and is expected to conclude in 2024. As directed by the board at the June 8, 2022 meeting, the emergency rule amendment will be considered for incorporation into the permanent rulemaking that is currently underway.

Citation of rules affected by this order:

New: None

Repealed: None

Amended: WAC 246-272A-0110

Suspended: None

Statutory authority for adoption: RCW 43.20.050(3)

Other authority:

EMERGENCY RULE

Under RCW 34.05.350 the agency for good cause finds:

☒ That immediate adoption, amendment, or repeal of a rule is necessary for the preservation of the public health, safety, or general welfare, and that observing the time requirements of notice and opportunity to comment upon adoption of a permanent rule would be contrary to the public interest.

☐ That state or federal law or federal rule or a federal deadline for state receipt of federal funds requires immediate adoption of a rule.

Reasons for this finding: The board finds that in order to protect the public's health, safety, and welfare it is necessary to adopt the emergency rule to amend WAC 246-272A-0110 to allow the department to consider written request from manufacturers of proprietary treatment products to substitute a proprietary treatment product component so their systems may continue to function properly without negatively impacting performance or diminish the effect of the treatment, operation, or maintenance during supply chain shortages.

Note: If any category is left blank, it will be calculated as zero. No descriptive text.

Count by whole WAC sections only, from the WAC number through the history note. A section may be counted in more than one category.

The number of sections adopted in order to comply with:

Federal statute:	New 0	Amended 0	Repealed 0
Federal rules or standards:	New 0	Amended 0	Repealed 0
Recently enacted state statutes:	New 0	Amended 0	Repealed 0

The number of sections adopted at the request of a nongovernmental entity:

New 0 Amended 0 Repealed 0

The number of sections adopted on the agency's own initiative:

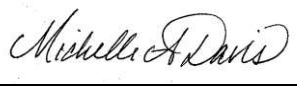
New 0 Amended 1 Repealed 0

The number of sections adopted in order to clarify, streamline, or reform agency procedures:

New 0 Amended 0 Repealed 0

The number of sections adopted using:

Negotiated rule making:	New 0	Amended 0	Repealed 0
Pilot rule making:	New 0	Amended 0	Repealed 0
Other alternative rule making:	New 0	Amended 1	Repealed 0

Date Adopted: February 2, 2024	**Signature:**
Name: Michelle Davis, MPA	
Title: Executive Director Washington State Board of Health	

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0110 Proprietary treatment products—Certification and registration. (1) Manufacturers shall register their proprietary treatment products with the department before the local health officer may permit their use.

(2) To qualify for product registration, manufacturers desiring to sell or distribute proprietary treatment products in Washington state shall:

(a) Verify product performance through testing using the testing protocol established in Table I and register their product with the department using the process described in WAC 246-272-0120;

(b) Report test results of influent and effluent sampling obtained throughout the testing period (including normal and stress loading phases) for evaluation of constituent reduction according to Table II;

(c) Demonstrate product performance according to Table III. All ((~~thirty-day~~)) 30-day averages and geometric means obtained throughout the test period must meet the identified threshold values to qualify for registration at that threshold level; and

(d) For registration at levels A, B, and C verify bacteriological reduction according to WAC 246-272A-0130.

(3) Manufacturers verifying product performance through testing according to the following standards or protocols shall have product testing conducted by a testing facility accredited by ANSI:

(a) ANSI/NSF Standard 40—Residential Wastewater Treatment Systems;

(b) NSF Standard 41: Non-Liquid Saturated Treatment Systems;

(c) NSF Protocol P157 Electrical Incinerating Toilets - Health and Sanitation; or

(d) Protocol for bacteriological reduction described in WAC 246-272A-0130.

(4) Manufacturers verifying product performance through testing according to the following standards or protocols shall have product testing conducted by a testing facility meeting the requirements established by the Testing Organization and Verification Organization, consistent with the test protocol and plan:

(a) EPA/NSF—Protocol for the Verification of Wastewater Treatment Technologies; or

(b) EPA Environmental Technology Verification Program protocol for the Verification of Residential Wastewater Treatment Technologies for Nutrient Reduction.

(5) Treatment levels used in these rules are not intended to be applied as field compliance standards. Their intended use is for establishing treatment product performance in a product testing setting under established protocols by qualified testing entities.

(6) Manufacturers may submit a written request to substitute components of a registered product's construction in cases of supply chain shortage or similar manufacturing disruptions impacting installations, operation, or maintenance. The substitution request must include a report stamped, signed, and dated by a professional engineer demonstrating the substituted component will not negatively impact performance or diminish the effect of the treatment, operation, and

maintenance of the original registered product. If approved, substitution is authorized until rescinded by the department.

TABLE I

Testing Requirements for Proprietary Treatment Products	
Treatment Component/ Sequence Category	**Required Testing Protocol**
Category 1 Designed to treat sewage with strength typical of a residential source when septic tank effluent is anticipated to be equal to or less than treatment level E.	ANSI/NSF 40— Residential Wastewater Treatment Systems (protocols dated between July 1996 and the effective date of these rules)
Category 2 Designed to treat high-strength sewage when septic tank effluent is anticipated to be greater than treatment level E. (Such as at restaurants, grocery stores, mini-marts, group homes, medical clinics, residences, etc.)	EPA/NSF Protocol for the Verification of Wastewater Treatment Technologies/ EPA Environmental Technology Verification (April 2001)
Category 3 Black water component of residential sewage (such as composting and incinerating toilets).	NSF/ANSI Standard 41: Non-Liquid Saturated Treatment Systems (September 1999) NSF Protocol P157 Electrical Incinerating Toilets - Health and Sanitation (April 2000)
Total Nitrogen Reduction in Categories 1 & 2 (Above)	Protocol for the Verification of Residential Wastewater Treatment Technologies for Nutrient Reduction/EPA Environmental Technology Verification Program (November, 2000)

TABLE II

Test Results Reporting Requirements for Proprietary Treatment Products	
Treatment Component/Sequence Category	**Testing Results Reported**
Category 1 Designed to treat sewage with strength typical of a residential source when septic tank effluent is anticipated to be equal to or less than treatment level E.	Report test results of influent and effluent sampling obtained throughout the testing period for evaluation of constituent reduction for the parameters: $CBOD_5$, and TSS:

Test Results Reporting Requirements for Proprietary Treatment Products	
	□ Average □ Standard Deviation □ Minimum □ Maximum □ Median □ Interquartile Range □ 30-day Average (for each month) For bacteriological reduction performance, report fecal coliform test results of influent and effluent sampling by geometric mean from samples drawn within ((~~thirty-day~~)) 30-day or monthly calendar periods, obtained from a minimum of three samples per week throughout the testing period. See WAC 246-272A-0130. Test report must also include the individual results of all samples drawn throughout the test period.
Category 2 Designed to treat high-strength sewage when septic tank effluent is anticipated to be greater than treatment level E. (Such as at restaurants, grocery stores, mini-marts, group homes, medical clinics, residences, etc.)	Report all individual test results and full test average values of influent and effluent sampling obtained throughout the testing period for: $CBOD_5$, TSS and O&G. Establish the treatment capacity of the product tested in pounds per day for $CBOD_5$.
Category 3 Black water component of residential sewage (such as composting and incinerating toilets).	Report test results on all required performance criteria according to the format prescribed in the NSF test protocol described in Table I.
Total Nitrogen Reduction in Categories 1 & 2 (Above)	Report test results on all required performance criteria according to the format prescribed in the test protocol described in Table I.

TABLE III

Product Performance Requirements for Proprietary Treatment Products						
Treatment Component/Sequence Category	**Product Performance Requirements**					
Category 1 Designed to treat sewage with strength typical of a residential source when septic tank effluent is anticipated to be equal to or less than treatment level E.	**Treatment System Performance Testing Levels**					
	Level	**Parameters**				
		$CBOD_5$	**TSS**	**O&G**	**FC**	**TN**
	A	10 mg/L	10 mg/L	——	200/100 ml	——
	B	15 mg/L	15 mg/L	——	1,000/100 ml	——
	C	25 mg/L	30 mg/L	——	50,000/100 ml	——
	D	25 mg/L	30 mg/L	——	——	——
	E	125 mg/L	80 mg/L	20 mg/L	——	——
	N	——	——	——	——	20 mg/L
	Values for Levels A - D are 30-day values (averages for $CBOD_5$, TSS, and geometric mean for FC.) All 30-day averages throughout the test period must meet these values in order to be registered at these levels. Values for Levels E and N are derived from full test averages.					
Category 2 Designed to treat high-strength sewage when septic tank effluent is anticipated to be greater than treatment level E.	All of the following requirements must be met:					

Product Performance Requirements for Proprietary Treatment Products	
Treatment Component/Sequence Category	**Product Performance Requirements**
(Such as at restaurants, grocery stores, mini-marts, group homes, medical clinics, residences, etc.)	(1) All full test averages must meet Level E; and (2) Establish the treatment capacity of the product tested in pounds per day for $CBOD_5$.
Category 3 Black water component of residential sewage (such as composting and incinerating toilets).	Test results must meet the performance requirements established in the NSF test protocol.
Total Nitrogen Reduction in Categories 1 & 2 (Above)	Test results must establish product performance effluent quality meeting Level N, when presented as the full test average.

RULE-MAKING ORDER
PERMANENT RULE ONLY

CR-103P (December 2017)
(Implements RCW 34.05.360)

CODE REVISER USE ONLY

OFFICE OF THE CODE REVISER
STATE OF WASHINGTON
FILED

DATE: March 01, 2024
TIME: 9:06 AM

WSR 24-06-046

Agency: State Board of Health

Effective date of rule:

Permanent Rules

☐ 31 days after filing.

☒ Other (specify)

WAC 246-272A-0110 is effective 31 days after filing.
WAC 246-272A-0340 is effective on February 1, 2025.
All other sections of WAC are effective April 1, 2025.

(If less than 31 days after filing, a specific finding under RCW 34.05.380(3) is required and should be stated below)

Any other findings required by other provisions of law as precondition to adoption or effectiveness of rule?

☐ Yes ☒ No If Yes, explain:

Purpose: On-Site Sewage System. The State Board of Health (board) has adopted amendments to chapter 246-272A WAC to address changes to existing requirements, including requirements governing local management plans, repairs, registration of proprietary treatment products, minimum lot sizes, treatment levels, and licensing of operations and maintenance providers. The adopted rule establishes new requirements, including requirements for field verification of proprietary products, property transfer inspections, remediation, and product supply chain issues. The adopted rule also makes several editorial updates to improve clarity and repeals obsolete rules.

Citation of rules affected by this order:

New: WAC 246-272A-0007, 246-272A-0013, 246-272A-0233, 246-272A-0278, 246-272A-0282
Repealed: WAC 246-272A-0020, 246-272A-0125, 246-272A-0135, 246-272A-0150, 246-272A-0175
Amended: WAC 246-272A-0001, 246-272A-0005, 246-272A-0010, 246-272A-0015, 246-272A-0025, 246-272A-0100, 246-272A-0110, 246-272A-0120, 246-272A-0130, 246-272A-0140, 246-272A-0145, 246-272A-0170, 246-272A-0200, 246-272A-0210, 246-272A-0220, 246-272A-0230, 246-272A-0232, 246-272A-0234, 246-272A-0238, 246-272A-0240, 246-272A-0250, 246-272A-0260, 246-272A-0265, 246-272A-0270, 246-272A-0280, 246-272A-0290, 246-272A-0300, 246-272A-0310, 246-272A-0320, 246-272A-0340, 246-272A-0400, 246-272A-0410, 246-272A-0420, 246-272A-0425, 246-272A-0430, 246-272A-0440
Suspended:

Statutory authority for adoption: RCW 43.20.050(3), chapter 70A.105 RCW, chapter 70A.110 RCW**,** and RCW 43.20.065

Other authority: None

PERMANENT RULE (Including Expedited Rule Making)

Adopted under notice filed as WSR 23-22-062 on 10/25/2023 (date).

Describe any changes other than editing from proposed to adopted version: Several non-significant corrections were made based on comments received during the formal comment period. Some of the changes include spelling, formatting, and grammar corrections. Some changes were technical, provided clarity, and did not change the effect of the rule.

- Consistently fixed 'mL' as the correct abbreviation for milliliter
- Consistently fixed 'E. coli' as the correct abbreviation for Escherichia coli
- Correctly site 'NSF International' standards
- Ensure formatting is in accordance to the Code Reviser's Bill Drafting Guide (2023)
- Added missing table footnotes
- Corrected citations to WAC and table titles
- Non-substantive changes to WAC 246-272A-0010, 246-272A-0100, 246-272A-0110, 246-272A-0120, 246-272A-0230, 246-272A-0280, 246-272A-0300, 246-272A-0420, and 246-272A-0430
- Amended WACs 246-272A-0140(2), 246-272A-0210(1), and 246-272A-0430(4) to replace "shall" with "must" to correctly align with the Code Reviser's Bill Drafting Guide (2023)
- Amended WAC 246-272A-0238(1)(c)(i) to provide clarity to the rule.

If a preliminary cost-benefit analysis was prepared under RCW 34.05.328, a final cost-benefit analysis is available by contacting:

Name: Peter Beaton
Address: Department of Health, PO Box 47824, Olympia WA 98504-7824
Phone: (360) 236-3150
Fax: N/A
TTY: 711
Email: peter.beaton@doh.wa.gov
Web site:
Other:

Note: If any category is left blank, it will be calculated as zero. No descriptive text.

Count by whole WAC sections only, from the WAC number through the history note. A section may be counted in more than one category.

The number of sections adopted in order to comply with:

Federal statute:	New	Amended	Repealed
Federal rules or standards:	New	Amended	Repealed
Recently enacted state statutes:	New	Amended	Repealed

The number of sections adopted at the request of a nongovernmental entity:

	New	Amended	Repealed

The number of sections adopted on the agency's own initiative:

New	5	Amended	36	Repealed	5

The number of sections adopted in order to clarify, streamline, or reform agency procedures:

New	5	Amended	36	Repealed	5

The number of sections adopted using:

Negotiated rule making:	New		Amended		Repealed	
Pilot rule making:	New		Amended		Repealed	
Other alternative rule making:	New	5	Amended	36	Repealed	5

Date Adopted: March 1, 2024	**Signature:**
Name: Michelle Davis, MPA	Michelle A Davis
Title: Executive Director, Washington State Board of Health	

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0001 Purpose, objectives, and authority. (1) The purpose of this chapter is to protect the public health by minimizing:

(a) The potential for public exposure to sewage from on-site sewage systems (OSS); and

(b) Adverse effects to public health that discharges from ((~~on-site sewage systems~~)) OSS may have on ground and surface waters.

(2) This chapter regulates the location, design, installation, operation, maintenance, and monitoring of ((~~on-site sewage systems~~)) OSS to:

(a) Achieve effective long-term sewage treatment and effluent dispersal; and

(b) Limit the discharge of contaminants to waters of the state.

(3) The state board of health is authorized under RCW 43.20.050 to establish minimum requirements for the department of health and local boards of health, and consistent with RCW 43.70.310 integrating the preservation of public health with protection of the environment in order to endorse policies in common.

(4) This chapter is intended to coordinate with other applicable statutes and rules for the design of ((~~on-site sewage systems~~)) OSS under chapter 18.210 RCW and chapter 196-33 WAC.

(5) This chapter is intended to coordinate with other applicable statutes for land use planning under chapters 36.70 and 36.70A RCW, and the statutes for subdivision of land under chapter 58.17 RCW.

(6) The local health officer may designate low-lying marine shorelines in their jurisdiction.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0005 Administration. The local health officers and the department shall administer this chapter under the authority and requirements of chapters 70.05, 70.08, ((~~70.118,~~)) 70.46, 70A.105, 70A.110, and 43.70 RCW. RCW 70.05.060(7) authorizes local health officers to charge fees for the administration of this chapter.

NEW SECTION

WAC 246-272A-0007 Applicability. (1) The local health officer:

(a) Shall apply this chapter to OSS for treatment, siting, design, installation, and operation and maintenance measures treating sewage and dispersing effluent from residential sources with design flows up to 3,500 gallons per day;

(b) May apply this chapter to OSS for nonresidential sources of sewage if treatment, siting, design, installation, and operation and maintenance measures provide treatment and effluent dispersal equal to that required of residential sources;

(c) May not apply this chapter to industrial wastewater.

(2) The department shall apply the requirements of this chapter for the registration of proprietary treatment and distribution products.

(3) A valid OSS design approval, or installation permit issued prior to the effective date of these rules:

(a) Shall be acted upon in accordance with the requirements of this chapter in force at the time of issuance;

(b) Remains valid for a period of not more than five years from the date of approval or issuance, or remains valid for an additional year beyond the effective date of this chapter, whichever has the most lenient expiration date; and

(c) May be modified to include additional requirements if the health officer determines that a serious threat to public health exists.

(4) This chapter does not apply to facilities regulated as reclaimed water use under chapters 90.46 RCW and 173-219 WAC.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0010 Definitions. ((~~(1) Acronyms used in this chapter:~~

~~**"ANSI"** means American National Standards Institute.~~

~~**"BOD"** means biochemical oxygen demand, typically expressed in mg/L.~~

~~**"$CBOD_5$"** means carbonaceous biochemical oxygen demand, typically expressed in mg/L.~~

~~**"FC"** means fecal coliform, typically expressed in number colonies/100 ml.~~

~~**"LOSS"** means a large on-site sewage system (see chapter 246-272B WAC).~~

~~**"NSF"** means National Sanitation Foundation International.~~

~~**"O&G" (formerly referred to as FOG)** means oil and grease, a component of sewage typically originating from food stuffs (animal fats or vegetable oils) or consisting of compounds of alcohol or glycerol with fatty acids (soaps and lotions). Typically expressed in mg/L.~~

~~**"OSS"** means on-site sewage system.~~

~~**"RS&G"** means recommended standards and guidance.~~

~~**"SSAS"** means a subsurface soil absorption system.~~

~~**"TAC"** means the technical advisory committee established in WAC 247-272A-0400.~~

~~**"TN"** means total nitrogen, typically expressed in mg/L.~~

~~**"TSS"** means total suspended solids, a measure of all suspended solids in a liquid, typically expressed in mg/L.~~

~~**"USEPA"** means United States Environmental Protection Agency.~~

~~(2) Definitions used in this chapter:~~))

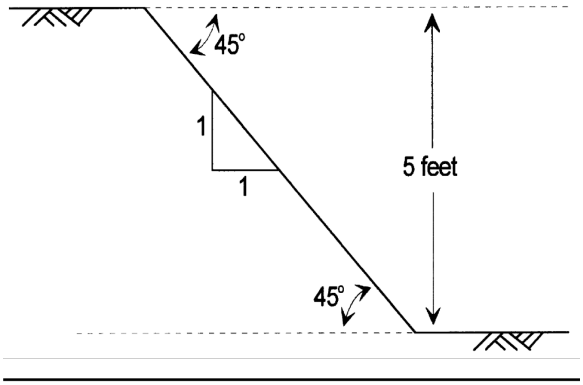
The definitions used in this section apply throughout this chapter unless the context clearly indicates otherwise:

(1) "Additive" means a commercial product added to an ((~~on-site sewage system~~)) OSS intended to affect the performance or aesthetics of an ((~~on-site sewage system~~)) OSS.

(2) "ANSI" means American National Standards Institute.

(3) "Approved" means a written statement of acceptability issued by the local health officer or the department.

(4) "Bank" means any naturally occurring slope greater than 100 percent (45 degrees) and extending vertically at least five feet from the toe of the slope to the top of the slope as follows:



(5) "Bed" means a soil dispersal component consisting of an excavation with a width greater than three feet.

(6) "BL" means bacterial level.

(7) "Black water" means any waste from toilets or urinals.

(8) "BOD" means biochemical oxygen demand, typically expressed in mg/L.

(9) "Building drain" means that part of the lowest piping of a building's drainage system that receives the discharge of sewage from pipes inside the walls of the building and conveys it to the building sewer beginning two feet outside the building wall.

(10) "Building sewer" means that part of the horizontal piping of a drainage system extending from the building drain, which collects sewage from all the drainage pipes inside a building, to an ((~~on-site sewage system~~)) OSS. It begins two feet outside the building wall and conveys sewage from the building drain to the ((~~remaining portions of the on-site sewage system~~)) OSS.

(11) "$CBOD_5$" means carbonaceous biochemical oxygen demand, typically expressed in mg/L.

(12) "Cesspool" means a pit receiving untreated sewage and allowing the liquid to seep into the surrounding soil or rock.

(13) "Conforming system" means any ((~~on-site sewage system~~)) OSS or component, meeting any of the following criteria:

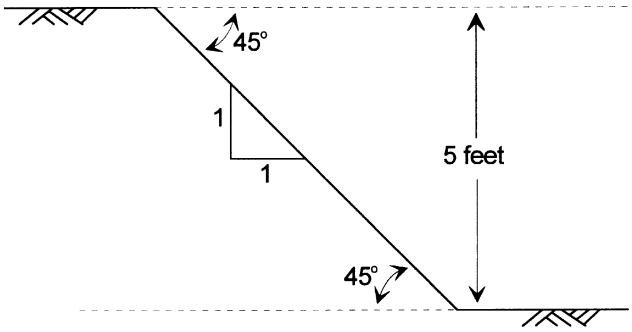
(a) In full compliance with new construction requirements under this chapter; or

(b) Approved, installed, and operating in accordance with requirements of previous editions of this chapter; or

(c) Permitted by the waiver process under WAC 246-272A-0420 ((~~that assures public health protection by higher treatment performance or other methods~~)).

(14) "Cover material" means soil placed over a soil dispersal component composed predominately of mineral material with no greater than ((~~ten~~)) 10 percent organic content. Cover material may contain an organic surface layer for establishing a vegetative landscape to reduce soil erosion.

(15) "Cuts ((~~and/or banks~~))" means any ((~~naturally occurring or~~)) artificially formed slope greater than ((~~one hundred~~)) 100 percent (((~~forty-five~~)) 45 degrees) and extending vertically at least five feet from the toe of the slope to the top of the slope as follows:



(16) "Department" means the Washington state department of health.

(17) "Designer" means a person who matches site and soil characteristics with appropriate on-site sewage technology. Throughout this chapter this term applies to both ((~~on-site sewage treatment system~~)) OSS designers licensed under chapter 18.210 RCW and professional engineers licensed under chapter 18.43 RCW.

(18) "Design flow" means the maximum volume of sewage a residence, structure, or other facility is estimated to generate in a ((~~twenty-four-hour~~)) 24-hour period. It incorporates both an operating capacity and a surge capacity for the ((~~system~~)) OSS during periodic heavy use events. The sizing and design of the ((~~on-site sewage system~~)) OSS components are based on the design flow.

(19) "Detention pond" means an earthen impoundment used for the collection and temporary storage of stormwater runoff.

(20) "Development" means the creation of a residence, structure, facility, subdivision, site, area, or similar activity resulting in the production of sewage.

(21) "Disinfection" means the process of destroying pathogenic microorganisms in sewage through the application of ultraviolet light, chlorination, or ozonation.

(22) "Distribution technology" means any arrangement of equipment ((~~and/~~))or materials that distributes sewage within an ((~~on-site sewage system~~)) OSS.

((~~"Drain field" see subsurface soil absorption system (SSAS) and soil dispersal component.~~))

(23) "Drainrock" means clean washed gravel or crushed rock ranging in size from three-quarters inch to two and one-half inches((~~,~~)) and containing no more than two percent by weight passing a US No. 8 sieve and no more than one percent by weight passing a US No. 200 sieve.

(24) "DS&G" means department standards and guidance.

(25) "*E. coli*" means *Escherichia coli* bacteria. Counts of these organisms are typically used to indicate potential contamination from sewage or to describe a level of needed disinfection, typically expressed as colony forming units/100 mL.

(26) "Effluent" means liquid discharged from a ((~~septic~~)) sewage tank or other ((~~on-site sewage system~~)) OSS component.

(27) "EPA" means United States Environmental Protection Agency.

(28) "Expanding clay" means a clay soil with the mineralogy of clay particles, such as those found in the Montmorillonite/Smectite Group, which causes the clay particles to expand when they absorb water, closing the soil pores, and contract when they dry out.

(29) "Expansion" means a change in a residence, facility, site, or use that:

(a) Causes the sewage quantity or quality to exceed the existing design flow of the ((~~on-site system~~)) OSS, for example, when a residence is increased from two to three bedrooms or a change in use from an office to a restaurant; or

(b) Reduces the treatment or dispersal capability of the existing ((~~on-site sewage system~~)) OSS or the reserve area, for example, when a building is placed over a reserve area.

(30) "Extremely gravelly" means soil with ((~~sixty~~)) 60 percent or more, but less than ((~~ninety~~)) 90 percent rock fragments by volume.

(31) "Failure" means a condition of an ((~~on-site sewage system~~)) OSS or component that threatens the public health by inadequately treating sewage or by creating a potential for direct or indirect contact between sewage and the public. Examples of failure include:

(a) Sewage on the surface of the ground;

(b) Sewage backing up into a structure caused by slow soil absorption of septic tank effluent;

(c) Sewage leaking from a sewage tank or collection system;

(d) Cesspools or seepage pits where evidence of groundwater or surface water quality degradation exists;

(e) Inadequately treated effluent contaminating groundwater or surface water; or

(f) Noncompliance with standards stipulated on the permit.

(32) "Fecal coliform" or "FC" means bacteria common to the digestive systems of warm-blooded animals that are cultured in standard tests. Counts of these organisms are typically used to indicate potential contamination from sewage or to describe a level of needed disinfection((~~. Generally~~)) typically expressed ((~~as colonies per~~)) in colony forming units/100 mL.

(33) "Fill" means unconsolidated material that:

(a) Meets soil types 1-6 textural criteria and is used as part of a soil dispersal component;

(b) Is used to change grade or to enhance surface water diversion; or

(c) Is any other human-transported material.

(34) "Flood plain" means an area that is low-lying and adjacent to a stream or river that is covered by water during a flood.

(35) "GPD" means gallons per day.

(36) "Gravelly" means soils with ((~~fifteen~~)) 15 percent or more, but less than ((~~thirty-five~~)) 35 percent rock fragments by volume.

((~~"Gray water" means sewage from~~)) (37) "Greywater" means sewage from any source in a residence or structure that has not come into contact with toilet or urinal wastes, including bathtubs, showers, bathroom sinks, washing machines, dishwashers, and kitchen sinks. ((~~It includes sewage from any source in a residence or structure that has not come into contact with toilet wastes.~~))

(38) "Groundwater" means subsurface water occupying the zone of saturated soil, permanently, seasonally, or as the result of the tides. Indications of groundwater may include:

(a) Water seeping into or standing in an open excavation from the soil surrounding the excavation or monitoring ports.

(b) Spots or blotches of different color or shades of color interspersed with a dominant color in soil, caused by reduction and oxidation of iron. These color patterns are redoximorphic features, commonly referred to as mottling. Redoximorphic features often indicate the intermittent presence of groundwater and may indicate poor aeration and impeded drainage. ((~~Also see "water table."~~))

(39) "Holding tank sewage system" means an ((~~on-site sewage system which~~)) OSS that incorporates a sewage tank without a discharge outlet, the services of a sewage pumper/hauler, and the offsite treatment and disposal for the sewage generated.

(40) "Hydraulic loading rate" means the amount of effluent applied to a given treatment step, ((~~in this chapter~~)) expressed as gallons per square foot per day or ((~~(~~))gal/sq.ft./day((~~)~~)).

(41) "Industrial wastewater" means the water or liquid carried waste from an industrial process. These wastes may result from any process or activity of industry, manufacture, trade or business, from the development of any natural resource, or from animal operations such as feedlots, poultry houses, or dairies. ((~~The term~~)) Industrial wastewater includes contaminated stormwater and leachate from solid waste facilities.

(42) "Infiltration pond" means an earthen impoundment used for the collection, temporary storage, and infiltration of stormwater runoff.

(43) "Infiltrative surface" means the surface within a treatment component or soil dispersal component to which effluent is applied and through which effluent moves into original, undisturbed soil or other porous treatment media.

(44) "Installer" means a person approved by the local health officer to install ((~~on-site sewage systems~~)) an OSS or OSS components.

(45) "Local health officer" means the health officer of the city, county, or city-county health department or district within the state of Washington, or a representative authorized by and under the direct supervision of the local health officer, as defined in chapter 70.05 RCW.

(46) "LOSS" means a large on-site sewage system under chapter 246-272B WAC.

(47) "Maintenance" means the actions necessary to keep the ((~~on-site sewage system~~)) OSS components functioning as designed.

(48) "Maintenance service provider" means a management entity certified by the local health officer and conducts a comprehensive analysis of an OSS.

(49) "Malfunction" means a damaged or deficient previously conforming OSS component that may be corrected by means of a minor repair.

(50) "Massive structure" means the condition of a soil layer in which the layer appears as a coherent or solid mass not separated into peds of any kind.

(51) "mg/L" means milligrams per liter.

(52) "mL" means milliliter.

(53) "Minimum usable land area" means the minimum land area within the minimum lot size required per development using an OSS, which is based on soil type and type of water supply. Minimum usable land area is free of all physical restrictions and meet minimum vertical and horizontal separations.

(54) "Minor repair" means the repair or replacement of any of the following existing damaged or malfunctioning OSS components except that the repair or replacement of a sewage tank, treatment component, or soil dispersal component is not considered a minor repair:

(a) Control panels;

(b) Building sewers;

(c) Any other portions of tightline in the OSS;

(d) Risers and riser lids;

(e) Sewage tank baffles;

(f) Effluent filters;
(g) Sewage tank pumps and lids;
(h) Pump control floats; and
(i) OSS inspection boxes and ports.

(55) "Moderate structure" means well-formed distinct peds evident in undisturbed soil. When disturbed, soil material parts into a mixture of whole peds, broken peds, and material that is not in peds.

(56) "Modification" means the alteration of an existing OSS component that does not result in an expansion of the system. A modification is not considered a repair.

(57) "Monitoring" means periodic or continuous checking of an ((~~on-site sewage system~~)) OSS, which is performed by observations and measurements, to determine if the system is functioning as intended and if system maintenance is needed. Monitoring also includes maintaining accurate records that document monitoring activities.

((~~"On-site sewage system" (OSS) means an integrated system of components, located on or nearby the property it serves, that conveys, stores, treats, and/or provides subsurface soil treatment and dispersal of sewage. It consists of a collection system, a treatment component or treatment sequence, and a soil dispersal component. An on-site sewage system also refers to a holding tank sewage system or other system that does not have a soil dispersal component.~~))

(58) "NSF" means NSF International.

(59) "O&G" means oil and grease, a component of sewage typically originating from food stuffs such as animal fats or vegetable oils, or consisting of compounds of alcohol or glycerol with fatty acids such as soaps and lotions, typically expressed in mg/L.

(60) "Operating capacity" means the average daily volume of sewage an OSS can treat and disperse on a sustained basis. The operating capacity, which is lower than the design flow, is an integral part of the design and is used as an index in OSS monitoring.

(61) "Ordinary high-water mark" means the mark on lakes, streams, springs, and tidal waters, found by examining the beds and banks and ascertaining where the presence and action of waters are so common and usual, and so long continued in all ordinary years, as to mark upon the soil a character distinct from that of the abutting upland with respect to vegetation, as that condition exists on the effective date of this chapter, or as it may naturally change thereafter. The following ((~~definitions~~)) conditions apply where the ordinary high-water mark cannot be found:

(a) The ordinary high-water mark adjoining marine water is the elevation at mean higher high tide; and

(b) The ordinary high-water mark adjoining freshwater is the line of mean high water.

(62) "OSS" means on-site sewage system, an integrated system of components, located on or nearby the property it serves, which conveys, stores, treats, and provides subsurface soil treatment and dispersal of sewage. It consists of a collection system, a treatment component or treatment component sequence, and a soil dispersal component. An OSS also refers to a holding tank sewage system or other system that does not have a soil dispersal component. The term "on-site sewage system (OSS)" does not include any system regulated by a water quality discharge permit issued under chapter 90.48 RCW.

(63) "PAG" means policy advisory group.

(64) "PDP" means product development permit.

(65) "Ped" means a unit of soil structure such as blocks, column, granule, plate, or prism formed by natural processes.

(66) "Person" means any individual, corporation, company, association, society, firm, partnership, joint stock company, or any governmental agency, or the authorized agents of these entities. For the purposes of WAC 246-272A-0430 and 246-272A-0440, a person is defined to include:

(a) Applicant;

(b) Reapplicant;

(c) Permit holder; or

(d) Any individual associated with (a), (b) or (c) of this subsection including, but not limited to:

(i) Board members;

(ii) Officers;

(iii) Managers;

(iv) Partners;

(v) Association members;

(vi) Agents; and

(vii) Third persons acting with the knowledge of such persons.

(67) "Planned unit development" means a subdivision characterized by a unified site design, clustered residential units ((~~and/~~))or commercial units, and areas of common open space.

(68) "Platy structure" means soil that contains flat peds that lie horizontally and often overlap. This type of structure ((~~will~~)) impedes the vertical movement of water.

(69) "Pressure distribution" means a system of small diameter pipes equally distributing effluent throughout ((~~a SSAS~~)) an OSS, as described in the ((~~department's "*Recommended Standards and Guidance*~~)) *DS&G for Pressure Distribution Systems*,((~~" 2001~~)) 2022. A subsurface drip system ((~~may be used wherever the chapter requires~~)) is considered a pressure distribution system.

(70) "Professional engineer" means a person who is currently licensed as an engineer under the provisions of chapter 18.43 RCW.

(71) "Proprietary product" means a sewage treatment and distribution technology, method, or material subject to a patent or trademark.

(72) "Public domain technology" means a sewage treatment and distribution technology, method, or material not subject to a patent or trademark.

(73) "Public sewer system" means a sewerage system:

(a) Owned or operated by a city, town, municipal corporation, county, or other approved ownership consisting of a collection system and necessary trunks, pumping facilities and a means of final treatment and disposal; and

(b) Approved by or under permit from the department of ecology, the department of health ((~~and/~~)), or a local health officer.

(74) "Puget Sound counties" means Clallam, Island, Kitsap, Jefferson, Mason, San Juan, Seattle-King, Skagit, Snohomish, Tacoma-Pierce, Thurston, and Whatcom. All other counties are defined as non-Puget Sound counties.

(75) "Pump chamber" means a watertight receptacle placed after a septic tank, sewage tank, or other treatment facility that contains the required controls and alarms to convey sewage effluent to a treatment or dispersal component.

(76) "Pumper" means a person approved by the local health officer to remove and transport sewage or septage from ((~~on-site sewage systems~~)) an OSS.

(77) "Record drawing" means an accurate graphic and written record of the location and features of the OSS that are needed to prop-

erly monitor, operate, and maintain that system. Also known as an "as-built" drawing.

(78) "Remediation" means any action, approved by the local health officer, which attempts to restore the function of a previously conforming OSS dispersal component that has failed. Remediation is not considered:

(a) A minor repair;

(b) A repair;

(c) An additive; or

(d) A treatment or distribution technology that allows the OSS to meet a specific treatment level.

(79) "Repair" means the relocation, replacement, or reconstruction of a failed ((~~on-site sewage system~~)) OSS, or any OSS components not included in the list for a minor repair, which have failed in order to restore the OSS to a nonfailure status.

(80) "Reserve area" means an area of land approved for the installation of a conforming ((~~system~~)) OSS that is protected and maintained for replacement of the OSS upon its failure.

(81) "Residential sewage" means sewage having the constituency and ((~~strength~~)) quality typical of ((~~wastewater from domestic households~~)) residential septic tank effluent consistent with treatment level E identified in Table III in WAC 246-272A-0110.

(82) "Restrictive layer" means a stratum impeding the vertical movement of water, air, and growth of plant roots, such as hardpan, claypan, fragipan, caliche, some compacted soils, bedrock, and unstructured clay soils.

(83) "Rock fragment" means rock or mineral fragments having a diameter of two millimeters or more((~~; for example~~)). Examples include, gravel, cobbles, stones, and boulders.

(84) "Seepage pit" means an excavation more than three feet deep where the sidewall of the excavation is designed to dispose of septic tank effluent. Seepage pits ((~~may~~)) are also ((~~be called "dry wells."~~)) known as dry wells.

(85) "Septage" means ((~~the mixture of solid wastes, scum, sludge, and liquids pumped from within septic tanks, pump chambers, holding tanks, and other OSS components~~)) liquid or solid material removed from sewage tanks, cesspools, portable toilets, type III marine sanitation devices, vault toilets, pit toilets, recreational vehicle holding tanks, or similar systems that receive only domestic sewage.

(86) "Septic tank" means a watertight treatment receptacle receiving the discharge of sewage from a building sewer or sewers, designed and constructed to ((~~permit separation of~~)) separate settleable and floating solids from the liquid, detention and anaerobic digestion of the organic matter, prior to discharge of the liquid.

((~~"Septic system" see on-site sewage system or OSS.~~))

(87) "Sewage" means any urine, feces, and the water carrying human wastes, including kitchen, bath, and laundry wastes from residences, buildings, industrial establishments, or other places.

(88) "Sewage quality" means contents in sewage that include:

(a) $CBOD_5$, TSS, and O&G;

(b) Other parameters that ((~~can~~)) may adversely affect treatment. Examples include pH, temperature, and dissolved oxygen; or

(c) Other constituents that create concerns due to specific site sensitivity. Examples include fecal coliform, *E. coli*, phosphorus, and nitrogen.

(89) "Sewage tank" means a prefabricated or cast-in-place septic tank, pump ((~~tank/dosing~~)) chamber, dosing chamber, holding tank, grease interceptor, recirculating filter tank or any other tanks as they relate to ((~~on-site sewage systems~~)) OSS including tanks for use with proprietary products.

(90) "Soil dispersal component" means a technology that releases effluent from a treatment component into the soil for dispersal, final treatment and recycling.

(91) "Soil log" means a detailed description of soil characteristics providing information on the soil's capacity to act as an acceptable treatment and dispersal medium for sewage.

(92) "Soil scientist" means a person certified by the American Society of Agronomy as a Certified Professional Soil Scientist.

(93) "Soil type" means one of seven numerical classifications of fine earth particles and rock fragments as described in WAC 246-272A-0220 (2)(e).

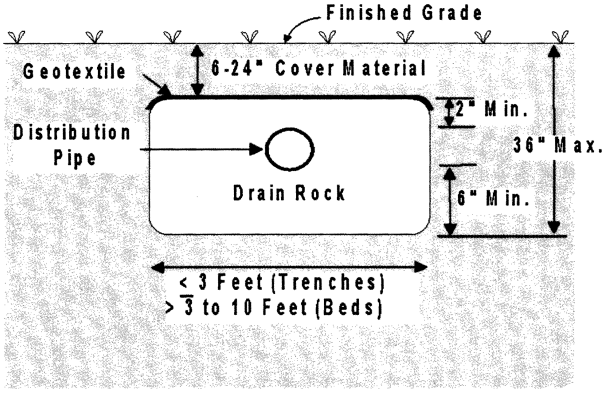
(94) "Standard methods" means the *((~~20th~~)) 23rd Edition of Standard Methods for the Examination of Water and Wastewater*, prepared and published jointly by the American Public Health Association, the American Water Works Association and the Water Environment Federation.

(95) "Strong structure" means peds are distinct in undisturbed soil. They separate cleanly when soil is disturbed, and the soil material separates mainly into whole peds when removed.

(96) "Subdivision" means a division of land or creation of lots or parcels, described under chapter 58.17 RCW, including both long and short subdivisions, planned unit developments, and mobile home parks.

(97) "Subsurface drip system" means an efficient pressurized wastewater distribution system that can deliver small, precise doses of effluent to soil surrounding the drip distribution piping ((~~(called~~)), also known as dripline((~~)~~)), as described in the ((~~department's "*Recommended Standards and Guidance*~~)) *DS&G for Subsurface Drip Systems*, 2020.((~~"~~))

((~~"Subsurface soil absorption system" (SSAS) means~~)) (98) "SSAS" means a subsurface soil absorption system that is a soil dispersal component of trenches or beds containing either a distribution pipe within a layer of drainrock covered with a geotextile, or an approved gravelless distribution technology, designed and installed in ((~~original, undisturbed, unsaturated soil providing at least minimal vertical separation as established in this chapter~~)) suitable soil, with either gravity or pressure distribution of the treatment component effluent.



(99) "Suitable" means original, undisturbed, unsaturated soil of soil types 1-6 with at least the vertical separation established in this chapter.

(100) "Surface water" means any fresh or marine body of water((~~, whether fresh or marine,~~)) flowing or contained in natural or artificial unlined depressions for significant periods of the year, including natural and artificial lakes, ponds, springs, rivers, streams, swamps, marshes, irrigation canals, and tidal waters.

(101) "TAG" means the technical advisory group established in WAC 246-272A-0400.

(102) "Timed dosing" means delivery of discrete volumes of sewage at prescribed time intervals.

(103) "TN" means total nitrogen, typically expressed in mg/L.

(104) "Treatment component" means a technology that treats sewage in preparation for further treatment ((~~and/~~))or dispersal into the soil environment. Some treatment components, such as mound systems, incorporate a soil dispersal component in lieu of separate treatment and soil dispersal components.

(105) "Treatment component sequence" means any series of treatment components that discharges treated sewage to the soil dispersal component.

(106) "Treatment level" means one of ((~~six~~)) the following levels (A, B, C, ((~~D~~)) BL1, BL2, BL3, E, & N) ((~~used in these rules~~)) to:

(a) Identify treatment component performance demonstrated through requirements specified in WAC 246-272A-0110; and

(b) Match site conditions of vertical separation and soil type with treatment components. ((~~Treatment levels used in these rules are not intended to be applied as field compliance standards. Their intended use is for establishing treatment product performance in a product testing setting under established protocols by qualified testing entities.~~

~~"Treatment sequence" means any series of treatment components that discharges treated sewage to the soil dispersal component.~~))

(107) "Trench" means a soil dispersal component consisting of an excavation with a width of three feet or less.

(108) "TSS" means total suspended solids, a measure of all suspended solids in a liquid, typically expressed in mg/L.

(109) "Unit volume of sewage" means:

(a) Flow from a single-family residence;

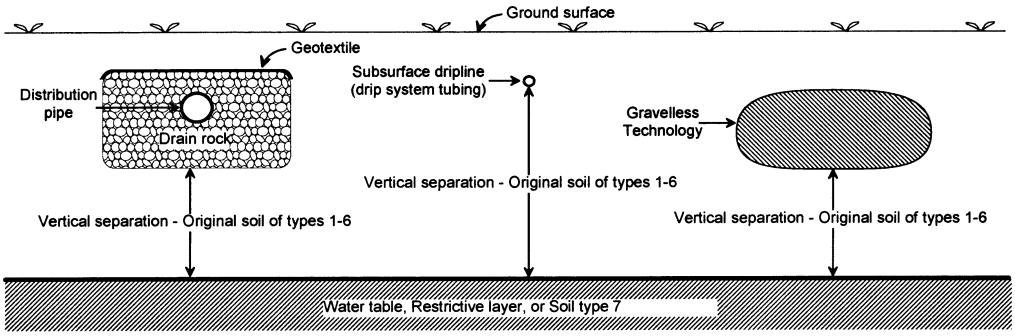
(b) Flow from a mobile home site in a mobile home park; or

(c) Four hundred fifty gallons of sewage per day where the proposed development is not single-family residences or a mobile home park.

(110) "Unknown OSS" means an OSS that was installed without the knowledge or approval of the local health jurisdiction, including those that were installed before such approval was required.

(111) "Unpermitted sewage discharge" means the discharge of sewage or treated effluent from an unknown OSS.

(112) "Vertical separation" means the depth of ((~~unsaturated, original, undisturbed soil of soil types 1-6~~)) suitable soils between the bottom infiltrative surface of a soil dispersal component and the highest seasonal water table, a restrictive layer, or soil type 7 as illustrated below by the profile drawing of subsurface soil absorption systems:



(113) "Very gravelly" means soil containing ((~~thirty-five~~)) 35 percent or more, but less than ((~~sixty~~)) 60 percent rock fragments by volume.

(114) "Water supply protection zone" means the land area around each existing or proposed well site to protect the water supply from contamination.

(115) "Water table" means the upper surface of the groundwater, whether permanent or seasonal. Also see "groundwater" as defined in this section.((")

(116) "Well" means any excavation that is constructed when the intended use of the well is for the location, diversion, artificial recharge, observation, monitoring, dewatering or withdrawal of groundwater for agricultural, municipal, industrial, domestic, or commercial use. ((~~Excluded are~~)) The following are not considered a well:

(a) A temporary observation or monitoring well used to determine the depth to a water table for locating an OSS;

(b) An observation or monitoring well used to measure the effect of an OSS on a water table; ((~~and~~))

(c) An interceptor or curtain drain constructed to lower a water table; and

(d) A dewatering well used temporarily for the purpose of a sewage tank or pump chamber installation.

GENERAL REQUIREMENTS

NEW SECTION

WAC 246-272A-0013 Local rules. (1) The local health officer shall enforce the requirements of this chapter until a local board of health adopts local OSS regulations. A local board of health may adopt and enforce local rules governing OSS when the local regulations are:

(a) Consistent with, and at least as stringent as this chapter; and

(b) Approved by the department prior to the effective date of local regulations.

(2) To apply for department approved local OSS regulations a local board of health shall submit the proposed local regulations to the department.

(3) Within 90 days of receipt of proposed local regulations, the department shall:

(a) Approve the proposed regulations; or

(b) Deny the proposed regulations if the department determines local regulations are not consistent with this chapter or less stringent than this chapter and provide specific reasons for the denial.

(4) Upon receipt of department approval, or after 90 days if the department fails to act, the local board may implement adopted regulations. The local board shall provide a copy of the adopted local regulations to the department.

(5) If the department denies approval of local regulations, the local board of health may:

(a) Resubmit revised regulations that address the specific reasons for the denial for department consideration; or

(b) Submit a request to the department to review its denial within 120 days from the date the local board of health receives the specific reasons for the denial.

(6) Upon receipt of request for review of the department denial, the department shall:

(a) Acknowledge the receipt of the request within 30 days; and

(b) Form a mutually acceptable advisory panel to review the department denial and reach an agreement within a reasonable time. The panel shall consist of:

(i) One representative from the department;

(ii) One representative from a local health jurisdiction other than that which requested the review; and

(iii) One member of the TAG.

(7) If good faith efforts to reach agreement are unsuccessful between the department and a local board of health, the local board of health may appeal the denial to the Washington state board of health for resolution.

(8) Nothing in this chapter shall prohibit the adoption and enforcement of more stringent regulations by a local board of health.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0015 Local management ((~~and regulation~~)) plans. (1) ((~~By July 1, 2007,~~)) The local health officer((~~s of health jurisdictions in the twelve counties bordering~~)) for each Puget Sound county shall develop a written local management plan ((~~that will~~)) to provide guidance to the local health jurisdiction regarding development and management activities for all OSS within the jurisdiction. The ((~~plan~~)) department will review the existing OSS local management plans for all Puget Sound counties within two years of the effective date of the rule. If the department determines a plan revision is necessary upon review, the local health officer shall revise the local management plan for all OSS within the local health jurisdiction consistent with subsection (2) of this section.

(2) At a minimum, the local management plan for Puget Sound counties must specify how the local health jurisdiction will:

(a) Progressively develop and maintain an inventory including the type and location of all known OSS in operation within the jurisdiction;

(b) Identify any areas where OSS could pose an increased public health risk. The following areas shall be given priority in this activity:

(i) Shellfish protection districts or shellfish growing areas;

(ii) Sole source aquifers as designated by the ((~~USEPA~~)) EPA;

(iii) Areas in which aquifers used for potable water as designated under the Washington State Growth Management Act((~~,~~)) under chapter 36.70A RCW are critically impacted by recharge;

(iv) Designated wellhead protection areas ((~~for~~)) in Group A public water ((~~systems~~)) supplies under chapter 246-290 WAC;

(v) Up-gradient areas directly influencing water recreation facilities designated for swimming in natural waters with artificial boundaries within the waters as described by the Water Recreation Facilities Act((~~,~~)) under chapter 70.90 RCW;

(vi) Areas designated ((~~by the department of ecology~~)) as special protection areas under WAC 173-200-090((~~, Water quality standards for groundwaters of the state of Washington~~));

(vii) Wetland areas under production of crops for human consumption;

(viii) Frequently flooded areas including areas delineated by the Federal Emergency Management Agency ((~~and~~)) or as designated under the Washington State Growth Management Act((~~,~~)) under chapter 36.70A RCW;

(ix) Areas where nitrogen has been identified as a contaminant of concern including, but not limited to, the marine waters of Puget Sound; ((~~and~~))

(x) Areas where phosphorous has been identified as a contaminant of concern;

(xi) Areas where sea level rise may impact adequate horizontal separations to surface water; and

(xii) Other areas designated by the local health officer.

(c) Identify operation, maintenance, and monitoring requirements commensurate with risks posed by OSS within the geographic areas identified in (b) of this subsection;

(d) ((~~Facilitate education of homeowners regarding their responsibilities under this chapter and provide operation and maintenance information for all types of systems in use within the jurisdiction;~~

~~(e) Remind and encourage homeowners to complete the operation and maintenance inspections required by WAC 246-272A-0270;~~

~~(f)~~)) Educate OSS owners about their responsibilities to perform OSS operation and maintenance, including information for owners to complete any inspection required by WAC 246-272A-0270;

(e) Maintain records required under this chapter, including ((~~of~~)) all operation and maintenance activities as identified; ((~~and~~))

((~~(g)~~)) (f) Enforce OSS owner permit application, operation, monitoring and maintenance and failure repair requirements ((~~defined~~)) in WAC 246-272A-0200((~~(1)~~)) (2), 246-272A-0260, 246-272A-0270, 246-272A-0275, and 246-272A-0280 ((~~(1) and (2)~~));

((~~(h)~~)) (g) Describe the capacity of the local health jurisdiction to ((~~adequately~~)) fund the local ((~~OSS plan, including~~)) management plan, which includes a summary of program expenditures by activity, source of funds, a strategy to fill any funding gaps, and the ability to find failing and unknown systems; and

((~~(i) Assure that it~~)) (h) Verify that the local management plan was developed ((~~to coordinate~~)) in coordination with the comprehensive

land use plan of the entities governing development ((~~in the health officer's~~)) within the local health jurisdiction.

((~~(2) After being approved by the local board of health following a public hearing, the local health officers required to develop a written plan under subsection (1) of this section shall:~~

~~(a) Supply a copy of the plan to the department;~~

~~(b) Supply a copy of the plan to the entities responsible for land use planning and development regulations in the health officer's jurisdiction; and~~

~~(c) Implement the plan described in subsection (1) of this section.~~

~~(3) The plans of local health jurisdictions required to develop a written plan under subsection (1) of this section shall be submitted to the department by July 1, 2007, and shall be reviewed to ensure the elements described in subsection (1) of this section have been addressed. The department shall provide in writing to the local board of health its review of the completeness of the plan.~~

~~(4) For purposes of this chapter, the local health jurisdictions in marine counties are Clallam, Island, Kitsap, Jefferson, Mason, San Juan, Seattle-King, Skagit, Snohomish, Tacoma-Pierce, Thurston and Whatcom.~~))

(3) The department shall review the local management plan for Puget Sound counties at least once every five years. If the department determines plan revision is necessary upon review of the local management plan described in subsection (2) of this section, the department shall notify the local health officer of their findings.

(4) The local health officer for Puget Sound counties shall:

(a) Review and update the local management plan, as necessary, or at least once every five years;

(b) If after the review the local management plan is updated, provide an opportunity for public input on the local management plan;

(c) Following local board of health approval, submit the local management plan to the department for review;

(d) Implement the local management plan;

(e) Submit an annual report to the department including all of the following in a format specified by the department:

(i) Number of OSS;

(ii) Number of unknown OSS identified;

(iii) Number of failures found;

(iv) Number of failures repaired; and

(v) Status of compliance with inspections required by WAC 246-272A-0270;

(f) Supply a copy of the local management plan to the entities responsible for land use planning and development regulations in the local health jurisdiction.

(5) The local health officer((~~s~~)) for ((~~all other jurisdictions not required to develop a written plan under subsection (1) of this section~~)) a non-Puget Sound county shall develop a written local management plan that will provide guidance to the local health jurisdiction regarding development and management activities for all OSS within the jurisdiction. At a minimum the plan shall include:

(a) A description of the capacity of the local health jurisdiction to provide education and operation and maintenance information for all types of systems in use within the jurisdiction;

(b) A description of how the local health officer will remind and encourage homeowners to complete the operation and maintenance inspection required by WAC 246-272A-0270; and

(c) A description of the capacity of the local health jurisdiction to adequately fund the local OSS plan.

(6) In order to implement the plan described in subsections (1) and (5) of this section, the local health officer shall require the owner of the OSS to:

(a) Comply with additional requirements identified in the plan for the location, design, or performance; and

(b) Comply with the conditions of the operational permit if one is required.

(7) In order to implement the plan described in subsections (1) and (5) of this section, the local health officer may require the owner of the OSS to:

(a) Ensure additional maintenance and monitoring of the OSS;

(b) Provide dedicated easements for inspections, maintenance, and potential future expansion of the OSS; and

(c) Place a notice to title identifying any additional requirements for OSS operation, maintenance, and monitoring((~~; and~~

~~(d) Have an inspection of the OSS at the time of property transfer including the preparation of a "record drawing" if necessary.~~

~~(8) No later than July 1, 2006, the department shall develop guidance on local management programs to assist marine local health jurisdictions in plan development.~~

~~(9) Until such time as the local board of health decides to adopt its own rules, the local health officer shall enforce this chapter. Local boards of health may adopt and enforce local rules and regulations governing on-site sewage systems when the local regulations are:~~

~~(a) Consistent with, and at least as stringent as, this chapter; and~~

~~(b) Approved by the department prior to the effective date of local regulations.~~

~~(10) A local board of health shall apply for departmental approval of local regulations by initiating the following procedure:~~

~~(a) The local board shall submit the proposed local regulations to the department.~~

~~(b) Within ninety days of receipt, the department shall:~~

~~(i) Approve the regulation in writing; or~~

~~(ii) Signify automatic tacit approval with the local regulations and permitting local implementation by failing to act; or~~

~~(iii) Deny approval of the regulations. If the department determines local regulations are not consistent with this chapter, the department shall provide specific reasons for denial.~~

~~(11) Upon receipt of departmental approval or after ninety days without notification, whichever comes first, the local board may implement adopted regulations. The local board shall provide a copy of the adopted local regulations to the department.~~

~~(12) If the department denies approval of local regulations, the local board of health may:~~

~~(a) Resubmit revised regulations for departmental consideration; or~~

~~(b) Submit a written request for a review of the departmental denial within one hundred twenty days from the date the local board of health receives the written reasons for the denial.~~

~~(13) Upon receipt of written request for review of the departmental denial, the department shall:~~

~~(a) Acknowledge the receipt of the request in writing; and~~

~~(b) Form a mutually acceptable advisory panel consisting of:~~

~~(i) One departmental employee;~~

~~(ii) One employee from a local health jurisdiction other than that which requested the review; and~~

~~(iii) One member of the technical advisory committee.~~

~~(14) If good faith efforts to reach agreement are unsuccessful, the local board of health may appeal the denial to the Washington state board of health for resolution.~~

~~(15) Nothing in this chapter shall prohibit the adoption and enforcement of more stringent regulations by local health departments.~~

~~(16) In the plan required in subsection (1) of this section and in local regulations, the local health officer may address water conservation and include options for the nonpotable reuse of gray water. Any treatment and dispersal of gray water outside the residence or structure must comply with this chapter~~)).

(8) The department shall maintain and update guidance and provide technical assistance to assist local health jurisdictions in local management plan development.

((~~GENERAL REQUIREMENTS~~))

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0025 Connection to public sewer system. (1) ((~~When~~)) Upon the failure of an existing OSS within the service area of a sewer utility, the local health officer shall:

(a) Permit the repair or replacement of the OSS only if a conforming OSS can be designed and installed, excluding OSS designed in compliance with or proposing to use Table X in WAC 246-272A-0280; or

(b) Require connection to a public sewer system if the sewer utility allows the connection and has adequate public sewer services ((~~are~~)) available within ((~~two hundred feet of the residence or facility, the local health officer, upon the failure of an existing on-site sewage system may:~~

~~(a) Require hook-up to a public sewer system; or~~

~~(b) Permit the repair or replacement of the on-site sewage system only if a conforming system can be designed and installed.~~

~~(2) Except as noted in subsection (1) of this section, the owner of a failure shall abandon the OSS under WAC 246-272A-0300 and connect the residence or other facility to a public sewer system when:~~

~~(a) The distance between the residence or other facility and an adequate public sewer is two hundred feet or less as measured along the usual or most feasible route of access; and~~

~~(b) The sewer utility allows the sewer connection.~~

~~(3)~~)) 200 feet from where the existing building drain connects to the existing building sewer, or where no building drain exists, within 200 feet from where the sewer line begins, as measured along the usual or most feasible route of access.

(2) The owner of a ((~~residence or other facility~~)) structure served by ((~~a system meeting the requirements of Table IX of this chapter~~)) an OSS permitted as a repair under Table X in WAC 246-272A-0280 shall abandon the OSS ((~~according to the requirements~~)) as specified in WAC 246-272A-0300, and connect the ((~~residence or other facility~~)) structure to a public sewer system when:

(a) Connection is deemed necessary to protect public health by the local health officer;

(b) An adequate public sewer becomes available within ((~~two hundred~~)) 200 feet of the ((~~residence or other facility~~)) existing structure, or in cases where no building drain exists, within 200 feet from where the sewer for the building begins, as measured along the usual or most economically feasible route of access; and

(c) The sewer utility allows the sewer connection.

((~~(4)~~)) (3) Local boards of health may require a new development to connect to a public sewer system to protect public health.

((~~(5)~~)) (4) Local boards of health shall require new development or a development with a failing ((~~system~~)) OSS to connect to a public sewer system if it is required by the comprehensive land use plan or development regulations.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0100 Sewage technologies. (1) The department ((~~may develop recommended~~)) shall maintain standards and guidance ((~~to assist~~)) for local health officers ((~~in permitting different types of~~)) to permit sewage treatment and distribution technologies ((~~including the following four broad categories:~~

~~(a) Public domain treatment technologies (e.g., sand filters);~~

~~(b) Proprietary treatment products (e.g., aerobic treatment systems and packed bed filters);~~

~~(c) Public domain distribution technologies (e.g., gravel or generic gravel substitutes, gravity and pressure distribution methods and materials);~~

~~(d) Proprietary distribution products (e.g., subsurface dripline products or gravelless distribution products)~~)).

(2) ((~~All types of~~)) Before the local health officer permits sewage technologies, the sewage technologies must ((~~have either standards~~)) be registered for use as described in this chapter, have standards for use as described or referenced in this chapter, or ((~~departmental recommended standards and guidance before the local health officer may permit them. Recommended standards and guidance may include information and detail such as:~~

~~(a) Application;~~

~~(b) Design;~~

~~(c) Installation;~~

~~(d) Operation, monitoring and maintenance;~~

~~(e) Performance expectations; and~~

~~(f) Sources of information.~~)) have DS&G describing sewage technologies uses as maintained by the department.

(3) The department may remove, restrict, or suspend a proprietary product's approval for use based on failure to meet required standards or conditions of approval or if the information provided by the manu-

facturer is false, erroneous, or unrepresentative of the approved product.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0110 Proprietary treatment products—((~~Certification and~~)) Eligibility for registration. (1) Manufacturers shall register ((~~their~~)) a proprietary treatment product((~~s~~)) with the department using the process described in WAC 246-272A-0120 before ((~~the~~)) a local health officer may permit ((~~their~~)) use of the product.

(2) To ((~~qualify~~)) be eligible for product registration, manufacturers desiring to sell or distribute proprietary treatment products in Washington state shall:

(a) Verify product performance through testing using the testing protocol established in Table I ((~~and register their product with the department using the process described in WAC 246-272-0120~~)) of this section;

(b) Report product test results of influent and effluent sampling obtained throughout the testing period (including normal and stress loading phases) for evaluation of constituent reduction according to the requirements in Table II of this section;

(c) Demonstrate product performance according to the requirements in Table III of this section. All ((~~thirty-day~~)) 30-day averages and geometric means obtained throughout the test period must meet the identified threshold values to qualify for registration at that threshold level; and

(d) ((~~For registration at levels A, B, and C~~)) Verify bacteriological reduction according to WAC 246-272A-0130 for product registration utilizing bacterial levels BL1, BL2, and BL3.

(3) Manufacturers verifying product performance through testing according to the following standards or protocols shall have product testing conducted by a testing facility accredited by ANSI:

(a) ((~~ANSI/NSF Standard~~)) NSF/ANSI 40((—)): Residential Wastewater Treatment Systems;

(b) ((~~NSF Standard~~)) NSF/ANSI 41: Non-Liquid Saturated Treatment Systems;

(c) NSF Protocol P157 Electrical Incinerating Toilets - Health and Sanitation; ((~~or~~))

(d) ((~~Protocol~~)) NSF/ANSI 245: Residential Wastewater Treatment Systems - Nitrogen Reduction; or

(e) NSF/ANSI 385: Residential Wastewater Treatment Systems - Disinfection Mechanics for Bacteriological Reduction described in WAC 246-272A-0130.

(4) Manufacturers verifying product performance through testing according to ((~~the following standards or protocols shall have product testing conducted by a testing facility meeting the requirements established by the Testing Organization and Verification Organization, consistent with the test protocol and plan:~~

~~(a) EPA/NSF—Protocol for the Verification of Wastewater Treatment Technologies; or~~

~~(b) EPA Environmental Technology Verification Program protocol for the Verification of Residential Wastewater Treatment Technologies~~

~~for Nutrient Reduction.~~)) EPA Method 1664, Revision B and using a wastewater laboratory certified by the Washington department of ecology shall provide supporting information, including flow data, and influent and effluent quality sampling results from a minimum of three installations with similar design loading to demonstrate product performance to Category 2 standards.

(5) Treatment levels ((~~used in these rules are not intended to be applied as field compliance standards. Their intended use is for establishing~~)) established in Table III of this section are intended to establish treatment product performance in a product testing setting under established protocols by qualified testing entities. Field compliance standards for proprietary treatment products shall follow the requirements in WAC 246-272A-0120(5).

(6) Manufacturers may submit a written request to substitute components of a registered product's construction in cases of supply chain shortage or similar manufacturing disruptions impacting installations, operation, or maintenance. The substitution request must include a report stamped, signed, and dated by a professional engineer demonstrating the substituted component will not negatively impact performance or diminish the effect of the treatment, operation, and maintenance of the original registered product. If approved, substitution is authorized until rescinded by the department.

((~~TABLE I~~))

Table I

Testing Requirements for Proprietary Treatment Products	
Treatment Component/Sequence Category	**Required Testing Protocol**
Category 1 Designed to treat ((~~sewage with strength typical of a residential source when~~)) septic tank effluent ((~~is~~)) anticipated to be equal to or less than treatment level E.	((~~ANSI/NSF~~)) NSF/ANSI 40—Residential Wastewater Treatment Systems (((~~protocols~~)) versions dated between ((~~July 1996 and the effective date of these rules~~)) January 2009 and May 31, 2021)
Category 2 Designed to treat ((~~high-strength sewage when septic tank~~)) effluent ((~~is~~)) or sewage with sewage quality parameters anticipated to be greater than treatment level E. (Such as at restaurants, grocery stores, mini-marts, group homes, medical clinics, residences, etc.)	((~~EPA/NSF Protocol for the Verification of Wastewater Treatment Technologies/ EPA Environmental Technology Verification (April 2001)~~)) EPA Method 1664, Revision B (February 2010)
Category 3 Black water component of residential sewage (such as composting* and incinerating** toilets).	NSF/ANSI ((~~Standard~~)) 41: Non-Liquid Saturated Treatment Systems (((~~September 1999~~)) Versions dated between February 2011 and May 31, 2021) **NSF Protocol P157 Electrical Incinerating Toilets - Health and Sanitation (April 2000)
Total Nitrogen Reduction in Categories 1 & 2 (Above)	((~~Protocol for the Verification of Residential Wastewater Treatment Technologies for Nutrient Reduction/EPA Environmental Technology Verification Program (November, 2000)~~)) NSF/ANSI 245: Residential Wastewater Treatment Systems – Nitrogen Reduction (Versions dated between January 2018 and May 31, 2021)

((~~TABLE II~~))

Table II

Test Results Reporting Requirements for Proprietary Treatment Products	
Treatment Component/Sequence Category	**Testing Results Reported**

Test Results Reporting Requirements for Proprietary Treatment Products	
Category 1 Designed to treat ((~~sewage with strength typical of a residential source when~~)) septic tank effluent ((~~is~~)) anticipated to be equal to or less than treatment level E.	Report the following test results of influent and effluent sampling obtained throughout the testing period for evaluation of ((~~constituent~~)) reduction ((~~for the parameters:~~)) of $CBOD_5$[2], and TSS: □ Average □ Standard Deviation □ Minimum □ Maximum □ Median □ Interquartile Range □ 30-day Average (for each month) For evaluation of bacteriological reduction performance((~~,~~)). Report complete treatment component sequence testing as described in Table III, Category 1. For evaluation of performance meeting treatment level BL1: (1) Report fecal coliform test results of influent and effluent sampling by geometric mean from samples drawn within ((~~thirty~~)) 30-day or monthly calendar periods, obtained from a minimum of three samples per week throughout the testing period. See WAC 246-272A-0130. (2) Report complete testing results for supplemental bacteriological reduction technology[1] when the required treatment levels for fecal coliform in Table III, Category 1 are not met by the primary proprietary treatment product. For evaluation of performance meeting treatment level BL2 or BL3: (1) Report fecal coliform test results of influent and effluent sampling by geometric mean from samples drawn within 30-day or monthly calendar periods, obtained from a minimum of three samples per week throughout the testing period as described in WAC 246-272A-0130; or (2) Report complete testing results for supplemental bacteriological reduction technology[1] when the required treatment levels for fecal coliform in Table III, Category 1 are not met by the primary proprietary treatment product. For all options, test report must also include the individual results of all samples drawn throughout the test period.
Category 2 Designed to treat ((~~high-strength sewage when septic tank~~)) effluent ((~~is~~)) or sewage with sewage quality parameters anticipated to be greater than treatment level E. (Such as at restaurants, grocery stores, mini-marts, group homes, medical clinics, atypical residences, etc.)	Report all individual test results and full test average values of influent and effluent sampling obtained throughout the testing period for the evaluation of reduction of: $CBOD_5$, TSS and O&G. Establish the treatment capacity of the product tested in pounds per day for $CBOD_5$.
Category 3 Black water component of residential sewage (such as composting and incinerating toilets).	Report test results on all required performance criteria according to the format prescribed in the NSF test protocol described in Table I.
Total Nitrogen Reduction in Categories 1 & 2 (Above)	Report test results on all required performance criteria according to the format prescribed in the test protocol described in Table I.

[1] Test results for BOD_5 may be submitted in lieu of test results for $CBOD_5$. In these cases numerical values for $CBOD_5$ will be determined using the following formula: ($BOD_5 \times 0.83 = CBOD_5$).

[2] Supplemental bacteriological reduction technology must be tested for influent/effluent fecal coliform or *E. coli* per WAC 246-272A-0130 (bacteriological reduction testing protocol). Supplemental fecal coliform or *E. coli* reducing technologies will be rated for log base 10 removal of fecal coliform or *E. coli*. The lowest 30-day geometric mean will be used to rate reduction level. The highest monthly geometric mean for treatment technology fecal coliform or *E. coli* reduction will be used as the baseline value for review.

((~~TABLE III~~))

Table III

((**Product Performance Requirements for Proprietary Treatment Products**

TreatmentComponent/Sequence Category	**Product Performance Requirements**
Category 1 Designed to treat sewage with strength typical of a residential source when septic tank effluent is anticipated to be equal to or less than treatment level E.	**Treatment System Performance Testing Levels**

Level	**Parameters**				
	$CBOD_5$	**TSS**	**O&G**	**FC**	**TN**
A	10 mg/L	10 mg/L	——	200/100 ml	——
B	15 mg/L	15 mg/L	——	1,000/100 ml	——
C	25 mg/L	30 mg/L	——	50,000/100 ml	——
D	25 mg/L	30 mg/L	——	——	——
E	125 mg/L	80 mg/L	20 mg/L	——	——
N	——	——	——	——	20 mg/L

Values for Levels A - D are 30-day values (averages for $CBOD_5$, TSS, and geometric mean for FC.) All 30-day averages throughout the test period must meet these values in order to be registered at these levels.
Values for Levels E and N are derived from full test averages.

Category 2 Designed to treat high-strength sewage when septic tank effluent is anticipated to be greater than treatment level E. (Such as at restaurants, grocery stores, mini-marts, group homes, medical clinics, residences, etc.)	All of the following requirements must be met: (1) All full test averages must meet Level E; and (2) Establish the treatment capacity of the product tested in pounds per day for $CBOD_5$.
Category 3 Black water component of residential sewage (such as composting and incinerating toilets).	Test results must meet the performance requirements established in the NSF test protocol.
Total Nitrogen Reduction in Categories 1 & 2 (Above)	Test results must establish product performance effluent quality meeting Level N, when presented as the full test average.))

Product Performance Requirements for Proprietary Treatment Products

Treatment Component/Sequence Category	**Product Performance Requirements**
Category 1 Designed to treat effluent anticipated to be equal to or less than treatment level E.	**Treatment System Performance Testing Levels**

Level	**Parameters**					
	$CBOD_5$ mg/L	**TSS mg/L**	**O&G mg/L**	**FC cfu/100 mL**	**TN mg/L**	***E. coli* cfu/100 mL**
A	10	10	——	——	——	——
B	15	15	——	——	——	——
C	25	30	——	——	——	——
BL1			——	200	——	126

Product Performance Requirements for Proprietary Treatment Products							
Treatment Component/Sequence Category	**Product Performance Requirements**						
	BL2	____	____	____	1,000	____	____
	BL3	____	____	____	50,000	____	____
	E	228	80	20	____	____	____
	N	____	____	____	____	30 (or 50% reduction based on mass loading as required in WAC 246-272A-0320)	____
	Values for Levels A - C are 30-day values (averages for $CBOD_5$, TSS, and geometric mean for FC.) All 30-day averages throughout the test period must meet these values in order to be registered at these levels. Values for Levels E and N are derived from full test averages.						
Category 2 Designed to treat high-strength sewage when septic tank effluent is anticipated to be greater than treatment level E. (Such as at restaurants, grocery stores, mini-marts, group homes, medical clinics, residences, etc.)	All of the following requirements must be met: (1) All full test averages must meet Level E; and (2) Establish the treatment capacity of the product tested in pounds per day for $CBOD_5$.						
Category 3 Black water component of residential sewage (such as composting and incinerating toilets).	Test results must meet the performance requirements established in the NSF test protocol.						
Total Nitrogen Reduction in Categories 1 & 2 (Above)	Test results must establish product performance effluent quality meeting Level N, when presented as the full test average.						

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0120 Proprietary treatment product registration—Process and requirements. (1) Manufacturers shall register ((~~their~~)) proprietary treatment ((~~product(s)~~)) products with the department by submitting a complete registration application for review and approval in the format provided by the department, including:

(a) Manufacturer's name, mailing address, ((~~street address and~~)) phone number, email address, and website address;

(b) Contact ((~~individual's~~)) person's name, title, mailing address, ((~~street~~)) email address, and phone number. The contact ((~~individual~~)) person must be vested with the authority to represent the manufacturer in this capacity;

(c) Name, including specific brand and model, of the proprietary treatment product;

(d) A description of the function of the proprietary treatment product along with any known limitation on the use of the product;

(e) Product description and technical information, including process flow drawings and schematics; materials and characteristics; component design specifications; design capacity, volumes and flow assumptions and calculations; components; dimensioned drawings and photos;

(f) For treatment systems in Category 2, daily capacity of the model or models in pounds per day of $CBOD_5$;

(g) Siting and installation requirements;

(h) Detailed description, procedure, and schedule of routine service and system maintenance events;

(i) Estimated operational costs for the first five years of the treatment component's life. This ((~~shall~~)) must include both estimated annual electricity costs, and routine maintenance costs, including replacement of parts;

(j) Identification of information subject to protection from disclosure of trade secrets;

(k) Most current dated copies of product brochures ((~~&~~)) and manuals: *Sales & Promotional; Design; Installation; Operation & Maintenance; and Homeowner Instructions*;

(l) The most recently available product test protocol dated no earlier than the dates in WAC 246-272A-0110 Table I and the results report;

(m) A signed and dated certification by the manufacturer's agent specifically including the following statement, "I certify that I represent (INSERT MANUFACTURING COMPANY NAME) and I am authorized to prepare or direct the preparation of this application for registration. I attest, under penalty of law, that this document and all attachments are true, accurate, and complete. I understand and accept that the product testing results reported with this application for registration are the parameters and values to be used for determining conformance with Treatment System Performance Testing Levels established in chapter 246-272A WAC";

(n) A signed and dated certification from the testing entity including the statement, "I certify that I represent (INSERT TESTING ENTITY NAME), that I am authorized to report the testing results for this proprietary treatment product. I attest, under penalty of law, that the report about the test protocol and results is true, accurate, and complete"; and

(o) The fee described in WAC ((~~246-272A-990~~)) 246-272-2000.

(2) Products within a single series or model line, ((~~,~~))sharing distinct similarities in design, materials, and capacities((~~,~~)), may be registered under a single application, consistent with the provisions of their test protocol for the certification of other products within a product series. Products outside of the series or model line must be registered under separate applications.

(3) Upon receipt of ((~~an~~)) a registration application the department shall:

(a) Verify that the application is complete including dated and current copies of all of the required manuals; and

(b) If ((~~complete~~)) approved, place the product on the department's list of ((~~proprietary~~)) registered on-site treatment and distribution products.

(4) All registrations are valid for up to one year, expiring on December 31st of each year. Fees are not prorated.

(5) In order to renew a proprietary treatment product technology registration, a manufacturer shall:

(a) Apply for renewal of product registration using the ((~~form or in the~~)) format provided by the department((~~.~~));

(b) Submit ((~~the results of~~)) any of the following applicable reports:

(i) A retesting((~~, if the product has completed retesting~~)) report from the testing entity according to the protocol required for registration ((~~and a report from the testing entity has been issued since initial registration or previous renewal. Renewal shall be based on the most recent test results.~~)) as identified in this section;

(ii) A field verification performance report as identified in the proprietary on-site wastewater treatment products DS&G, dated February 1, 2025. If field performance results demonstrate that the product has failed to meet the requirements in the DS&G, the manufacturer shall report to the department describing the reasons for the failure to meet the requirements consistent with the DS&G;

(c) Provide an ((~~affidavit~~)) attestation to the department verifying whether or not the product has changed over the previous year. If the product has changed, the ((~~affidavit~~)) attestation must also include a full description of the changes. If the product has changed in a way that affects performance, the product may not be renewed and shall meet the requirements for initial registration((~~.~~));

(d) Provide a statement that all required dated manuals are current, or submit the updated and dated new manuals; and

(e) Submit the fee established in WAC ((~~246-272A-990~~)) 246-272-2000.

(6) As part of product registration renewal, the department shall:

(a) Request field assessment comments from local health officers no later than October 31st of each year. These comments may include concerns about a variety of field assessment issues, including:

(i) Product function, including verification of field performance testing as identified in the DS&G;

(ii) Product reliability((~~,~~));and

(iii) Problems arising with operation and maintenance;

(b) Discuss with the ((~~TAC~~)) TAG any field assessment information that may impact product registration renewal;

(c) Notify the manufacturer of any product to be discussed with the ((~~TAC~~)) TAG, prior to discussion with the ((~~TAC~~)) TAG, regarding the nature of comments received; ((~~and~~))

(d) Renew the product registration unless:

(i) The manufacturer of a product does not apply for renewal; or

(ii) The department, after deliberation with the ((~~TAC~~)) TAG, concludes product registration renewal should not be given or should be delayed until the manufacturer submits information that satisfactorily answers concerns and issues; and

(e) Provide a compliance plan to the manufacturer within 90 days based on departmental concerns of public health risk related to the product.

(7) The department shall maintain a list of ((~~proprietary treatment~~)) registered on-site treatment and distribution products meeting the registration requirements established in this chapter. The product registration is a condition of approval for use.

(8) Manufacturers shall have readily accessible product information for designers, ((~~homeowners,~~)) regulators, ((~~system~~)) OSS owners

and other interested parties ((~~about their product~~)) posted on the manufacturer's website including the most current dated version of:

(a) Product manuals;

(b) Design instructions;

(c) Installation instructions;

(d) Operation and maintenance;

(e) ((~~Homeowner~~)) Owner instructions; and

(f) How to locate a list of representatives and manufacturer certified maintenance service providers, if any.

AMENDATORY SECTION (Amending WSR 06-01-020, filed 12/12/05, effective 1/12/06)

WAC 246-272A-0130 Bacteriological reduction. This section establishes the requirements for registering bacteriological reduction processes.

(1) Manufacturers shall, for the purpose of product registration as described in WAC 246-272A-0110 and 246-272A-0120 ((~~for meeting treatment levels A, B, or C, verify bacteriological reduction performance by sampling for fecal coliform.~~

~~(a) For products not yet tested according to ANSI/NSF Standard 40 testing protocol dated July 1996 or later, the requirements of both ANSI/NSF Standard 40 and the protocol specified in subsection (2) of this section for verifying bacteriological reduction must be met.~~

~~(b) For products that have been tested according to ANSI/NSF Standard 40 dated July 1996 or later but have not yet been tested for bacteriological reduction, treatment performance of the treatment product or sequence may be established based on test results for $CBOD_5$ and TSS obtained from the previous ANSI/NSF Standard 40 testing and bacteriological reduction performance based on testing according to the protocol in subsection (2) of this section. Provided that the testing entity must verify the influent wastewater stream throughout the bacteriological testing period meets the influent threshold levels for $CBOD_5$ and TSS required by ANSI/NSF Standard 40 testing protocol~~)):

(a) For meeting treatment level BL1, verify bacteriological reduction performance by sampling for fecal coliform or *E. coli*.

(b) For meeting treatment level BL2 or BL3, verify bacteriological reduction performance by sampling for fecal coliform.

(2) All test data submitted for product registration shall be produced by an ANSI accredited, third-party testing and certification organization whose accreditation is specific to on-site wastewater treatment products. Bacteriological reduction performance must be determined ((~~while~~)) either:

(a) According to the procedures in NSF/ANSI 385 for supplemental bacteriological reduction; or

(b) Concurrent with testing protocol. The treatment product or treatment component sequence ((~~is tested~~)) testing according to the ((~~ANSI/NSF Standard~~)) NSF/ANSI 40 testing protocol. ((~~During this~~))

(3) Testing under subsection (2)(b) of this section shall be completed in compliance with the following requirements ((~~apply~~)):

(a) Collect samples from both the influent and effluent streams, identifying the treatment performance achieved by the full treatment process, ((~~(~~))component or sequence((~~)~~));

(b) Obtain influent characteristics falling within a range of $10^{((6))}$ ^{4} - 10^{8} fecal coliform/100 mL or 10^{2} - 10^{6} *E. coli*/100 mL calculated as ((~~thirty~~)) 30-day geometric means during the test((~~.~~));

(c) Test the influent to any disinfection unit and report the following at each occasion of sampling performed in (d) of this subsection:

(i) Flow rate;

(ii) pH;

(iii) Temperature;

(iv) Turbidity; and

(v) Color((~~.~~));

(d) Obtain samples for fecal coliform or *E. coli* analysis during both the design loading and stress loading periods identified by ((~~NSF Standard~~)) NSF/ANSI 40. Grab samples shall be collected from both the influent and effluent on three separate days of the week. Each set of influent and effluent grab samples must be taken from a different dosing time frame, either ((~~(~~))morning, afternoon, or evening((~~)~~)), so that samples have been taken from each dosing time frame by the end of the week((~~.~~));

(e) Conduct analyses according to standard methods;

(f) Report the geometric mean of fecal coliform or *E. coli* test results from all samples taken within ((~~thirty~~)) 30-day or monthly calendar periods;

(g) Report the individual results of all samples taken throughout the test period design and stress loading; and

(h) Report all maintenance and servicing conducted during the testing period, including for example, instances of cleaning a UV lamp, or replenishment of chlorine chemicals.

((~~(3)~~)) (4) Manufacturers may register products in treatment levels ((~~A~~)) BL1 and ((~~B~~)) BL2 using disinfection.

((~~(4)~~)) (5) Manufacturers may not register products for treatment level ((~~C~~)) BL3 using disinfection.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0140 Proprietary distribution products—Certification ((~~and registration~~)) requirements. (1) ((~~Manufacturers shall register proprietary distribution products, including gravelless distribution products and subsurface dripline products, with the department before the local health officer may permit their use.~~

~~(2) Manufacturers desiring to sell proprietary distribution products shall certify that the product(s) meets the standards established in this chapter and register their product(s) with the department using the process described in WAC 246-272A-0145.~~

~~(3)~~)) Proprietary distribution products, including gravelless distribution products and subsurface dripline products, must be registered with the department before permitting, sale, and use. To be eligible for registration as described in WAC 246-272A-0145, products must first be certified as described in this section.

(2) To be certified, proprietary gravelless distribution products ((~~shall~~)) must:

(a) Be constructed or manufactured from materials that are nondecaying and nondeteriorating and do not leach chemicals when exposed to sewage and the subsurface soil environment;

(b) Provide liquid storage volume at least equal to the storage volume provided within the ((~~thirty~~)) 30 percent void space in a ((~~twelve~~)) 12-inch layer of drainrock in a drainrock-filled distribution system. This storage volume must be established by the gravelless distribution products, ((~~system~~)) OSS design and installation and must be maintained for the life of the ((~~system~~)) OSS. This requirement may be met on a lineal-foot, or on an overall system design basis;

(c) Provide ((~~suitable~~)) effluent distribution to the infiltrative surface at the soil interface; and

(d) Maintain the integrity of the trench or bed. The material used, by its nature and its manufacturer-prescribed installation procedure, must withstand the physical forces of the soil sidewalls, soil backfill, and the weight of equipment used in the backfilling.

((~~(4)~~)) (3) Proprietary subsurface dripline products shall:

(a) Be warranted by the manufacturer for use with sewage and for resistance to root intrusion((~~.~~));

(b) Incorporate emitters with a maximum nominal rated discharge of 1.3 gallons per hour. Emitter discharge rate may be controlled either by use of pressure-compensating emitters or with a pressure regulator((~~.~~)); and

(c) Be color-coded purple to identify that the pipe contains nonpotable water from a sewage source.

(4) To be certified by the department, the manufacturer must submit:

(a) A signed and dated statement by the manufacturer's agent specifically including the following statement, "I certify that I represent (INSERT MANUFACTURING COMPANY NAME) and I am authorized to prepare or direct the preparation of this application for product registration. I attest, under penalty of law, that this document and all attachments, are true, accurate, and complete."

(b) A signed and dated statement from the licensed professional engineer including the statement, "I certify that I represent (INSERT PROFESSIONAL ENGINEERING FIRM NAME), that I am authorized to certify the performance characteristics for the proprietary distribution product presented in this application. I attest, under penalty of law, that the technology report is true, accurate, and complete."

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0145 Proprietary distribution product registration ——Process and requirements. (1) Manufacturers shall register their proprietary distribution ((~~product(s)~~)) products with the department by submitting a complete application for review and approval in the format provided by the department, including:

(a) Manufacturer's name, mailing address, ((~~street address, and~~)) phone number, email address, and website address;

(b) Contact ((~~individual's~~)) person's name, title, mailing address, ((~~street~~)) email address, and phone number. The contact ((~~indi-~~

~~vidual~~)) person must be vested with the authority to ((~~act as~~)) represent the agent of the manufacturer in this capacity;

(c) Name, including specific brand and model, of the proprietary distribution product;

(d) A description of the function of the proprietary distribution product along with any known limitations on ((~~its~~)) the use of the product;

(e) Product description and technical information, including schematics; materials and characteristics; component design specifications; design capacity, volumes and flow assumptions and calculations; components; dimensioned drawings and photos;

(f) Siting and installation requirements;

(g) Detailed description, procedure, and schedule of routine service and system maintenance events;

(h) Identification of information subject to protection from disclosure of trade secrets;

(i) Most current, dated copies of product brochures and manuals: *Sales & Promotional; Design; Installation; Operation & Maintenance; and ((~~Homeowner~~)) Owner Instructions*;

(j) For gravelless chamber systems a quantitative description of the actual exposed trench-bottom infiltrative surface area for each model seeking registration;

(k) A statement from a professional engineer that certifies the technology meets the standards established in WAC 246-272A-0140;

(l) ((~~A signed and dated certification by the manufacturer's agent specifically including the following statement, "I certify that I represent (INSERT MANUFACTURING COMPANY NAME) and I am authorized to prepare or direct the preparation of this application for product registration. I attest, under penalty of law, that this document and all attachments, are true, accurate, and complete."~~

~~(m) A signed and dated certification from the licensed professional engineer including the statement, "I certify that I represent (INSERT PROFESSIONAL ENGINEERING FIRM NAME), that I am authorized to certify the performance characteristics for the proprietary distribution product presented in this application. I attest, under penalty of law, that the technology report is true, accurate, and complete."~~

~~(n)~~)) The fee established in WAC ((~~246-272A-0990~~)) 246-272-2000.

(2) Products within a single series or model line, ((~~(~~))sharing distinct similarities in design, materials, and capacities((~~)~~)), may be registered under a single application. Products outside of the series or model line must be registered under separate applications.

(3) Upon receipt of an application the department shall:

(a) Verify that the application is complete, including dated and current copies of all required manuals; and

(b) If ((~~complete~~)) approved, place the product on the list of ((~~proprietary~~)) registered on-site treatment and distribution products.

(4) All registrations are valid for up to one year, expiring on December 31st of each year. Required fees are not prorated.

(5) In order to renew a proprietary distribution product registration, a manufacturer ((~~must~~)) shall:

(a) Apply for renewal of product registration using the form or in the format provided by the department;

(b) Provide an ((~~affidavit~~)) attestation to the department verifying whether or not the product has changed over the previous year. If the product has changed, the ((~~affidavit~~)) attestation must also include a full description of the changes. If the product has changed

in a way that affects performance, the product may not be renewed and shall meet the requirements of initial registration; ((~~and~~))

(c) Provide a statement that all required dated manuals are current, or submit the updated and dated new manuals; and

(d) Submit the fee established in WAC ((~~246-272A-0990~~)) 246-272-2000.

(6) As part of product registration renewal, the department ((~~shall~~)) will:

(a) Request field assessment comments from local health officers ((~~no later than October 31st~~)) before November 1st of each year. These comments may include concerns about a variety of field assessment issues, including product function, product reliability, and problems arising with operation and maintenance;

(b) Discuss with the ((~~TAC~~)) TAG any field assessment information that may impact product registration renewal;

(c) Notify the manufacturer of any product to be discussed with the ((~~TAC~~)) TAG, prior to discussion with the ((~~TAC~~)) TAG, regarding the nature of comments received; ((~~and~~))

(d) Renew the product registration unless:

(i) The manufacturer of a product does not apply for renewal; or

(ii) The department, after deliberation with the ((~~TAC~~)) TAG, concludes product registration renewal should not be given or should be delayed until the manufacturer submits information that satisfactorily answers concerns and issues; and

(e) Provide a compliance plan to the manufacturer within 90 days based on departmental concerns of public health risk related to the product.

(7) The department shall maintain a list of proprietary distribution products meeting the registration requirements established in this chapter. The product registration is a condition of approval for use.

(8) Manufacturers shall have readily accessible product information for designers, ((~~homeowners,~~)) regulators, ((~~system~~)) OSS owners and other interested parties ((~~about their product~~)) posted on the manufacturer's website including the most current dated version of:

(a) Product manuals;

(b) Design instructions;

(c) Installation instructions;

(d) Operation and maintenance;

(e) ((~~Homeowner~~)) Owner instructions; and

(f) How to locate a list of representatives and manufacturer certified maintenance service providers, if any.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0170 Product development permits. (1) A local health officer may issue a ((~~product development permit (PDP)~~)) PDP for any proprietary treatment component or sequence to be used during a development period. ((~~In order~~)) To protect public health during the development period, a complete ((~~system~~)) OSS meeting the requirements of this chapter and the site must already be installed. The ((~~product~~)) component or sequence under development may then be added to the treatment system allowing the ((~~product~~)) developer to gather data

about ((~~the product's~~)) performance in the field. The PDP allows ((~~product~~)) developers to explore ((~~and develop~~)) new technologies prior to product testing and registration under WAC 246-272A-0110 and 246-272A-0120. The PDP is not an alternative to testing and registration.

(2) An ((~~application~~)) applicant for a PDP ((~~shall include~~)) must submit an application to the local health officer including all of the following:

(a) Proof of an existing conforming ((~~system~~)) OSS in compliance with all local requirements, or a permit for a conforming ((~~system~~)) OSS. The conforming ((~~system~~)) OSS must be installed in its entirety before the PDP becomes valid;

(b) A description of the product under development including performance goals and a description of how the system will be used to treat sewage;

(c) ((~~Documentation of~~)) Financial assurance ((~~that will cover~~)) covering the correction of any potential public health threats or environmental damage resulting from the use of the product under development. Instruments of financial assurance include:

(i) An irrevocable letter of credit in the amount required by the local health officer issued by an entity authorized to issue letters of credit in Washington state;

(ii) Cash or security deposit payable to the local health jurisdiction in the amount required by the local health officer; or

(iii) Any other financial assurance that satisfies the local health officer.

(d) Documentation signed by the owner of the proposed product development site allowing access to the local health officer for inspection of the site; and

(e) Any other information required by the local health officer.

(3) The local health officer may ((~~stipulate~~)) impose additional requirements for a PDP necessary to ((~~assure~~)) safeguard the performance of the conforming ((~~system~~)) OSS, including providing performance data to the local health officer.

(4) A PDP is a site-specific permit. Product development at multiple sites requires a PDP for each site.

(5) During the term of the PDP, product development, testing and sampling are under the full control of the product developer and all data collected is considered proprietary information.

(6) A PDP is valid for one year and may be renewed by the local health officer.

(7) The product development period is over when the original PDP or any subsequently renewed permits have expired. At this time, the product developer:

(a) Shall, at the direction of the local health officer, remove the product under development from the site, reestablishing all appropriate plumbing and power connections for the conforming ((~~system~~)) OSS.

(b) May subject the product to performance testing described in WAC 246-272A-0110 ((~~in order~~)) to allow the product to be eligible for registration with the department.

(8) The local health officer may revoke or amend a PDP:

(a) If the continued operation or presence of the product under development:

(i) Presents a risk to ((~~the~~)) public health or the environment;

(ii) Causes adverse effects on the proper function of the conforming ((~~system~~)) OSS on the site; or

(iii) Leaks or discharges sewage on the surface of the ground.

(b) If the developer fails to comply with any requirements stipulated on the permit by the local health officer.

(9) The local health officer may charge fees adequate to administer the PDP program.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0200 Permit requirements. (1) ((~~Prior to beginning the construction process~~)) A permit is not required for a minor repair. The local health officer may require the owner to submit information regarding any activities defined as a minor repair for record-keeping purposes.

(2) Except for a minor repair, a person proposing the installation, repair, modification, connection to, or expansion of an OSS, shall ((~~report the following~~)) submit an application and obtain a permit from the local health officer prior to beginning construction. The permit application must include the following:

(a) General information including:

(i) Name and address of the property owner and the applicant at the head of each page of the submission;

(ii) Parcel number and if available, the address of the site;

(iii) Source of drinking water supply;

(iv) Identification if the property is within the boundaries of a recognized sewer utility;

(v) Size of the parcel;

(vi) Type of permit for which application is being made((~~,~~)). For example, new installation, repair, expansion, modification, or operational;

(vii) Source of sewage((~~,~~)). For example, residence, restaurant, or other type of business;

(viii) Location of utilities;

(ix) Name of the site evaluator;

(x) Name, signature and stamp of the designer;

(xi) Date of application; and

(xii) Name and signature of the fee simple owner, the contract purchaser of the property, or the owner's authorized agent.

(b) The soil and site evaluation as specified under WAC 246-272A-0220((~~.~~));

(c) A dimensioned site plan of the proposed initial ((~~system~~)) OSS, the reserve area and those areas immediately adjacent that contain characteristics impacting design including:

(i) Designated areas for the proposed initial ((~~system~~)) OSS and the reserve area;

(ii) The location of all soil logs and other soil tests for the OSS;

(iii) General topography and((~~/or~~)) slope;

(iv) Drainage characteristics;

(v) Horizontal separations as noted in Table IV in WAC 246-272-0210;

(vi) The location of existing and proposed encumbrances affecting ((~~system~~)) OSS placement, including legal access documents if any com-

ponent of the OSS is not on the lot where the sewage is generated; ((~~and~~

~~(vi)~~)) (vii) An arrow indicating north;

(viii) A legend of symbols used;

(ix) Plan scale and a graphic scale bar;

(x) Vertical datum used (such as "assumed," "North American Vertical Datum of 1988 (NAVD 88)," "National Shoreline Reference Station (NSRS)," or "unknown");

(xi) An elevation benchmark and relative elevations of system components;

(xii) Name, signature, stamp, and contact information of the designer; and

(xiii) A statement on limitation of use indicating the site plan is not a survey.

(d) A detailed ((~~system~~)) OSS design meeting the requirements under WAC 246-272A-0230, 246-272A-0232, 246-272A-0234, and 246-272A-0238 including:

(i) A drawing showing the dimensioned location of components of the proposed OSS, and the system designed for the reserve area if reserve site characteristics differ significantly from the initial area;

(ii) Vertical cross-section drawings showing:

(A) The depth of the soil dispersal component, the vertical separation, and depth of cover material; and

(B) Other new OSS components constructed at the site.

(iii) Calculations and assumptions supporting the proposed design, including:

(A) System operating capacity and design flow;

(B) Soil type; ((~~and~~))

(C) Hydraulic loading rate in the soil dispersal component; and

(e) Any additional information as deemed necessary by the local health officer.

((~~(2) A permit is not required for replacement, addition, or modification of broken or malfunctioning building sewers, risers and lids, sewage tank lids, sewage tank baffles, sewage tank pumps, pump control floats, pipes connecting multiple sewage tanks, and OSS inspection boxes and ports where a sewage tank, treatment component, or soil dispersal component does not need to be replaced. The local health officer may require the owner to submit information regarding these activities for recordkeeping purposes.~~))

(3) The local health officer may develop the information required in subsection ((~~(1)~~)) (2) of this section if authorized by local ((~~regulations~~)) rules.

(4) The local health officer shall:

(a) Respond to an application within ((~~thirty~~)) 30 days as required in RCW 70.05.074((~~.~~));

(b) Permit only public domain treatment technologies that ((~~have departmental RS&G.~~)) are described in this chapter or in a current DS&G;

(c) Permit only proprietary products that are registered by the department((~~. During the period of transition from the list of approved systems and products to the registered list, the local health officer may permit products on the list of approved systems and products.~~

~~(c)~~));

(d) Issue a permit when the information submitted under subsection ((~~(1)~~)) (2) of this section meets the requirements contained in this chapter and in local ((~~regulations~~)) rules;

((~~(d)~~)) (e) Identify the permit as a new installation, repair, expansion, modification, or operational permit;

((~~(e)~~)) (f) Specify the expiration date on the permit. The expiration date may not exceed five years from the date of permit issuance;

((~~(f)~~)) (g) Include a reminder on the permit application of the applicant's right of appeal; and

((~~(g)~~)) (h) If requiring an operational permit, state the period of validity and the date and conditions of renewal including any required field compliance.

(5) The local health officer may revoke or deny a permit for just cause. Examples include, but are not limited to:

(a) Construction or continued use of an OSS that threatens ((~~the~~)) public health;

(b) Misrepresentation or concealment of material fact in information submitted to the local health officer; or

(c) ((~~Failure to meet~~)) Noncompliance with the conditions of the permit, this chapter or any local ((~~regulations~~)) rules.

(6) ((~~Before the local health officer issues a permit for the installation of an OSS to serve more than one development, the applicant shall show:~~

~~(a) An approved public entity owning or managing the OSS in perpetuity; or~~

~~(b) A management arrangement acceptable to the local health officer, recorded in covenant, lasting until the on-site system is no longer needed, and containing, but not limited to:~~

~~(i) A recorded easement allowing access for construction, operation, monitoring maintenance, and repair of the OSS; and~~

~~(ii) Identification of an adequate financing mechanism to assure the funding of operation, maintenance, and repair of the OSS.~~)) An applicant for a permit to install an OSS serving more than one development must submit an application that proves the OSS:

(a) Is owned or managed in perpetuity by a public entity;

(b) Is described in a separate writing including, but not limited to, an easement, covenant, contract, or other legal document authorizing access for construction, operation maintenance, and repair; and

(c) If owned privately, is adequately financed.

(7) The local health officer shall not delegate the authority to issue permits.

(8) The local health officer may stipulate additional requirements for a particular permit if necessary ((~~for~~)) to protect public health ((~~protection~~)).

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0210 Location. (1) ((~~Persons shall design and install~~)) OSS must be designed and installed to meet at least the minimum horizontal separations shown in Table IV((~~, Minimum Horizontal Separations~~)):

Table IV
Minimum Horizontal Separations

Items Requiring Setback	From edge of soil dispersal component and reserve area	From sewage tank and distribution box	From building sewer, and nonperforated distribution pipe
Well ((~~or suction line~~))	100 ft.	50 ft.	50 ft.
Public drinking water well	100 ft.	100 ft.	100 ft.
Nonpublic drinking water well	100 ft.	50 ft.	50 ft.
Public drinking water spring or surface water measured from the ordinary high-water mark	200 ft.	200 ft.	100 ft.
Nonpublic drinking water spring or surface water ((~~used as drinking water source~~)) measured from the ordinary high-water mark[1]	100 ft.	50 ft.	50 ft.
Nonpublic, in-ground, drinking water containment vessel[3]	20 ft.	10 ft.	10 ft.
Pressurized water supply line or easement for water supply line	10 ft.	10 ft.	10 ft.
Closed geothermal loop[4] or pressurized nonpotable water line	10 ft.	10 ft.	10 ft.
Decommissioned well (decommissioned in accordance with chapter 173-160 WAC)	10 ft.	N/A	N/A
Surface water measured from the ordinary high-water mark	100 ft.	50 ft.	10 ft.
Building foundation/in-ground swimming pool	10 ft.	5 ft.	2 ft.
Property or easement line	5 ft.	5 ft.	N/A
Lined[5] stormwater detention pond[6]			
Down-gradient[7]:	30 ft.	N/A	N/A
Up-gradient[7]:	10 ft.	N/A	N/A
Unlined[8] stormwater infiltration pond[6] (up or down-gradient)[7]	100 ft.	50 ft.	10 ft.
Irrigation canal or irrigation pond (up or down-gradient)	100 ft.	50 ft.	10 ft.
Interceptor/curtain drains/foundation drains/ drainage ditches			
Down-gradient[2]:	30 ft.	5 ft.	N/A
Up-gradient[2]:	10 ft.	N/A	N/A
Subsurface stormwater infiltration or dispersion component[6]			
Down-gradient[7]:	30 ft.	10 ft.	N/A
Up-gradient[7]:	30 ft.	10 ft.	N/A
Other site features that may allow effluent to surface			
Down-gradient[2]:	30 ft.	5 ft.	N/A
Up-gradient[2]:	10 ft.	N/A	N/A
Down-gradient cuts or banks with at least 5 ft. of original, undisturbed soil above a restrictive layer due to a structural or textural change	25 ft.	N/A	N/A
Down-gradient cuts or banks with less than 5 ft. of original, undisturbed soil above a restrictive layer due to a structural or textural change	50 ft.	N/A	N/A

Items Requiring Setback	From edge of soil dispersal component and reserve area	From sewage tank and distribution box	From building sewer, and nonperforated distribution pipe
((~~Other adjacent~~)) Soil dispersal components((~~/ subsurface stormwater infiltration systems~~)) serving a separate OSS	10 ft.	N/A	N/A

[1] If surface water is used as a public drinking water supply, the designer shall locate the OSS outside of the required source water protection area.
[2] The item is down-gradient when liquid will flow toward it upon encountering a water table or a restrictive layer. The item is up-gradient when liquid will flow away from it upon encountering a water table or restrictive layer.
[3] Any in-ground containment vessel used to store drinking water.
[4] A network of underground piping carrying fluid under pressure used to heat and cool a structure.
[5] Lined means any component that has the intended function of detaining the stormwater with no intention of dispersal into surrounding soil.
[6] OSS components take precedence in cases of horizontal setback conflicts between OSS and stormwater components.
[7] Down-gradient means that subsurface water flows toward and is usually located lower in elevation. Up-gradient means subsurface water does not flow toward and generally flat, or flows away from and generally located higher in elevation.
[8] Unlined means any component that has the ability to or intended function of infiltrating the stormwater.

(2) ((~~If any condition indicates~~)) When conditions indicate a greater potential for contamination or pollution, the local health officer may increase the minimum horizontal separations. Examples of such conditions include, but are not limited to, excessively permeable soils, unconfined aquifers, shallow or saturated soils, dug wells, and improperly abandoned wells.

(3) The local health officer may allow a reduced horizontal separation to not less than two feet from where the property line, easement line, ((~~in-ground swimming pool,~~)) or building foundation is up-gradient.

(4) The local health officer may require an applicant to demonstrate the OSS meets (a), (b), or (c) of this subsection when determining if a horizontal separation to a minimum of 75 feet between an OSS dispersal component and ((~~an individual~~)) a water well, ((~~individual~~)) spring, or surface water that is not a public water source ((~~can be reduced to a minimum of seventy-five feet, by the local health officer, and be described as a conforming system upon signed approval by the health officer if the applicant demonstrates~~)) is allowed:

(a) Adequate protective site-specific conditions, such as physical settings with low ((~~hydro-geologic~~)) hydrogeologic susceptibility from contaminant infiltration. Examples of such conditions include evidence of confining layers ((~~and/or aquatards separating~~)), an aquatard that separates potable water from the OSS treatment zone, excessive depth to groundwater, down-gradient contaminant source, or outside the zone of influence; or

(b) Design and proper operation of an OSS ((~~system assuring~~)) with enhanced treatment performance beyond that accomplished by meeting the vertical separation and effluent distribution requirements described in Table VI in WAC 246-272A-0230 ((~~Table VI~~)); or

(c) Evidence ((~~of protective conditions involving both~~)) the OSS satisfies the requirements of (a) and (b) of this subsection.

(5) Persons shall design ((~~and/~~))or install a soil dispersal component only if:

(a) The slope is less than ((~~forty-five~~)) 45 percent ((~~(twenty-four~~)) or 24 degrees((~~)~~));

(b) The area is not subject to:

(i) Encroachment by buildings or construction such as placement of power poles and underground utilities;

(ii) Cover by impervious material;

(iii) Vehicular traffic; or

(iv) Other activities adversely affecting the soil or the performance of the OSS.

(c) Sufficient reserve area for replacement exists to treat and dispose one hundred percent of the design flow;

(d) The land is stable; and

(e) Surface drainage is directed away from the site.

(6) The local health officer may approve a sewer transport line within ten feet of a water supply line if the sewer line is constructed in accordance with section ((~~C1-9~~)) C1-9.1 of the department of ecology's "*Criteria For Sewage Works Design*," ((~~December 1998~~)) 2008.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0220 Soil and site evaluation. (1) Only professional engineers, designers, or local health officers may perform soil and site evaluations. Soil scientists may only perform soil evaluations.

(2) The person evaluating the soil and site shall:

(a) Report:

(i) A sufficient number of soil logs to evaluate conditions within:

(A) The initial soil dispersal component; and

(B) The reserve area.

(ii) The groundwater conditions, the date of the observation, and the probable maximum height;

(iii) The topography of the proposed initial ((~~system~~)) OSS, the reserve area, and those areas immediately adjacent that contain characteristics impacting the design;

(iv) The drainage characteristics of the proposed initial ((~~system~~)) OSS, the reserve area and those areas immediately adjacent that contain characteristics impacting the design;

(v) The existence of structurally deficient soils subject to major wind or water erosion events such as slide zones and dunes;

(vi) The existence of designated flood plains ((~~and~~));

(vii) Other areas identified in the local management plan required in WAC 246-272A-0015; and

((~~(vii)~~)) (viii) The location of existing features affecting ((~~system~~)) OSS placement, such as, but not limited to:

(A) Wells ((~~and suction lines~~));

(B) Water sources and supply lines;

(C) Surface water and stormwater infiltration areas;

(D) Abandoned wells;

(E) Outcrops of bedrock and restrictive layers;

(F) Buildings;

(G) Property lines and lines of easement;

(H) Interceptors such as footing drains, curtain drains, and drainage ditches;

(I) Cuts, banks, and fills;

(J) Driveways and parking areas;

(K) Existing OSS; and

(L) Underground utilities;

(b) Use the soil and site evaluation procedures and terminology in accordance with Chapter 5 of the *On-site Wastewater Treatment Systems Manual*, EPA 625/R-00/008, February 2002 except where modified by,

or in conflict with, this chapter ((~~(available upon request to the department)~~));

(c) Use the soil names and particle size limits of the United States Department of Agriculture Natural Resources Conservation Service classification system;

(d) Determine texture, structure, compaction, and other soil characteristics that affect the treatment and water movement potential of the soil by using normal field ((~~and/~~))or laboratory procedures such as particle size analysis; and

(e) Classify the soil as in Table V((~~, Soil Type Descriptions~~)):

((~~TABLE V~~)) **Table V**
Soil Type Descriptions

Soil Type	Soil Textural Classifications
1	Gravelly and very gravelly coarse sands, all extremely gravelly soils excluding those with soil types 5 and 6 as the nongravel portion, and all soil types with greater than or equal to 90% rock fragments.
2	Coarse sands.
3	Medium sands, loamy coarse sands, loamy medium sands.
4	Fine sands, loamy fine sands, sandy loams, loams.
5	Very fine sands, loamy very fine sands; or silt loams, sandy clay loams, clay loams and silty clay loams with a moderate or strong structure (excluding platy structure).
6	Other silt loams, sandy clay loams, clay loams, silty clay loams.
7 Unsuitable for treatment or dispersal	Sandy clay, clay, silty clay, strongly cemented or firm soils, soil with a moderate or strong platy structure, any soil with a massive structure, any soil with appreciable amounts of expanding clays.

(3) The owner of the property or ((~~his~~)) the owner's agent shall:

(a) Prepare the soil log excavation to:

(i) Allow examination of the soil profile in its original position by:

(A) Excavating pits of sufficient dimensions to enable observation of soil characteristics by visual and tactile means to a depth three feet deeper than the anticipated infiltrative surface at the bottom of the soil dispersal component; or

(B) Stopping at a shallower depth if a water table or restrictive layer is encountered;

(ii) Allow determination of the soil's texture, structure, color, bulk density or compaction, water absorption capabilities or permeability, and elevation of the highest seasonal water table; and

(b) Assume responsibility for constructing and maintaining the soil log excavation in a manner to prevent injury as required by chapter 296-155 WAC.

(4) The local health officer:

(a) Shall render a decision on the height of the water table within ((~~twelve~~)) 12 months of receiving the application under precipitation conditions typical for the region;

(b) May require water table measurements to be recorded during months of probable high-water table conditions, if insufficient information is available to determine the highest seasonal water table;

(c) May require any other soil and site information affecting location, design, or installation; ((~~and~~))

(d) May reduce the required number of soil logs for OSS serving a single-family residence if adequate soils information has previously been developed; and

(e) May require another site and soil evaluation if the site has been altered since the initial site and soil evaluation was submitted to the local health officer.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0230 Design requirements—General. (1) ((~~On-site sewage systems may~~)) OSS must only be designed by a professional engineer((~~s~~)), licensed under chapter 18.43 RCW, or ((~~on-site sewage treatment system~~)) an OSS designer((~~s~~)), licensed under chapter 18.210 RCW, except:

(a) If at the discretion of the local health officer, a resident owner of a single-family residence not ((~~adjacent to~~)) within 200 feet of a marine shoreline is allowed to design ((~~a system~~)) an OSS for that residence; or

(b) If the local health officer performs the soil and site evaluation, the health officer ((~~is allowed to~~)) may design ((~~a system~~)) the OSS.

(2) The designer shall use the following criteria when developing a design for an OSS:

(a) All sewage from the building served is directed to the OSS;

(b) Sewage tanks ((~~have been reviewed and approved by the department~~)) are in compliance with chapter 246-272C WAC;

(c) Drainage from the surface, footing drains, roof drains, subsurface stormwater infiltration systems, and other nonsewage drains is prevented from entering the OSS, the area where the OSS is located, and the reserve area;

(d) The OSS is designed to treat and disperse the sewage volume as follows:

(i) For single-family residences:

(A) The operating capacity is based on 45 gpd per capita with two people per bedroom((~~.~~));

(B) The minimum design flow per bedroom per day is the operating capacity of ((~~ninety~~)) 90 gallons multiplied by 1.33 to account for a 33 percent surge capacity. This results in a minimum design flow of ((~~one hundred twenty~~)) 120 gallons per bedroom per day((~~.~~));

(C) ((~~A factor greater than 0.33 to account for surge capacity may be required by~~)) The local health officer((~~.~~)) may require a factor greater than 33 percent to account for surge capacity;

(D) The minimum design flow of the OSS is 240 gpd; and

(E) The local health officer may require an increase of the design flow for dwellings with anticipated greater flows, such as larger dwellings((~~.~~

~~(E) The minimum design flow is two hundred forty gallons per day.~~));

(ii) For single-family residences with one additional dwelling served by the same OSS:

(A) All requirements in (d)(i) of this subsection apply;

(B) The minimum design flow for one additional dwelling is 120 gallons per bedroom; and

(C) The local health officer may require an increase of the design flow for dwellings with anticipated greater flows; or

(iii) For three or more dwellings served by the same OSS:

(A) All requirements in (d)(i) of this subsection apply;

(B) The minimum design flow for the first dwelling is 240 gallons per day;

(C) The minimum design flow for each additional dwelling is 120 gallons per bedroom;

(D) The local health officer may require an increase of the design flow for dwellings with anticipated greater flows; and

(E) The local health officer shall require documentation including, but not limited to, an easement, covenant, contract, or other legal document authorizing access for construction, operation, maintenance, and repair; or

(iv) For other facilities, the design flows noted in "*On-site Wastewater Treatment Systems Manual*," USEPA, EPA-625/R-00/008, February 2002 ((~~(available upon request to the department) shall~~)) must be used. Sewage flows from other sources of information may be used in determining system design flows if they incorporate both an operating capacity and a surge capacity((~~.~~));

(e) The OSS is designed to address sewage quality as follows:

(i) For all systems, the designer shall consider:

(A) $CBOD_5$, TSS, and O&G;

(B) Other parameters that can adversely affect treatment anywhere along the treatment component sequence. Examples include pH, temperature, and dissolved oxygen;

(C) The sensitivity of the site where the OSS will be installed. Examples include areas where fecal coliform constituents can result in public health concerns, such as shellfish growing areas, designated swimming areas, and other areas identified by the local management plan required in WAC 246-272A-0015((~~.~~)); and

(D) Nitrogen contributions. Where nitrogen has been identified as a contaminant of concern by the local management plan required in WAC 246-272A-0015, it ((~~shall~~)) must be addressed through lot size ((~~and/or~~)), treatment, or both.

(ii) For OSS treating sewage from a nonresidential source, the designer shall provide the following information showing:

(A) ((~~Information to show~~)) The sewage is not industrial wastewater;

(B) ((~~Information regarding~~)) The sewage effluent quality and identifying chemicals found in the sewage ((~~that~~)) effluent are not found in sewage effluent from a residential source; and

(C) A site-specific design providing the necessary treatment ((~~level equal to that required of~~)) equaling required treatment of sewage effluent quality from a residential source;

(f) The vertical separation ((~~to be~~)) used to establish the treatment levels and application rates. The selected vertical separation ((~~shall~~)) must be used consistently throughout the design process((~~.~~)); and

(g) Treatment levels:

(i) Requirements for matching treatment component and method of distribution with soil conditions of the soil dispersal component are listed in Table VI of this section. The treatment levels correspond with those established for treatment components under the product performance testing requirements in Table III of WAC 246-272A-0110. The method of distribution applies to the soil dispersal component.

(ii) Disinfection may not be used ((~~to achieve the fecal coliform requirements to meet:~~

~~(A) Treatment levels A or B in Type 1 soils; or~~

~~(B) Treatment level C~~)):

(A) To achieve BL1 or BL2 in type 1 soils; or

(B) BL3.

((~~TABLE VI~~)) Table VI
Treatment Component Performance Levels and Method of Distribution[1]

Vertical Separation in inches	Soil Type		
	1	2	3-6
12 < 18	A & BL1 - pressure with timed dosing	B & BL2 - pressure with timed dosing	B & BL2 - pressure with timed dosing
≥18 < 24	B & BL2 - pressure with timed dosing	((~~B~~)) C & BL3 - pressure with timed dosing	((~~B~~)) C & BL3 - pressure with timed dosing
≥24 < 36	B & BL2 - pressure with timed dosing	C & BL3 - pressure with timed dosing	E - pressure with timed dosing
≥36 < 60	B & BL2 - pressure with timed dosing	E - pressure	E - gravity
≥60	C & BL2 - pressure	E - gravity	E - gravity

[1] The treatment component performance levels correspond with those established for treatment components under the product testing requirements in WAC 246-272A-0110.

(3) The coarsest textured soil within the vertical separation selected by the designer ((~~shall~~)) determines the minimum treatment level and method of distribution.

(4) The local health officer shall not approve designs for:

(a) Cesspools; or

(b) Seepage pits.

(5) The local health officer may approve a design for the reserve area different from the design approved for the initial OSS, if both designs meet the requirements of this chapter for new construction.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0232 Design requirements—Septic tank sizing. Septic tanks ((~~shall~~)) must:

(1) Have at least two compartments with the first compartment liquid volume equal to one-half to two-thirds of the total liquid volume. This standard may be met by one tank with two compartments or by two single compartment tanks in series.

(2) Have the following minimum liquid volumes:

(a) For a single-family residence use Table VII((~~, Required Minimum Liquid Volumes of Septic Tanks~~)):

((~~TABLE VII~~)) **Table VII**
Required Minimum Liquid Volumes of Septic Tanks

Number of Bedrooms	Required Minimum Liquid Tank Volume in Gallons
((~~≤3~~	~~900~~
4	~~1000~~))
≤4	1,000
Each additional bedroom	250

(b) For OSS treating sewage from a residential source, other than one single-family residence, ((~~two hundred fifty~~)) 250 gallons per bedroom with a minimum of ((~~one thousand~~)) 1,000 gallons;

(c) For OSS treating sewage from a nonresidential source, three times the design flow.

(3) Comply with chapter 246-272C WAC.

NEW SECTION

WAC 246-272A-0233 Design requirements—Pump chambers. (1) All pump chambers, except pump basins, must be designed to meet the following requirements:

(a) Have a minimum volume of 1,000 gallons;

(b) Provide an internal volume to account for the design flow, full-time pump submergence, space for sludge accumulation below the pump inlet and emergency storage volume of at least 75 percent of the design flow;

(c) Follow any applicable DS&G or proprietary product design manual for all OSS components included in the pump chamber; and

(d) Comply with chapter 246-272C WAC.

(2) For the purposes of this section, "pump basin" means a watertight receptacle that contains a pump to convey sewage from a limited use area that is separate from the main wastewater sewer pipe leaving a structure, to the main treatment component of an OSS; typically much smaller than a pump chamber and separate from the main sewer pipe due to elevation restrictions. Pump basins are intended for limited, specialized uses, and not intended as a replacement or substitute for a pump chamber. Pump basins must be in compliance with chapter 246-272C WAC.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0234 Design requirements—Soil dispersal components. (1) All soil dispersal components, except one using a subsurface dripline product, ((~~shall~~)) must be designed to meet the following requirements:

(a) Maximum hydraulic loading rates ((~~shall be based on the rates~~)) described in Table VIII((~~:~~)).

((~~TABLE VIII~~))

Table VIII

Maximum Hydraulic Loading Rate

		Column A	Column B
Soil Type	**Soil Textural Classification Description**	**Loading Rate for Residential Septic Tank Effluent Using Gravity or Pressure Distribution gal./sq. ft./day**	**Loading Rate for Residential Effluent Meeting Treatment Level C & BL3 or Higher Effluent Quality Using Pressure Distribution gal./sq. ft./day**
1	Gravelly and very gravelly coarse sands, all extremely gravelly soils excluding those with soil types 5 & 6 as the nongravel portion, all soil types with greater than or equal to 90% rock fragments.	1.0	1.2
2	Coarse sands.	1.0	1.2
3	Medium sands, loamy coarse sands, loamy medium sands.	0.8	1.0
4	Fine sands, loamy fine sands, sandy loams, loams.	0.6	0.8
5	Very fine sands, loamy very fine sands; or silt loams, sandy clay loams, clay loams and silty clay loams with a moderate structure or strong structure (excluding a platy structure).	0.4	0.56
6	Other silt loams, sandy clay loams, clay loams, silty clay loams.	0.2	0.2
7	Sandy clay, clay, silty clay and strongly cemented firm soils, soil with a moderate or strong platy structure, any soil with a massive structure, any soil with appreciable amounts of expanding clays.	((~~Not suitable~~)) Unsuitable	Unsuitable

(b) Calculation of the absorption area is based on:

(i) The design flow in WAC 246-272A-0230(2); and

(ii) Loading rates equal to or less than those in Table VIII of this section as applied to the infiltrative surface of the soil dispersal component or the finest textured soil within the vertical separation selected by the designer, whichever has the finest texture.

(c) Requirements for the method of distribution ((~~shall~~)) must correspond to those in WAC 246-272A-0230, Table VI.

(d) Soil dispersal components having daily design flow between ((~~one thousand and three thousand five hundred~~)) 1,000 and 3,500 gallons of sewage per day ((~~shall~~)) must:

(i) Only be located in soil types 1-5;

(ii) Only be located on slopes of less than ((~~thirty~~)) 30 percent, or ((~~seventeen~~)) 17 degrees; and

(iii) Have pressure distribution including time dosing.

(2) The local health officer may allow the maximum hydraulic loading rates in Table VIII of this section. Loading rates identified in Column B must not be combined with any dispersal component size reductions.

(3) All soil dispersal components using a subsurface dripline product must be designed to meet the following requirements:

(a) ((~~Calculation of~~)) The absorption area calculation is based on:

(i) The design flow in WAC 246-272A-0230(2); and

(ii) Loading rates ((~~that are~~)) dependent on the soil type, other soil and site characteristics, and the spacing of dripline and emitters as directed in Table VIII of this section;

(b) ((~~The dripline must be installed~~)) A minimum installation of six inches into original, undisturbed soil;

(c) Timed dosing; and

(d) ((~~Soil dispersal components having~~)) Daily design flows greater than ((~~one thousand~~)) 1,000 gallons of sewage per day ((~~may~~)):

(i) ((~~Only be~~)) Located only in soil types 1-5;

(ii) ((~~Only be~~)) Located only on slopes of less than ((~~thirty~~)) 30 percent, or ((~~seventeen~~)) 17 degrees.

((~~(3)~~)) (4) All SSAS ((~~shall~~)) must meet the following requirements:

(a) The infiltrative surface may not be deeper than three feet below the finished grade, except under special conditions approved by the local health officer. The depth of such system ((~~shall~~)) must not exceed ((~~ten~~)) 10 feet from the finished grade;

(b) A minimum of six inches of sidewall must be located in ((~~original undisturbed~~)) suitable soil;

(c) Beds are only designed in soil types 1, 2, 3 or in fine sands with a width not exceeding ((~~ten~~)) 10 feet. Gravity beds must have a minimum of one lateral for every three feet in width;

(d) Individual laterals greater than ((~~one hundred~~)) 100 feet in length must use pressure distribution;

(e) A layer of between six and ((~~twenty-four~~)) 24 inches of cover material; and

(f) Other features ((~~shall~~)) must conform with the "*On-site Wastewater Treatment Systems Manual*," United States Environmental Protection Agency EPA-625/R-00/008 February 2002 ((~~(available upon request to the department)~~)) except where modified by, or in conflict with this section or local ((~~regulations~~)) rules.

((~~(4) For~~)) (5) SSAS with drainrock and distribution pipe must meet the following requirements:

(a) A minimum of two inches of drainrock ((~~is required~~)) above the distribution pipe;

(b) A minimum of six inches of drainrock below the distribution pipe; and

(c) Location of the sidewall below the invert of the distribution pipe ((~~is located~~)) in original undisturbed soil.

((~~(5)~~)) (6) The local health officer may allow the infiltrative surface area in a SSAS to include six inches of the SSAS sidewall height when meeting the required absorption area where total recharge by annual precipitation and irrigation is less than ((~~twelve~~)) 12 inches per year.

((~~(6)~~)) (7) The local health officer may permit ((~~systems~~)) OSS consisting ((~~solely~~)) of ((~~a~~)) septic tanks and a gravity SSAS in soil type 1 if all the following criteria are met:

(a) The ((~~system~~)) OSS serves a single-family residence;

(b) The lot size is ((~~greater than~~)) two and one-half acres or larger;

(c) Annual precipitation in the region is less than ((~~twenty-five~~)) 25 inches per year ((~~as described by "*Washington Climate*" published jointly by the Cooperative Extension Service, College of Agriculture, and Washington State University (available for inspection at Washington state libraries)~~)) from a reputable source approved by the local health officer;

(d) The ((~~system~~)) OSS is located outside the ((~~twelve~~)) 12 counties bordering Puget Sound; and

(e) The geologic conditions beneath the dispersal component must satisfy the minimum unsaturated depth requirements to groundwater as determined by the local health officer. The method for determination is described by "*Design Guideline for Gravity Systems in Soil Type 1*," ((~~(available upon request to the department)~~)) 2017.

((~~(7) The local health officer may increase the loading rate in Table VIII up to a factor of two for soil types 1-4 and up to a factor of 1.5 for soil types 5 and 6 if a product tested to meet treatment level D is used. This reduction may not be combined with any other SSAS size reductions.~~

~~(8)(a)~~)) (8) Both the primary and reserve areas must be sized ((~~to~~)) at least ((~~one hundred~~)) 100 percent of the approved loading rates ((~~listed in Table VIII.~~

~~(b) However, the local health officer may allow a legal lot of record created prior to the effective date of this chapter that cannot meet this primary and reserve area requirement to be developed if all the following conditions are met:~~

~~(i) The lot cannot meet the minimum primary and reserve area requirements due to the loading rates for medium sand, fine sand and very fine sand listed in Table VIII of this chapter;~~

~~(ii) The primary and reserve areas are sufficient to allow installation of a SSAS using maximum loading rates of 1.0 gallons/square foot per day for medium sand, 0.8 gallons/square foot/day for fine sand, and 0.6 gallons/square foot/day for very fine sand; and~~

~~(iii) A treatment product meeting at least Treatment Level D and pressure distribution with timed-dosing is used~~)). The local health officer may require the sizing of the reserve area using the loading rate in Table VIII of this section. Column A must be used when sizing the primary area using Column B.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0238 Design requirements—Facilitate operation, monitoring and maintenance. (1) The OSS must be designed to facilitate routine operation, monitoring, and maintenance according to the following criteria:

(a) For gravity ((~~systems, septic~~)) OSS:

(i) Sewage tank access for maintenance and inspection at finished grade is required. ((~~If effluent filters are used, access to the filter at finished grade is required.~~)) The local health officer may allow access for maintenance and inspection of a ((~~system consisting of a septic~~)) sewage tank ((~~and gravity flow SSAS~~)) to be a maximum of six inches below finished grade provided a marker showing the location of the tank access is installed at finished grade.

(ii) Each SSAS lateral must include at least one observation port installed in a representative location in order to facilitate SSAS monitoring.

(b) For all other ((~~systems~~)) OSS, service access and monitoring ports at finished grade are required for all system components. Specific component requirements include:

(i) Septic tanks must have service access maintenance holes (formerly manholes) and monitoring ports for the inlet and outlet((~~. If effluent filters are used, access to the filter at finished grade is required~~));

(ii) Surge, flow equalization or other sewage tanks must have service access ((~~manholes~~)) maintenance holes;

(iii) Other pretreatment units ((~~(~~))such as aerobic treatment units and packed-bed filters((~~)~~)) must have service access ((~~manholes~~)) maintenance holes and monitoring ports;

(iv) Pump chambers, tanks, and vaults must have service access ((~~manholes~~)) maintenance holes;

(v) Disinfection units must have service access and be installed to facilitate complete maintenance and cleaning, including an easy-access, freefall sampling port; and

(vi) Soil dispersal components ((~~shall~~)), excluding subsurface drip, must have monitoring ports for both distribution devices and the infiltrative surface.

(c) For systems using pumps, clearly accessible controls and warning devices are required including:

(i) Process controls such as floats ((~~and~~)), pressure activated pump on/off switches, and pump-run timers ((~~and process flow controls~~));

(ii) Diagnostic tools including dose cycle counters and hour meters on the sewage stream, or flow meters on either the water supply or sewage stream; and

(iii) Audible and visual alarms designed to alert a resident of a malfunction. The alarm must be placed on a circuit independent of the pump circuit.

(2) All accesses must be designed to allow for monitoring and maintenance and shall be secured to minimize injury or unauthorized access in a manner approved by the local health officer.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0240 Holding tank sewage systems. (1) A person may not install or use holding tank sewage systems for residential development or expansion of residences, whether seasonal or year-round, except as set forth under subsection (2) of this section.

(2) The local health officer may approve installation of holding tank sewage systems only:

(a) For permanent uses limited to controlled, part-time, commercial usage situations, such as recreational vehicle parks and trailer dump stations;

(b) For interim uses limited to handling of emergency situations; or

(c) For repairs as permitted under WAC 246-272A-0280 (1)((~~(c)~~)) (d)(i).

(3) A person proposing to use a holding tank sewage system shall:

(a) Follow design criteria established by the department;

(b) Submit a management program to the local health officer assuring ongoing operation, monitoring, and maintenance before the local health officer issues the installation permit; and

(c) Use a holding tank reviewed and approved by the department.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0250 Installation. (1) Only installers may construct OSS, except as noted under subsection (2) of this section.

(2) The local health officer may allow the resident owner of a single-family residence ((~~not adjacent to a marine shoreline~~)) to install the OSS for that single-family residence except when:

(a) The primary and reserve areas are within 200 feet of marine water;

(b) The primary and reserve areas are within 100 feet of surface water; or

(c) The installation permit meets Table X standards in WAC 246-272A-0280.

(3) The installer described by either subsection (1) or (2) of this section shall:

(a) Follow the approved design;

(b) Have the approved design in possession during installation;

(c) Make no changes to the approved design without the prior authorization of the designer and the local health officer;

(d) Only install ((~~septic tanks, pump chambers, and holding~~)) sewage tanks approved by the department consistent with chapter 246-272C WAC;

(e) Be on the site at all times during the excavation and construction of the OSS;

(f) Install the OSS to be watertight, except for the soil dispersal component;

(g) Cover the installation only after the local health officer has given approval to cover; and

(h) Back fill with six to ((~~twenty-four~~)) 24 inches of cover material and grade the site to prevent surface water from accumulating over any component of the OSS.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0260 Inspection. (1) For all activities requiring a permit, the local health officer shall inspect the OSS. The local health officer shall:

(a) Visit the OSS site during the site evaluation, construction, or final construction inspection;

(b) Either inspect the OSS before cover or allow the designer of the OSS to perform the inspection before cover if the designer is not also named as installer of the system((.)); and

(c) Keep the record drawings on file, with the approved design documents.

(2) Prior to any inspection, the local health officer or inspector authorized by the local health officer shall coordinate with the OSS owner to obtain access. When the owner does not authorize access, the local health officer may follow the administrative search warrant procedures in RCW 70A.105.030 to gain access.

(3) For any OSS located on a single property serving one dwelling unit on the same property, the local health officer shall not require a property owner to grant inspection and maintenance easements as a condition of receiving a permit.

(4) During the final construction inspection, the local health officer or the designer of the OSS must confirm the OSS meets the approved design.

(5) To comply with the requirements of WAC 246-272A-0270 (1)(e) or (k), an inspection must include, at a minimum:

(a) Inspection and evaluation of:

(i) The status of all sewage tanks including baffles, effluent filters, tank contents such as water level, scum, sludge, solids, water tightness, and general structural conditions;

(ii) The status of all lids, accesses, and risers;

(iii) The OSS and reserve area for any indicators of OSS failure or conditions that may impact system function, operation, or repair; and

(iv) Any other components such as distribution boxes;

(b) A review of the record drawing and related documents, if they exist, including previous reports to confirm the system is operating as designed; and

(c) Any proprietary products following the procedures of the accepted operations and maintenance manual associated with those products.

(6) Evidence of an OSS property transfer inspection as required in WAC 246-272A-0270 (1)(k) must be provided to the local health jurisdiction on a form approved by the local health officer, including at a minimum:

(a) All applicable information from subsection (5) of this section;

(b) The address of the property served by the OSS;

(c) The date of the inspection;

(d) The permitted type and design flow for known OSS; and

(e) Verification that the record drawing is accurate, if it exists, or an OSS site plan showing the location of all system components relative to structures and prominent site features.

(7) A local health jurisdiction may require an additional inspection report, or additional information, for an inspection required un-

der WAC 246-272A-0270(1). The person responsible for the final construction inspection shall assure the OSS meets the approved design.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0265 Record drawings. Upon completion of ((~~the~~)) new construction, alteration, or repair of the OSS, the OSS owner shall submit a complete and detailed record drawing ((~~shall be submitted to both~~)) to the local health officer ((~~and the OSS owner~~)) that includes at a minimum ((~~the following~~)):

(1) Measurements and directions accurate to +/- 1/2 foot, unless otherwise determined by the local health officer, ((~~to assure~~)) so that the following parts of the OSS can be easily located:

(a) All sewage tank openings requiring access;

(b) The ends, and all changes in direction, of installed and found buried pipes and electrical cables that are part of the OSS; and

(c) Any other OSS component which, in the judgment of the local health officer or the designer, must be accessed for observation, maintenance, or operation;

(2) Location and dimensions of the reserve area;

(3) Record that materials and equipment meet the specifications contained in the design;

(4) Initial settings of electrical or mechanical devices that must be known to operate the system in the manner intended by the designer or installer; and

(5) For proprietary products, manufacturer's standard product literature, including performance specifications and maintenance recommendations needed for operation, monitoring, maintenance, or repair of the OSS.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0270 Operation, monitoring, and maintenance—Owner responsibilities. (1) The OSS owner is responsible for operating, monitoring, and maintaining the OSS to minimize the risk of failure, and ((~~to accomplish this purpose,~~)) shall:

(a) Request assistance from the local health officer upon occurrence of a system failure or suspected system failure;

(b) Obtain approval from the local health officer before:

(i) Repairing, altering, or expanding an OSS((~~;~~
~~(b)~~)) as required by WAC 246-272A-0200; or

(ii) Before beginning the use of any newly constructed OSS;

(c) Secure and renew contracts for periodic maintenance ((~~where~~)) if required by the local health jurisdiction;

((~~(c)~~)) (d) Obtain and renew operation permits if required by the local health jurisdiction;

((~~(d) Assure a complete evaluation of the system components and/or~~)) (e) Obtain an inspection, as required in WAC

246-272A-0260(5), by a maintenance service provider authorized by the local health officer of all OSS and property to determine functionality, maintenance needs and compliance with ((~~regulations~~)) this chapter and local rules, and any permits:

(i) At least once every three years, unless more frequent inspections are specified by the local health officer, for all ((~~systems~~)) OSS consisting solely of a ((~~septic~~)) sewage tank and gravity SSAS;

(ii) Annually for all other ((~~systems~~)) OSS unless more frequent inspections are specified by the local health officer;

((~~(e)~~)) (iii) Submit the results of the inspection to the local health jurisdiction, using a form approved by the local health officer and in compliance with WAC 246-272A-0260(5);

(f) Employ an approved pumper to remove the septage from the tank when the level of solids and scum indicates that removal is necessary;

((~~(f)~~)) (g) Provide ongoing maintenance and complete any needed repairs to promptly return the ((~~system~~)) OSS to a proper operating condition;

((~~(g)~~)) (h) Protect the OSS area and the reserve area from:

(i) Cover by structures or impervious material;

(ii) Surface drainage, and direct drains, such as footing or roof drains. The drainage must be directed away from the area where the OSS is located;

(iii) Soil compaction((~~,~~)). For example by vehicular traffic or livestock; and

(iv) Damage by soil removal and grade alteration((~~,~~ ~~(h)~~)).

(i) Keep the flow of sewage to the OSS at or below the approved operating capacity and sewage quality;

((~~(i)~~)) (j) Operate and maintain ((~~systems~~)) OSS as directed by the local health officer((~~,~~

~~(j) Request assistance from the local health officer upon occurrence of a system failure or suspected system failure~~)); and

(k) At the time of property transfer((~~,~~)):

(i) Provide to the buyer, all available OSS maintenance and repair records((~~, if available,~~)) in addition to the completed seller disclosure statement in accordance with chapter 64.06 RCW for residential real property transfers;

(ii) Beginning February 1, 2027, obtain an inspection, as required in WAC 246-272A-0260(5), by a third-party inspector authorized by the local health officer. The local health officer may:

(A) Remove the requirement for an inspection at the time of property transfer if the local health jurisdiction has evidence that the OSS is in compliance with (e) of this subsection and the OSS was inspected by a third-party inspector authorized by the local health officer;

(B) Verify the results of the property inspection for compliance with WAC 246-272A-0260; and

(C) Require additional inspections and other requirements not listed in WAC 246-272A-0260;

(iii) Beginning February 1, 2027, obtain an inspection of proprietary treatment products per the product manufacturer recommendations, as required in WAC 246-272A-0260, by a third-party inspector authorized by the local health officer. The local health officer may:

(A) Remove the requirement for an inspection at the time of property transfer if the local health jurisdiction has evidence that the OSS is in compliance with (e) of this subsection and the OSS was in-

spected by a third-party inspector authorized by the local health officer;

(B) Verify the results of the property inspection for compliance with WAC 246-272A-0260; and

(C) Require additional inspections and other requirements not listed in WAC 246-272A-0260;

(iv) Submit the results of the inspection, and any additional information or reports required by the local health officer, to the local health jurisdiction, using an inspection report form approved by the local health officer. The local health officer may require a compliance schedule for repair of a failure discovered during the property transfer inspection.

(2) ((~~Persons shall~~)) A person may not:

(a) Use or introduce strong bases, acids, or chlorinated organic solvents into an OSS for the purpose of system cleaning;

(b) Use ((~~a sewage system~~)) an OSS additive unless it is specifically approved by the department; ((~~or~~))

(c) Use an OSS to dispose of waste components atypical of sewage from a residential source; or

(d) Use any remediation process or activity unless it is approved by the local health officer and is in compliance with WAC 246-272A-0278.

NEW SECTION

WAC 246-272A-0278 Remediation. (1) The local health officer may establish a program and requirements for reviewing and approving remediation activities.

(2) Remediation must not:

(a) Result in damage to the OSS;

(b) Result in insufficient soil treatment in the zone between the soil dispersal component and the highest seasonal water table, restrictive layer, or soil type 7; or

(c) Disturb the soil in or below the soil dispersal component if the vertical separation requirements of WAC 246-272A-0230 are not met.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0280 Repair of failures. ((~~(1) When an OSS failure occurs, the OSS owner shall:~~

~~(a) Repair or replace the OSS with a conforming system or component, or a system meeting the requirements of Table IX either on the:~~

~~(i) Property served; or~~

~~(ii) Nearby or adjacent property if easements are obtained; or~~

~~(b) Connect the residence or facility to a:~~

~~(i) Publicly owned LOSS;~~

~~(ii) Privately owned LOSS where it is deemed economically feasible; or~~

~~(iii) Public sewer; or~~

(c) Perform one of the following when requirements in (a) and (b) of this subsection are not feasible:

(i) Use a holding tank; or

(ii) Obtain a National Pollution Discharge Elimination System or state discharge permit from the Washington state department of ecology issued to a public entity or jointly to a public entity and the system owner only when the local health officer determines:

(A) An OSS is not feasible; and

(B) The only realistic method of final dispersal of treated effluent is discharge to the surface of the land or into surface water; or

(iii) Abandon the property.

(2) Prior to repairing the soil dispersal component, the OSS owner shall develop and submit information required under WAC 246-272A-0200(1).

(3) The local health officer shall permit a system that meets the requirements of Table IX only if the following are not feasible:

(a) Installation of a conforming system or component; and

(b) Connection to either an approved LOSS or a public sewer.

(4) The person responsible for the design shall locate and design repairs to:

(a) Meet the requirements of Table IX if the effluent treatment and soil dispersal component to be repaired or replaced is closer to any surface water, well, or spring than prescribed by the minimum separation required in Table IV of WAC 246-272A-0210(1). Pressure distribution with timed dosing in the soil dispersal component is required in all cases where a conforming system is not feasible.

TABLE IX

Treatment Component Performance Levels for Repair of OSS Not Meeting Vertical and Horizontal Separations[1]

Vertical Separation (in inches)	Horizontal Separation[2]											
	< 25 feet			25 < 50 feet			50 < 100 feet[3]			≥100 feet		
	Soil Type			Soil Type			Soil Type			Soil Type		
	1	2	3-6	1	2	3-6	1	2	3-6	1	2	3-6
< 12	A	A	A	A	A	A	A	A	B	B	B	B
≥ 12 < 18	A	A	A	A	B	B	A	B	B	Conforming Systems		
≥ 18 < 24	A	A	A	A	B	B	A	B	C			
≥ 24 < 36	A	B	B	B	C	C	B	C	C			
≥ 36	A	B	B	B	C	C	B	C	E			

[1]The treatment component performance levels correspond with those established for treatment components under the product performance testing requirements in Table III of WAC 246-272A-0110.

[2]The horizontal separation indicated in Table IX is the distance between the soil dispersal component and the surface water, well, or spring. If the soil dispersal component is up-gradient of a surface water, well, or spring to be used as a potable water source, or beach where shellfish are harvested, the next higher treatment level shall apply unless treatment level A is already required.

[3]On a site where there is a horizontal setback of 75 - 100 feet between an OSS dispersal component and an individual water well, individual spring, nonmarine surface water or surface water that is not a public water source and a vertical separation of greater than twelve inches, a conforming system that complies with WAC 246-272A-0210(4) shall be installed if feasible.

(b) Protect drinking water sources and shellfish harvesting areas;

(c) Minimize nitrogen discharge in areas where nitrogen has been identified as a contaminant of concern in the local plan under WAC 246-272A-0015;

(d) Prevent the direct discharge of sewage to groundwater, surface water, or upon the surface of the ground;

~~(e) Meet the horizontal separations under WAC 246-272A-0210(1) to public drinking water sources;~~

~~(f) Meet other requirements of this chapter to the maximum extent permitted by the site; and~~

~~(g) Maximize the:~~

~~(i) Vertical separation;~~

~~(ii) Distance from a well, spring, or suction line; and~~

~~(iii) Distance to surface water.~~

~~(5) Prior to designing the repair system, the designer shall consider the contributing factors of the failure to enable the repair to address identified causes.~~

~~(6) If the vertical separation is less than twelve inches, the local health officer may permit ASTM C-33 sand or coarser to be used as fill to prevent direct discharge of treated effluent to groundwater, surface water, or upon the surface of the ground.~~

~~(7) For a repair using the requirements of Table IX, disinfection may not be used to achieve the fecal coliform requirements to meet:~~

~~(a) Treatment levels A or B where there is less than eighteen inches of vertical separation;~~

~~(b) Treatment levels A or B in type 1 soils; or~~

~~(c) Treatment level C.~~

~~(8) The local health officer shall identify repair permits meeting the requirements of Table IX for the purpose of tracking future performance.~~

~~(9) An OSS owner receiving a repair permit for a system meeting the requirements of Table IX from the local health officer shall:~~

~~(a) Immediately report any failure to the local health officer;~~

~~(b) Comply with all local and state requirements stipulated on the permit.~~))

(1) When an OSS failure occurs the local health officer shall:

(a) Allow an OSS to be repaired using the least costly alternative that meets standards and is likely to provide comparable or better long-term sewage treatment and effluent dispersal outcomes;

(b) Permit an OSS meeting the requirements in Table X of this section only if the OSS has failed and the following are not feasible:

(i) Installation of a conforming OSS or component; or

(ii) Connection to either an approved LOSS or a public sewer.

(c) Identify repair permits meeting the requirements in Table X of this section for the purpose of tracking future performance;

(d) Give first priority to allowing repair and second priority to allowing replacement of an existing conventional OSS, consisting of a septic tank and drainfield, with a similar conventional OSS;

(e) Evaluate all unpermitted sewage discharges to determine if they pose a public health threat. If determined by the local health officer to be a public health threat, the local health officer shall require a compliance schedule;

(f) Report failures within 200 feet of shellfish growing areas to the department; and

(g) Not impose or allow the imposition of more stringent performance requirements of equivalent OSS on private entities than public entities.

(2) The local health officer may:

(a) Require a compliance schedule for failures discovered during property transfer inspections;

(b) Allow a repair of a failure using ASTM C-33 sand or coarser as fill to prevent direct discharge of treated effluent to groundwa-

ter, surface water, or upon the surface of the ground if the vertical separation is less than 12 inches.

(3) The OSS owner shall notify the local health officer when there is a failure and indicate which methods will be used to address the failure in accordance with Table IX of this section:

(a) The owner may use option D only if the local health officer determines options A through C are not feasible and may use option E or F only if options A through D are not feasible.

(b) For options A through F, the owner shall develop and submit information and obtain a permit as required under WAC 246-272A-0200 prior to any repair or replacement of an OSS on the property served or a nearby property if the owner obtains an appropriate documentation including, but not limited to, an easement, covenant, contract, or other legal document authorizing access for construction, operation, maintenance, and repair.

(c) If options A through F are not feasible, the owner shall discontinue use of the OSS, abandon the OSS according to the requirements in WAC 246-272A-0300, and cease all sewage generating activities on the property.

Table IX
Options and Methods to Address an OSS Failure

Options	Method
A	Repair or replace the OSS, with a similar OSS, if the OSS provides comparable or better long-term sewage treatment and effluent dispersal outcomes where: 1. The effluent treatment and soil dispersal component to be repaired or replaced is not closer to any surface water, well, or spring than the minimum separation distance required in Table IV of WAC 246-272A-0210(1); 2. The soil dispersal component to be repaired or replaced complies with the treatment level and distribution method requirements in Table VI of WAC 246-272A-0230; 3. The local health officer has a permit or record of the OSS on file; and 4. The repair or replacement will not result in an OSS that meets the definition of failure.
B	Repair or replace the OSS with an OSS in compliance with new construction requirements under this chapter.
C	Connect the residence or facility to a: 1. Publicly owned LOSS; 2. Privately owned LOSS where it is deemed economically feasible; or 3. Public sewer.
D	Repair or replace the OSS in conformance with Table X of this section.
E	Use a holding tank.
F	Obtain a National Pollution Discharge Elimination System or state discharge permit from the Washington state department of ecology issued to a public entity or jointly to a public entity and the OSS owner only when the local health officer determines: 1. An OSS is not feasible; and 2. The only realistic method of final dispersal of treated effluent is discharge to the surface of the land or into surface water.

(4) When there is an OSS failure, the OSS designer shall:

(a) Evaluate the causes of failure prior to designing the repair or replacement of the OSS;

(b) Prevent the direct discharge of sewage or treated effluent to groundwater, surface water, or upon the surface of the ground;

(c) Meet the horizontal separations under WAC 246-272A-0210(1) to public drinking water sources;

(d) Protect all drinking water sources, shellfish harvesting areas, and water recreation facilities designated for swimming in natural waters;

(e) Minimize nitrogen discharge in areas where nitrogen has been identified as a contaminant of concern in the local management plan under WAC 246-272A-0015;

(f) Not use disinfection to achieve fecal coliform or *E. coli* requirements in Table X of this section to meet:

(i) Treatment level BL1 or BL2 with less than 18 inches of vertical separation; or

(ii) Treatment level BL1 or BL2 in type 1 soils; or

(iii) Treatment level BL3.

(g) Minimize impact of phosphorus discharge in areas where the local health officer has identified phosphorus as a contaminant of concern in the local management plan under WAC 246-272A-0015;

(h) Locate and design repairs meeting the requirements in Table X of this section if the effluent treatment and soil dispersal component to be repaired or replaced is closer to any surface water, well, or spring than prescribed by the minimum separation required in Table IV of WAC 246-272A-0210(1);

(i) Design any nonconforming OSS using pressure distribution with timed dosing in the soil dispersal component; and

(j) Meet all other design requirements of this chapter to the maximum extent permitted by the site, to maximize the:

(i) Vertical separation;

(ii) Distance from a well or spring; and

(iii) Distance to surface water.

Table X
Treatment Component Performance Levels for Repair of OSS Not Meeting Vertical and Horizontal Separations[1]

Vertical Separation (in inches)	Horizontal Separation[2]											
	< 30 feet			≥ 30 < 50 feet			≥ 50 < 100 feet[3]			≥ 100 feet		
	Soil Type			Soil Type			Soil Type			Soil Type		
	1	2	3-6	1	2	3-6	1	2	3-6	1	2	3-6
< 12	A & BL1	A & BL1	A & BL1	A & BL1	A & BL1	A & BL1	A & BL1	A & BL1	A & BL1	B & BL2	B & BL2	B & BL2
≥ 12 < 18	A & BL1	A & BL1	A & BL1	A & BL1	B & BL2	B & BL2	A & BL1	B & BL2	B & BL2	Conforming OSS		
≥ 18 < 24	A & BL1	A & BL1	A & BL1	A & BL1	B & BL2	B & BL2	A & BL1	B & BL2	B & BL2			
≥ 24 < 36	A & BL1	B & BL2	B & BL2	B & BL2	B & BL2	B & BL2	B & BL2	B & BL2	C & BL3			
≥ 36	A & BL1	B & BL2	B & BL2	B & BL2	C & BL3	C & BL3	B & BL2	C & BL3	C & BL3			

[1] The treatment component performance levels correspond with those established for treatment components under the product performance testing requirements in Table III in WAC 246-272A-0110.

[2] The horizontal separation indicated in Table X of this section is the distance between the soil dispersal component and the surface water, well, or spring. If the soil dispersal component is up-gradient of a surface water, well, or spring to be used as a potable water source, or beach where shellfish are harvested, the next higher treatment level shall apply unless treatment level A and BL1 is already required.

[3] On a site where there is a horizontal setback of 75-100 feet between an OSS dispersal component and an individual water well, individual spring, nonmarine surface water or surface water that is not a public water source and a vertical separation of greater than 12 inches, a conforming OSS that complies with WAC 246-272A-0210(4) shall be installed if feasible.

NEW SECTION

WAC 246-272A-0282 Minor repair of malfunctions. The local health officer:

(1) Shall require the minor repair of a malfunction to a functioning state;

(2) May require a permit for a minor repair of a malfunction; and

(3) May require the OSS owner to submit information regarding minor repairs of a malfunction.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0290 Expansions. (1) The local health officer shall require an OSS and a reserve area in full compliance with the new ((~~system~~)) construction standards specified in this chapter for an OSS expansion ((~~of a residence or other facility~~)).

(2) A local health officer may allow expansion of an existing ((~~on-site sewage system adjacent to~~)) OSS within 200 feet of a marine shoreline that does not meet the minimum horizontal separation between the soil dispersal component and the ordinary high-water mark required by WAC 246-272A-0210, Table IV, provided that:

(a) The ((~~system~~)) OSS meets all requirements of WAC 246-272A-0230, 246-272A-0232, 246-272A-0234, and 246-272A-0238;

(b) The ((~~system~~)) OSS complies with all other requirements of WAC 246-272A-0210 and this section;

(c) Horizontal separation between the soil dispersal component and the ordinary high-water mark is ((~~fifty~~)) 50 feet or greater; and

(d) Vertical separation is two feet or greater.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0300 Abandonment. Persons permanently abandoning a ((~~septic~~)) sewage tank, seepage pit, cesspool, or other sewage container shall:

(1) Have the septage removed by an approved pumper; and

(2) Perform one of the following:

(a) Remove and dispose of sewage tanks and other components in a manner approved by the local health officer; or

(b) Leave the sewage tanks and components in place. Remove or destroy the lid((~~,~~)) if possible and ((~~(3)~~)) fill the void with soil or gravel; and

(3) Grade the site to the surroundings.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0310 Septage management. ((~~(1) The local health officer shall approve an individual before they may remove septage from an OSS.~~

~~(2) Persons~~)) A person removing septage from an OSS shall obtain approval from the local health officer before removal and:

((~~(a)~~)) (1) Transport septage or sewage only in vehicles clearly identified with the name of the business and approved by the local health officer;

((~~(b)~~)) (2) Record and report septage removal as required by the local health officer; and

((~~(c)~~)) (3) Dispose of septage, or apply septage biosolids to land only in a manner consistent with applicable laws.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0320 Developments, subdivisions, and minimum land area requirements. ((~~(1) A person proposing a subdivision where the use of OSS is planned shall obtain a recommendation for approval from the local health officer as required by RCW 58.17.150.~~

~~(2) The local health officer shall require the following prior to approving any development:~~

~~(a) Site evaluations as required under WAC 246-272A-0220, excluding subsections (3)(a)(i) and (4)(d);~~

~~(b) Where a subdivision with individual wells is proposed:~~

~~(i) Configuration of each lot to allow a one hundred-foot radius water supply protection zone to fit within the lot lines; or~~

~~(ii) Establishment of a one hundred-foot protection zone around each existing and proposed well site;~~

~~(c) Where preliminary approval of a subdivision is requested, provision of at least one soil log per proposed lot, unless the local health officer determines existing soils information allows fewer soil logs;~~

~~(d) Determination of the minimum lot size or minimum land area required for the development using Method I and/or Method II:~~

~~METHOD I~~. Table X, Single-Family Residence Minimum Lot Size or Minimum Land Area Required Per Unit Volume of Sewage, shows the minimum lot size required per single-family residence. For developments other than single-family residences, the minimum land areas shown are required for each unit volume of sewage. However, the local health officer may require larger lot sizes where the local health officer has identified nitrogen as a concern either through planning activities described in WAC 246-272A-0015 or another process.

~~TABLE X~~
~~Minimum Land Area Requirement~~
~~Single-Family Residence or Unit Volume of Sewage~~

~~Type of Water Supply~~	~~Soil Type (defined by WAC 246-272A-0220)~~					
	~~1~~	~~2~~	~~3~~	~~4~~	~~5~~	~~6~~

Public	0.5 acre 2.5 acre[1]	12,500 sq. ft.	15,000 sq. ft.	18,000 sq. ft.	20,000 sq. ft.	22,000 sq. ft.
Individual, on each lot	1.0 acre 2.5 acres[1]	1 acre	1 acre	1 acre	2 acres	2 acres

[1]See WAC 246-272A-0234(6).

METHOD II. A minimum land area proposal using Method II is acceptable only when the applicant:

(i) Justifies the proposal through a written analysis of the:

(A) Soil type and depth;

(B) Area drainage, and/or lot drainage;

(C) Public health impact on ground and surface water quality;

(D) Setbacks from property lines, water supplies, etc.;

(E) Source of domestic water;

(F) Topography, geology, and ground cover;

(G) Climatic conditions;

(H) Availability of public sewers;

(I) Activity or land use, present, and anticipated;

(J) Growth patterns;

(K) Reserve areas for additional subsurface treatment and dispersal;

(L) Anticipated sewage volume;

(M) Compliance with current planning and zoning requirements;

(N) Types of proposed systems or designs, including the use of systems designed for removal of nitrogen;

(O) Existing encumbrances, such as those listed in WAC 246-272A-0200 (1)(c)(v) and 246-272A-0220 (2)(a)(vii); and

(P) Estimated nitrogen loading from OSS effluent to existing ground and surface water;

(Q) Any other information required by the local health officer.

(ii) Shows development with public water supplies having:

(A) At least twelve thousand five hundred square feet lot sizes per single-family residence;

(B) No more than 3.5 unit volumes of sewage per day per acre for developments other than single-family residences; and

(iii) Shows development with individual water supplies having at least one acre per unit volume of sewage; and

(iv) Shows land area under surface water is not included in the minimum land area calculation; and

(c) Regardless of which method is used for determining required minimum lot sizes or minimum land area, submittal to the health officer of information consisting of field data, plans, and reports supporting a conclusion the land area provided is sufficient to:

(i) Install conforming OSS;

(ii) Assure preservation of reserve areas for proposed and existing OSS;

(iii) Properly treat and dispose of the sewage; and

(iv) Minimize public health effects from the accumulation of contaminants in surface and groundwater.

(3) The department shall develop guidelines for the application of Method II by (*insert date one year from the effective date*).

(4) The local health officer shall require lot areas of twelve thousand five hundred square feet or larger except when a person proposes:

(a) OSS within the boundaries of a recognized sewer utility having a finalized assessment roll; or

~~(b) A planned unit development with:~~

~~(i) A signed, notarized, and recorded deed covenant restricting any development of lots or parcels above the approved density with the overall density meeting the minimum land area requirements of subsection (2)(d) of this section;~~

~~(ii) A public entity responsible for operation and maintenance of the OSS, or a single individual owning the OSS;~~

~~(iii) Management requirements under chapter 246-272B WAC when installing a LOSS; and~~

~~(iv) Extinguishment of the deed covenant and higher density development allowed only when the development connects to public sewers.~~

~~(5) The local health officer may:~~

~~(a) Allow inclusion of the area to the centerline of a road or street right of way in a Method II determination under subsection (2)(d) of this section to be included in the minimum land area calculation if:~~

~~(i) The dedicated road or street right of ways are along the perimeter of the development;~~

~~(ii) The road or street right of ways are dedicated as part of the proposed development; and~~

~~(iii) Lots are at least twelve thousand five hundred square feet in size.~~

~~(b) Require detailed plot plans and OSS designs prior to final approval of subdivision proposals;~~

~~(c) Require larger land areas or lot sizes to achieve public health protection;~~

~~(d) Prohibit development on individual lots within the boundaries of an approved subdivision if the proposed OSS design does not protect public health by meeting requirements of these regulations; and~~

~~(e) Permit the installation of an OSS, where the minimum land area requirements or lot sizes cannot be met, only when all of the following criteria are met:~~

~~(i) The lot is registered as a legal lot of record created prior to the effective date of this chapter;~~

~~(ii) The lot is outside an area identified by the local plan developed under WAC 246-272A-0015 where minimum land area has been listed as a design parameter necessary for public health protection; and~~

~~(iii) The proposed system meets all requirements of these regulations other than minimum land area.~~

~~(6) The use of a reduced-sized SSAS does not provide for a reduction in the minimum land area requirements established in this section. Site development incorporating reduced-sized SSAS must meet the minimum land area requirements established in state and local codes.~~))

(1) Prior to approving any development, the local health officer shall:

(a) Require site evaluations under WAC 246-272A-0220;

(b) Require information consisting of field data, plans, and reports supporting a conclusion that the proposed land area is sufficient to:

(i) Install conforming OSS;

(ii) Preserve reserve areas for proposed and existing OSS; and

(iii) Properly treat and dispose of the sewage;

(c) Require information demonstrating that the proposed development will minimize adverse public health effects from the accumulation of contaminants in groundwater and surface water;

(d) Determine the minimum land area required for the development using Table XI of this section, or the alternative methodology in Ta-

ble XII of this section. The local health officer may require larger lot sizes than the minimum standards established in Table XI or Table XII of this section;

Table XI
Minimum Land Area Requirement For Each Single-Family Residence or Unit Volume of Sewage and Minimum Usable Land Area

		Soil Type (defined by WAC 246-272A-0220)					
		1	2	3	4	5	6
Minimum Land Area	Public Water Supply	21,780 sq. ft. (0.5 acre)	13,000 sq. ft.	16,000 sq. ft.	19,000 sq. ft.	21,000 sq. ft.	23,000 sq. ft.
		2.5 acres[1]					
	Nonpublic Water Supply	1.0 acre	1.0 acre	1.0 acre	1.0 acre	2.0 acres	2.0 acres
		2.5 acres[1]					
Minimum Usable Land Area		2,000 sq. ft.	2,000 sq. ft.	2,500 sq. ft.	3,333 sq. ft.	5,000 sq. ft.	10,000 sq. ft.

[1] OSS consisting of only sewage tanks and gravity SSAS must have a minimum land area of 2.5 acres per WAC 246-272A-0234(7).

Table XII
Maximum Allowable Total Nitrogen (TN) Load Per Day by Type of Water Supply, Soil Type, and Land Area[1]

Water Supply Type	Maximum Daily TN Load	Soil Type[2]					
		1	2	3	4	5	6
Public	mg per sq. ft.	3.8	6.3	5.1	4.3	3.9	3.6
	lb per acre	0.36	0.60	0.49	0.41	0.37	0.34
Nonpublic	mg per sq. ft.	1.9	1.9	1.9	1.9	0.9	0.9
	lb per acre	0.18	0.18	0.18	0.18	0.09	0.09

[1] Based on 60 mg/L TN and 360 gal/day OSS effluent.
[2] As defined in Table V in WAC 246-272A-0220.

(e) Require all proposals not meeting the minimum land area requirements in Table XI of this section to demonstrate the proposed development:

(i) Minimizes adverse impacts to public health, surface water, or groundwater quality;

(ii) Considers:

(A) Topography, geology, and ground cover;

(B) Climactic conditions;

(C) Availability of public sewers; and

(D) Present and anticipated land use and growth patterns;

(iii) Complies with current planning and zoning requirements;

(iv) Does not exceed the nitrogen limit per land area as identified in Table XII of this section; and

(v) Does not allow new lots smaller than 13,000 square feet if served by nonpublic water supplies;

(f) Require minimum land area of 13,000 square feet or larger, except when a proposal includes:

(i) OSS within the boundaries of a recognized sewer utility having a finalized assessment roll; or

(ii) A planned unit development with a signed, notarized, and recorded deed covenant restricting any development of lots or parcels above the approved density with the overall density meeting the minimum land area requirements of (d) or (e) of this subsection in per-

petuity or until the OSS is no longer needed as identified in WAC 246-272A-0200(6);

(g) Require that developments other than single-family residences:

(i) Meet the minimum land areas required for each unit's volume of sewage;

(ii) Do not exceed 3.35 unit volumes of sewage per day per acre if served by public water supplies; and

(iii) Do not exceed 1.0 unit volume of sewage per day per acre for nonpublic water supplies; and

(h) Require that the use of a reduced-sized dispersal component does not result in a reduction of the minimum land area requirements established in this section.

(2) The local health officer shall require the following prior to approving any subdivision:

(a) A recommendation for approval as required by RCW 58.17.150;

(b) Where a subdivision with nonpublic wells are proposed:

(i) Configuration of each lot line to allow a supply protection zone to fit within the lot lines; or

(ii) Water supply protection zones on more than one lot when the person proposing the subdivision or development provides a copy of a recorded restrictive covenant to each property that is sited partially or completely within the water supply protection zone;

(iii) Water supply protection zone of at least 100 foot radius for each existing or proposed well site.

(3) The local health officer may:

(a) Require detailed site plans and OSS designs prior to final approval of subdivision proposals;

(b) Require larger land areas or lot sizes to achieve public health protection;

(c) Prohibit development on individual lots within the boundaries of an approved subdivision if the proposed OSS design does not meet the requirements of this chapter; and

(d) Permit the installation of an OSS, where the minimum land area requirements or lot sizes in Table XI of this section or maximum total nitrogen in Table XII of this section cannot be met, only when the following criteria are met:

(i) The lot is registered as a legal lot of record created prior to the effective date of the rule;

(ii) The lot is not within an area identified in the local management plan developed under WAC 246-272A-0015 where minimum land area is listed as a design parameter necessary for public health protection; and

(iii) The proposed OSS meets all requirements of this chapter without the use of a waiver under WAC 246-272A-0420.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 7/1/07)

WAC 246-272A-0340 ((~~Certification~~)) Approval of installers, pumpers, and maintenance service providers. (1) OSS installers ((~~and~~)), pumpers ((~~must~~)), and maintenance service providers shall obtain approval from the local health officer prior to providing services including, but not limited to, conducting inspections in accord-

ance with WAC 246-272A-0260 and 246-272A-0270, within a local health jurisdiction.

(2) The local health officer ((~~may~~)) shall establish ((~~programs and requirements~~)) procedures for approving OSS installers, pumpers, and maintenance service providers no later than February 1, 2025. These procedures must include, but are not limited to, conducting inspections in accordance with WAC 246-272A-0260 and 246-272A-0270. The local health officer may approve OSS installers, pumpers, and maintenance service providers through reciprocity by other Washington local health jurisdictions.

(3) The local health officer may establish a homeowner OSS inspection certification process.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0400 Technical advisory ((~~committee~~)) group (TAG). ((~~(1)~~)) The department shall:

((~~(a)~~)) (1) Maintain a ((~~technical advisory committee~~)) TAG to advise the department regarding:

((~~(i)~~)) (a) OSS design and siting;

((~~(ii)~~)) (b) Public domain technologies ((~~and recommended standards and guidance~~)), DS&G for ((~~their~~)) product use; and

((~~(iii)~~)) (c) Testing and design standards used for proprietary product registration and ((~~recommended standards and guidance~~)) DS&G for use of proprietary products.

((~~(b)~~)) (2) Select members for the ((~~technical advisory committee with~~)) TAG for three-year terms that have technical or scientific knowledge applicable to OSS from agencies, professions, and organizations including:

((~~(i)~~)) (a) Local health ((~~departments~~)) jurisdictions;

((~~(ii)~~)) (b) Engineering firms;

((~~(iii)~~)) (c) The Washington department of ecology;

((~~(iv)~~)) (d) Land sales, development and building industries;

((~~(v)~~)) (e) Public sewer utilities;

((~~(vi) On-site sewage system design and installation firms;~~

~~(vii)~~)) (f) OSS:

(i) Designers;

(ii) Installers;

(iii) Maintenance service providers;

(iv) Product manufacturers;

(g) Environmental organizations;

((~~(viii)~~)) (h) University((~~/~~)) and college academic communities;

((~~(ix) On-site sewage system or related product manufacturers~~)) (i) Certified professional soil scientists; and

((~~(x)~~)) (j) Other interested organizations or groups.

((~~(c) Convene meetings as needed.~~

~~(2) The department may have a representative on the technical advisory committee.~~))

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0410 Policy advisory ((~~committee~~)) group. ((~~(1)~~)) The department shall:

((~~(a)~~)) (1) Maintain a policy advisory ((~~committee~~)) group to:

((~~(i)~~)) (a) Make recommendations concerning OSS departmental policy and ((~~regulations~~)) rules;

((~~(ii)~~)) (b) Review OSS program services; and

((~~(iii)~~)) (c) Provide input to the department regarding the ((~~on-site sewage~~)) OSS program;

((~~(b)~~)) (2) Select members for three-year terms from agencies, professions, organizations having knowledge and interest in OSS, and ((~~groups~~)) communities which are affected by ((~~the regulations; and~~

~~(c) Convene meetings as needed.~~

~~(2) The department may have a representative on the policy advisory committee~~)) this chapter.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0420 Waivers ((~~of state regulations~~)). (1) The local health officer may grant a waiver from specific requirements of this chapter ((~~if~~)). A request for waiver must be:

(a) ((~~The waiver request is~~)) Evaluated by the local health officer on an individual, site-by-site basis;

(b) ((~~The local health officer determines that the waiver is~~)) Consistent with the ((~~standards in, and the intent of, these rules;~~

~~(c)~~)) purposes of this chapter.

(2)(a) The local health officer must submit((~~s~~)) quarterly reports to the department ((~~regarding any~~)) showing waivers approved or denied((~~; and~~

~~(d) Based on review of the quarterly reports~~)).

(b) Upon review, if the department finds that the waivers previously granted ((~~have not been consistent~~)) are inconsistent, with the ((~~standards in, and the intent of these rules~~)) purposes of this chapter, and DS&G for granting waivers, the department shall provide technical assistance to the local health officer to correct the inconsistency, and may notify the local and state boards of health of the department's concerns.

(c) If upon further review ((~~of the quarterly reports~~)), the department finds ((~~that the inconsistency between the waivers granted and the state board of health standards has not been corrected~~)) waivers previously granted continue to be inconsistent with the purposes of this chapter and DS&G, the department may suspend the authority of the local health officer to grant waivers under this section until such inconsistencies have been corrected.

((~~(2)~~)) (3) The department shall ((~~develop~~)) maintain and update guidance to assist local health officers in the application of waivers.

(4) The department shall publish an annual report summarizing the waivers issued over the previous year.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0425 Required ((~~rule~~)) review of rules. The department shall review this chapter to evaluate the effectiveness of the rules ((~~and determine areas where revisions may be necessary. The department will provide the results of their review along with their~~)), determine where revisions may be necessary, and make recommendations to the state board of health and all local health officers by September ((~~2009~~)) 2026 and every four years thereafter.

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0430 Enforcement. (1) When an OSS is out of compliance with any law or rule regulating OSS and administered by the department or the local health officer, the department or the local health officer((~~:~~

~~(a) Shall enforce the rules of chapter 246-272A WAC; or~~

~~(b) May refer cases within their jurisdiction to the local prosecutor's office or office of the attorney general, as appropriate.~~

~~(2) When a person violates the provisions under this chapter, the department, local health officer, local prosecutor's office, or office of the attorney general may initiate enforcement or disciplinary actions, or any other legal proceeding authorized by law including, but not limited to, any one or a combination of the following:~~

~~(a) Informal administrative conferences, convened at the request of the department or owner, to explore facts and resolve problems;~~

~~(b) Orders directed to the owner and/or operator of the OSS and/or person causing or responsible for the violation of the rules of chapter 246-272A WAC;~~

~~(c) Denial, suspension, modification, or revocation of permits, approvals, registrations, or certification;~~

~~(d) The penalties under chapter 70.05 RCW and RCW 43.70.190; and~~

~~(e) Civil or criminal action.~~

~~(3) Orders authorized under this section include the following:~~

~~(a) Orders requiring corrective measures necessary to effect compliance with chapter 246-272A WAC which may include a compliance schedule; and~~

~~(b) Orders to stop work and/or refrain from using any OSS or portion of the OSS or improvements to the OSS until all permits, certifications, and approvals required by rule or statute are obtained.~~

~~(4) Enforcement orders~~)) may initiate enforcement action. Enforcement action may include, but is not necessarily limited to:

(a) A notice of correction describing the condition that is not in compliance and the text of the specific section or subsection of the applicable state or federal law or rule, a statement of what is required to achieve compliance, and the date by which compliance is to be achieved;

(b) A notice of violation with or without a civil penalty;

(c) An order requiring specific actions or ceasing unacceptable activities within a designated time period;

(d) Suspension, revocation, or modification or denial of permits and licenses as authorized by RCW 43.70.115; and

(e) Civil or criminal penalties authorized under chapter 70.05 RCW and RCW 43.70.190.

(2) An informal conference may be held at the request of any party to resolve disputes arising from enforcement of this chapter.

(3) Notices and orders issued under this section ((~~shall~~)) must:

(a) Be in writing;

(b) Name the person or persons to whom the order is directed;

(c) Briefly describe each action or inaction constituting a violation of the rules of chapter 246-272A WAC, or applicable local ((~~code~~)) rules;

(d) Specify any required corrective action, if applicable;

(e) Specify the effective date of the order, with time or times of compliance;

(f) Provide notice of the consequences of failure to comply or repeated violation, as appropriate((~~. Such notices may include a statement that continued or repeated violation may subject the violator to:~~

~~(i) Denial, suspension, or revocation of a permit approval, or certification;~~

~~(ii) Referral to the office of the county prosecutor or attorney general; and/or~~

~~(iii) Other appropriate remedies.~~

~~(g) Provide the name, business address, and phone number of an appropriate staff person who may be contacted regarding an order~~)).

((~~(5)~~)) (4) Enforcement orders ((~~shall~~)) must be personally served in the manner of service of a summons in a civil action or in ((~~a~~)) another manner showing proof of receipt.

((~~(6)~~)) (5) The department shall have cause to deny the application or reapplication for ((~~an operational~~)) a permit or to revoke, suspend, or modify a required ((~~operational~~)) permit of any person who has:

(a) Failed or refused to comply with the provisions of chapter 246-272A WAC, or any other statutory provision or rule regulating the operation of an OSS; or

(b) Obtained or attempted to obtain a permit or any other required certificate or approval by misrepresentation.

((~~(7) For the purposes of subsection (6) of this section and WAC 246-272A-0440, a person is defined to include:~~

~~(a) Applicant;~~

~~(b) Reapplicant;~~

~~(c) Permit holder; or~~

~~(d) Any individual associated with (a), (b) or (c) of this subsection including, but not limited to:~~

~~(i) Board members;~~

~~(ii) Officers;~~

~~(iii) Managers;~~

~~(iv) Partners;~~

~~(v) Association members;~~

~~(vi) Agents; and~~

~~(vii) Third persons acting with the knowledge of such persons.~~))

AMENDATORY SECTION (Amending WSR 05-15-119, filed 7/18/05, effective 9/15/05)

WAC 246-272A-0440 Notice of decision—Adjudicative proceeding. (1) All local boards of health shall:

(a) Maintain an ((~~administrative appeals~~)) adjudicative process to ((~~consider~~)) resolve procedural and technical conflicts arising from the administration of local regulations; and

(b) Establish rules for conducting hearings requested to contest a local health officer's actions.

(2) The department shall provide notice of the department's denial, suspension, modification, or revocation of a permit, certification, or approval consistent with RCW 43.70.115, chapter 34.05 RCW, and chapter 246-10 WAC.

(3) A person contesting a departmental decision regarding a permit, certificate, or approval may file a written request for an adjudicative proceeding consistent with chapter 246-10 WAC.

(4) Department actions are governed ((~~under the Administrative Procedure Act~~)) by chapter 34.05 RCW, RCW 43.70.115, this chapter, and chapter 246-10 WAC.

REPEALER

The following sections of the Washington Administrative Code are repealed:

WAC 246-272A-0020	Applicability.
WAC 246-272A-0125	Transition from the list of approved systems and products to the registered list—Treatment products.
WAC 246-272A-0135	Transition from the list of approved systems and products to the registered list—Bacteriological reduction.
WAC 246-272A-0150	Transition from the list of approved systems and products to the registered list—Distribution products.
WAC 246-272A-0175	Transition from the experimental system program to application for product registration.

WASHINGTON STATE BOARD OF HEALTH

HEALTH PROMOTION COMMITTEE SPECIAL MEETING SUMMARY NOTES

What: Health Promotion (HP) Committee

When: February 1, 2024

Participating: Board of Health (Board) Members Dimyana Abdelmalek (Committee Chair), Patty Hayes, Steve Kutz, Kelly Oshiro; Board staff Molly Dinardo, Andrew Kamali, Michelle Davis, Michelle Larson, Melanie Hisaw, Ashley Bell, Shay Bauman; Department of Health (Department) staff; and approximately five members of the public also attended the meeting.

Summary Notes:

Rulemaking and Other Project Updates

- Molly Dinardo, Board staff, stated that since the last committee meeting in December, the Board hasn't had any new rule filings, and there are no new major updates to Board rule projects.
- Molly then brought up the topic of kratom and mentioned that Member Kutz has asked if the Board or Health Promotion Committee could receive a briefing on the topic. Molly asked Committee Members about their interest in receiving a briefing on kratom for the May Health Promotion Committee meeting.
- Member Steve Kutz, Member Kelly Oshiro, and Member Patty Hayes supported receiving a briefing on this topic at the May meeting.
- Member Kutz also expressed concern about another substance called tianeptine. Member Kutz asked if tianeptine could be included in a future briefing and if there could eventually be a broader briefing brought to the full Board on these topics.
- Member Hayes encouraged Member Kutz to work with staff to organize a briefing for the May Health Promotion meeting as a starting point. Member Hayes said staff should see if the Food and Drug Administration (FDA) is looking at these topics, and if they are, perhaps the Board could pass a resolution of concern or write a letter to ask the FDA to examine this further.
- Molly shared some information about tianeptine, stating that it is not an approved drug or substance in the U.S. Molly also added that the FDA has sent out several letters and alerts about tianeptine products.
- HP Committee Chair Dimyana Abdelmalek added that the Centers for Disease Control (CDC) recently reported on tianeptine in its Morbidity and Mortality Weekly Report (MMWR).

(Continued on the next page)

- Member Kutz asked Chair Abdelmalek to send the article. Member Kutz stated that unless these substances rise to the level of a poison center notification, these addictive substances can go largely unnoticed. Member Kutz inquired how the Board can send their concern to the FDA and what mechanisms the state uses to identify substances of concern. Member Kutz emphasized that these substances are not only a concern for adults, but also kids.
- Michelle Davis, Board Executive Director, said staff would pull up the article and add it to the meeting notes so both Committee Members and the public can access it: https://www.cdc.gov/mmwr/volumes/73/wr/mm7304a5.htm?s_cid=mm7304a5_w

2024 Legislative Session Updates

- Molly Dinardo, Board staff, shared that January 31 was the House of Origin Policy Committee cutoff and provided information about the number of bills Board staff are tracking and analyzing.
- Molly gave an overview of health promotion-related bill topics for this session and flagged two newborn screening related bills that staff are monitoring closely. Molly asked Committee Members if they had any questions.
- Member Hayes asked about the syphilis bill, and Molly said it passed the Senate Policy Committee and it's waiting on a floor vote in the Rules committee.

Preview March Board Meeting

- Molly Dinardo, Board staff, shared that staff are in the process of organizing two State Health report community panels, one at the March meeting, and the other at the April meeting to help inform the 2024 State Health Report to the Governor. Molly recapped Board Member feedback and questions from the January briefing and said these are considerations that staff are trying to keep in mind as they plan these upcoming panels.
- Member Oshiro thanked staff for taking Board Member feedback into consideration and stated that it's a good idea to have these panels split into two.
- Member Hayes said it would be helpful for the Board to review what we've done before as a grounding. Member Hayes also expressed interest in brainstorming how to better align Board reports, recommendations, and policies over time and stated that the high balcony framework of issues across the state should be informed by what is happening locally. Member Hayes shared conversations with Department staff about the State Health Improvement Plan (SHIP) and a desire to somehow align reports and priorities down the line to give the Board's State Health Report more meaning.
- Member Kutz provided feedback on recent Board community panels and stated that it would be helpful to include more time for discussion after the panels.
- John Thompson, Department staff, provided an overview of the presentation that the Department plans to present for the Newborn Screening Annual Report presentation at the March meeting. John shared that the Newborn Screening Program has quarterly reports that feed into the annual report and that in September 2022, they hired an epidemiologist, Anna Howard, to help take these

reports and input them into a data dashboard. John said they anticipate the dashboard to be live by the March meeting. Anna Howard, Department staff, shared a preview of what the dashboard visualizations will look like.

- Member Kutz expressed interest in some of the program's quality improvement measures. John said they plan to present some information on this during the full presentation in March.
- Ashley Bell, Board staff, introduced Pro-Equity Anti-Racism (PEAR) Planning and stated that the goal of a PEAR plan is to bridge disparity gaps and reduce disparities statewide and across state government. Ashley provided information about how the PEAR plan works, the goals and outcomes of a PEAR plan, and how the Board can engage in this work. Ashley shared a proposed timeline with Committee Members and added that the presentation in March will provide more details and background on the Board's upcoming PEAR plan work.
- Member Hayes asked if the PEAR Plan comes from a legislative mandate or executive order and inquired if there's a model plan that the Board could work from. Member Hayes also mentioned the Foundational Public Health Services (FPHS) Equity Technical Working Group and said that in the meetings so far, no one has brought up the topic of PEAR. Member Hayes said that if the Department isn't going to bring up the topic of a PEAR plan, maybe the Board could bring that perspective in. Member Hayes also indicated that the Board has not developed an updated strategic plan and was curious about how the Board could harmonize the PEAR Plan and Strategic Plan.
- Ashley said the plan is for the Board to connect PEAR with the future Strategic Plan. Ashley also clarified that the PEAR Plan and the Office of Equity are both Executive Orders in the state.
- Michelle Davis, Board Executive Director, thanked Member Hayes for the questions and said that staff need to develop a timeline for the Strategic Plan, and in the meantime, the PEAR plan can provide us with a foundation.

Preview April Board Meeting

- Molly Dinardo, Board staff, shared that in April, the Board can expect a briefing from the Department on the recent implementation of the updated notifiable conditions rule.
- Member Hayes noted that this subject has generated a lot of public interest in the past and that Board staff may want to anticipate public comments related to this topic.
- Member Kutz commented that the notifiable conditions rule has become a treadmill. Member Kutz added that it would be helpful for the Board to get feedback about a year or so after rule implementation to assess what impact recent rule changes have had.
- Michelle Davis, Board Executive Director, said that Member Kutz brought up a good point, and hearing about implementation from the Department will be helpful.

Committee Member Comments, Questions, and Next Steps

- Member Kutz thanked staff for preparing the background work for the meeting.
- HP Committee Chair Abdelmalek thanked staff and presenters.

To request this document in an alternate format or a different language, please contact the State Board of Health at 360-236-4110 or by email wsboh@sboh.wa.gov. TTY users can dial 711.

WASHINGTON STATE BOARD OF HEALTH

ENVIRONMENTAL HEALTH COMMITTEE SPECIAL MEETING SUMMARY NOTES

What: Environmental Health (EH) Committee

When: February 8, 2024

Participating: Board of Health (Board) Members Kate Dean (Committee Chair), Paj Nandi, Mindy Flores; Board staff Michelle Davis, Ashley Bell, Andrew Kamali, Shay Bauman, Anna Burns, Melanie Hisaw, Molly Dinardo; Department of Health (Department) staff; and approximately seven members of the public.

Summary Notes:

General Updates

- Andrew Kamali, Board staff, provided a legislative session update and provided the number of bills the Board is tracking.
- Joe Laxon, Department staff, shared information about the house of origin cut-off the following week. Joe also shared information on long committee meetings, the Rules Committee being very busy, and the Department tracking several bills. The Department and the Board are watching floor action, watching for amendments on the floor, and working on amendments from the perspective of the Department. Joe said draft budgets are expected next week.
- Specific bills that Joe discussed:
  1. HB2301, Organics bill, puts the Department and Ecology in the lead. It relates to decreasing organic waste through increasing donations and diverting food waste.
  2. SB 6187, Body Scanner bill, this bill raises concerns about higher radiation scanners in certain departments of corrections facilities.
  3. HB 1010, Crab bill, requires rulemaking by the Board. The bill is still in rules committee and has been relieved of further consideration, question is if it will be reviewed before cutoff.
- Member Nandi shared appreciation of the work everyone is doing and the number of bills.

Preview March Meeting

- Molly Dinardo, Board staff, provided the update. Molly and Hannah, Board staff, have created two community panels to help inform themes for the 2024 State Health report.

(Continued on the next page)

- Molly shared additional background on this report and the development of the two community group panels. Molly said the State Health report is submitted every two years. The first community group panel is at the March 13 meeting at the Swinomish Tribal community and April 12 is focused on community groups in the Eastern part of the state.
- Molly shared that the topics were developed using previous reports and Board Member feedback. The topics include maternal and pregnant person health, data equity, and culturally appropriate care. Molly shared that the State Health report focuses on timely issues, community-aligned and actionable items. Board Members want to learn from local health, making sure the Board identifies areas to meet in communities. Molly said the community group panels are to be interactive and discussion based. Molly asked how staff can help Board Members.
- Member Flores thanked Molly and Hannah.
- Member Nandi talked about responsiveness and working with Molly and Hannah. EH Committee Chair Dean agreed with Member Nandi on responsiveness.
- EH Committee Chair Dean talked about the many determinants of health and how to stay in our lane but keep the lens broad enough and be inclusive respectfully. Molly agreed on concerns about over-promising, but to incorporate community voice and feedback.
- EH Committee Chair Dean mentioned that this is the first report that the new Governor will be receiving. Member Nandi mentioned the misnomer of State Health report and to be clear of the intent and framing of it as more of a guiding document that the Board is putting out for the Governor. Molly talked about the disclaimer on the report, that it is a snippet of policy direction and a guiding document.

Preview April Board Meeting

- Ashley Bell, Board staff, talked about the Pro-Equity Anti-Racism (PEAR) playbook and plan. Ashley discussed the background on PEAR and how it drives systemic change, aiming to dismantle oppressive systems and promote equity in all facets of society. Ashley also shared PEAR recognizes that systems of oppression are the upstream sources of all our inequities, and therefore, addressing these systems is crucial to creating a more equitable world. Ashley discussed the 15 Determinants of Equity.
- EH Committee Chair Dean said they are anxious to learn more about PEAR in March. Member Nandi said this is near and dear to their heart, they were at the Department when this was being conceived. Member Nandi stated they are happy to help and support and curious to see outcomes. EH Committee Chair Dean said they are looking forward to learning how this nests with other equity work and how it differs. Ashley asked for any helpful feedback.

Other Environmental Health (EH) Rulemaking Updates

- Andrew Kamali, Board staff, said the Board is moving several items to June, including shellfish, instead of April. Katitza Holthaus, Department staff, said in November the Board delegated this rulemaking. Initially, the Department staff

thought they could give the Board an abbreviated rulemaking update in April. Katitza said that currently, the CR-102 is moving, but a public hearing will be later in April, so they need to move this agenda item to June.

- EH Committee Chair Dean asked when Department staff anticipate the final rule to be completed. Katitza said if all goes well, the CR-103 should be done sometime in May, so rules become effective 30 days after. That gives a year for the development of the Climate Resilience Plans by the utilities, Mike Means, Department staff said not all water systems will have this. EH Committee Chair Dean talked about counties planning updates.
- Andrew gave an update on the On-site Sewage System (OSS) rule. Katitza shared that the CR-103 is moving through an internal process and hoping to file end of February, the packet is large and takes longer to review. Department staff are trying to get all OSS rules in order and have timelines worked out. Andrew said moving forward steadily and can be mostly behind us.

Committee Member Comments, Questions, and Next Steps

- EH Committee Chair Dean opened the space for discussion.
- Michelle Davis, Board Executive Director, wondered about some unstructured time to discuss.
- EH Committee Chair Dean wondered if the Committee could discuss the School Rules. Michelle said indoor air quality affects many areas of our rulemaking authority, school rules, transient accommodations, and more. Andrew, Board staff, wondered if it would be helpful to draft an overview to help facilitate discussion. Andrew recognized that the indoor air quality panel was in-depth and had a lot of information. Member Nandi liked that idea and wondered about some specific asks the Board can act on or provide support. Andrew can work on that.
- EH Committee Chair Dean talked about the effort supported by the Washington State Association of Local Public Health Officials sponsoring a proviso for a statewide septage study.
- Michelle thanked Member Flores for all the help facilitating the Boards March meeting. Michelle noted that this is the first time the Board is meeting in a Tribal facility, and it would not be possible without Member Flores efforts. Member Flores thanked the Swinomish Tribe for all their collaboration.

From: bill teachingsmiles.com
To: DOH WSBOH
Subject: Re: Hearing
Date: Wednesday, January 24, 2024 3:28:22 PM
Attachments: image001.png

External Email

“Not to speak is to speak.
Not to act is to act.”

— Dietrich Bonhoeffer

Please remind the Board, the harm is serious.

To my request for the Board to hold a forum to protect the public health, the Board responded in effect: “later maybe.”

85,085 births reported in WA in 2019, 56% on fluoridation is 47,648 infants born in fluoridated communities X 4.3/1,000 infant fatalities = 205 deaths in fluoridated communities. If the data is reasonable reporting an increased rate of about 20% higher infant mortality in fluoridate states, perhaps every 10 to 14 days a baby dies because of the Board's silence. 40 a year more deaths in fluoridated communities. The longer the Board delays, the more babies may die. What about miscarriage? What about IQ loss? What about our schools and prisons with more because their brains are being poisoned? Thyroid harm? ADHD increased? Dental fluorosis functional and cosmetic harm? Increased bone fractures?

Fluoridation possibly saving a quarter or half a filling per child is not worth lower IQ or a single infant death.

Bill Osmunson DDS MPH

From: DOH WSBOH <WSBOH@SBOH.WA.GOV>
Sent: Wednesday, January 24, 2024 9:23 AM
To: bill teachingsmiles.com <bill@teachingsmiles.com>
Subject: RE: Hearing

Dear Mr. Osmunson,

Thank you for reaching out. Board staff will bring your request to the next full Board meeting for Board Member input. The next meeting will be March 13, 2024.

Please note that when staff recently brought the topic of fluoride exposure to the

From: bill teachingsmiles.com
To: DOH WSBOH
Subject: Re: Hearing
Date: Wednesday, January 24, 2024 1:18:14 PM
Attachments: image001.png

External Email

Thank you for responding, although I'm disappointed.

Bill

From: DOH WSBOH <WSBOH@SBOH.WA.GOV>
Sent: Wednesday, January 24, 2024 9:23 AM
To: bill teachingsmiles.com <bill@teachingsmiles.com>
Subject: RE: Hearing

Dear Mr. Osmunson,

Thank you for reaching out. Board staff will bring your request to the next full Board meeting for Board Member input. The next meeting will be March 13, 2024.

Please note that when staff recently brought the topic of fluoride exposure to the Board's May 2023 Environmental Health and Health Promotion Committee meetings, Committee Members stated they were not interested in holding a forum on water fluoridation.

We will follow up after the Board meeting with Board Member feedback on your request.
Best,

WASHINGTON STATE BOARD OF HEALTH

Phone: (360) 236-4110
Mailing Address: P.O. Box 47990, Olympia, WA 98504-7990

Location · Website · Email · Facebook · Twitter · Subscribe

From: bill teachingsmiles.com <bill@teachingsmiles.com>
Sent: Saturday, January 20, 2024 9:32 AM
To: DOH WSBOH <WSBOH@SBOH.WA.GOV>
Subject: Hearing

Dear Patty Hayes,

Several years ago, we made 19 petitions to the Board of Health to protect the public health from excess fluoride exposure. All were denied. In part because the Board members failed to understand the science and laws and simply followed the dental lobby. In fact, the Board took a more aggressive approach on their website promoting fluoridation with false misleading statements. The Board is complicit in the serious harm from excess fluoride exposure.

The scientific evidence has increased and measurements of harm have increased. The National Toxicology Program determination that fluoride is a presumed developmental neurotoxin, measurements of thyroid harm, bone harm, teeth harm, a cost benefit analysis, increased infant mortality. . . yes, the death of infants. . . and more need to be understood by the Board for judgment.

A 2 or 3 minute presentation at the Board meetings is inadequate to provide an understanding of the harm from excess fluoride. And simply saying "The Board is increasing the deaths of babies" maybe precise and possible, but without foundation appears sensational and cannot be scientifically understood.

RCW 43.20.050 in part says, "It is further empowered to hold hearings and explore ways to improve the health status of the citizenry."

How do I go about requesting the Board to hold hearings and explore ways to improve the health status of the citizenry regarding fluoride exposure, where experts on both sides of the fluoridation controversy can present evidence?

I am unaware of hearings at our last petitions. And hearings can be manipulated depending on the choice of the speakers. For example, if only the tobacco lobbyists were invited to a hearing on tobacco safety, the conclusion can be predicted before the hearings.

Local, national and international quality well published researchers, Federal Government scientists with world-wide respect and knowledge could be asked to present at no charge to the Board.

Under the Toxic Substances Control Act, a 9-day hearing in the Superior Court of Northern California on just the developmental neurotoxicity of fluoride will begin in 10

days. Short segments of their sworn testimony from both plaintiffs and defendant (EPA) could be presented and save the Board days of hearings. Other risks, dosage, benefit, and jurisdiction will not be covered in that Court case.

My request is for hearings on fluoride exposure. How do I go about that request?

Sincerely,
Bill Osmunson DDS MPH
425.466.0100

Washington State Department of HEALTH

ANNUAL REPORT - 2023

Newborn Screening Program

Reporting Requirements

(1) The department shall report to the board annually the following information concerning tests conducted under WAC 246-650-020:

- (a) The costs of tests as charged by the department;
- (b) The results of each category of tests, by county of birth and racial or ethnic group, as reported on the newborn screening specimen/information form; and
- (c) Follow-up procedures and the results of such follow-up procedures.

(2) The department shall compile an annual report for the public that includes:

- (a) The compliance rate of each hospital in meeting the deadlines established under RCW **70.83.020** for newborn screenings; and
- (b) The performance rate of each individual hospital.
- (3) The reports must be made available in a format that does not disclose the identifying information related to any infant, parent or guardian, or health care provider.
- (4) The report must be posted in an accessible location on the department of health's website.

Program Updates

- Screening Costs
  - Fee charged for each infant through the facility that collected the initial specimen
  - The charge was increased from **$119.30** to **$135.10** on 7/1/23
    - Cystic Fibrosis DNA testing expanded
      - New real-time PCR instruments with higher testing capacity
    - Courier implementation (ongoing)
      - Next-day delivery for newborn specimens from any birth setting

Program Updates

- Cases by County
  - King – 82
  - Pierce – 29
  - Snohomish – 18
  - Spokane – 17
  - Clark – 10

  - Douglas – 1 (out of only 6 births in the county!)

Program Updates

- Cases by Race & Ethnicity (N = 217)

- 34.5% of babies were non-white
- 55.6% of cases were non-white

- Most common condition in all races was congenital hypothyroidism, except in black babies (hemoglobin disorders)

Program Updates

- Follow-up of Confirmed Cases
  - 205/206 were seen by specialists
  - 1 baby died (unrelated to their NBS condition)

- Median Age at Treatment
  - Amino acid disorders – 7d
  - Hemoglobin disorders – 10.5d
  - Cystic fibrosis – 15d
  - Organic acid disorders – 124.5d

Program Updates

- New Conditions
  - OTCD deficiency
  - GAMT deficiency
  - ARG1 deficiency

- Candidate Conditions
  - cCMV infection – 2025 review
  - MPS-II – 2025 review
  - BCKDK deficiency – likely 2025 review

Program Updates

- Regional Newborn Screening Program
  - Washington State
  - Hawaii (2018)
  - Idaho (2021)

- Organizational Changes – New Positions
  - NBS deputy director
  - Method development chemist
  - NBS quality improvement specialist
  - NBS health information exchange epidemiologist

Public Report

- Report will be available as a Power BI dashboard on the Newborn Screening Program website
- Includes compliance metrics, transit performance, and specimen quality indicators

Infants Detected with Newborn Screening Disorders by County of Residence (births by county of occurrence)

County	Births	Amino acid disorders	Biotinidase deficiency	Congenital adrenal hyperplasia	Congenital hypothyroidism	Cystic fibrosis	Fatty acid oxidation disorders	Galactosemia	Hemoglobinopathies	Organic acid disorders	Mucopolysaccharidosis type I	Pompe	Severe combined immunodeficiency	Spinal muscular atrophy	X-linked adrenoleukodystrophy	All Infants Detected
Adams	405	-	-	-	1	-	-	-	-	-	-	-	-	-	-	1
Asotin	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Benton	4,699	-	-	-	3[a]	1	-	-	-	-	-	-	1	-	-	5
Chelan	1,417	-	-	1	1	-	-	-	-	-	-	-	-	-	-	2
Clallam	452	-	-	1	-	-	-	-	-	-	-	-	-	-	-	1
Clark	5,479	-	-	1	8	-	-	-	1	-	-	-	-	-	-	10
Columbia	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cowlitz	813	-	-	-	2	-	-	-	-	-	-	-	-	-	-	2
Douglas	6	-	-	1	-	-	-	-	-	-	-	-	-	-	-	1
Franklin	23	-	-	1	3	-	-	-	-	-	-	-	-	-	-	4
Grant	1,072	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Grays Harbor	326	-	-	-	1	-	-	-	-	-	-	-	-	-	-	1
Island	265	1	-	-	2	-	-	1	-	-	-	-	1	-	1	6
Jefferson	96	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
King	27,971	2	-	3	57	2	3	1	6	-	-	-	3	1	4	82
Kitsap	1,984	-	-	-	5	1	-	-	-	-	-	-	-	-	-	6
Kittitas	249	-	-	-	1	-	-	-	-	-	-	-	-	-	-	1
Klickitat	24	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Lewis	714	-	-	-	2	-	-	-	-	-	-	-	-	-	-	2
Lincoln	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Mason	391	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Okanogan	325	-	-	-	2	-	-	-	-	-	-	-	-	-	-	2
Pacific	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Pend Oreille	44	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Pierce	11,226	2	-	1	13	3	1	-	5	-	-	-	3	-	1	29
San Juan	7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Skagit	1,403	-	-	-	2	-	-	-	-	-	-	-	-	-	-	2
Skamania	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Snohomish	5,970	-	-	-	12	1	-	-	1	1[b]	-	-	1	1	1	18
Spokane	6,675	-	-	1	9	3	1	-	-	1	-	-	-	1	1	17
Stevens	223	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Thurston	2,626	-	-	-	5	1	-	-	1	-	-	-	-	-	2	9
Walla Walla	618	-	-	-	1	-	-	-	-	-	-	-	-	-	-	1
Whatcom	2,041	1	-	-	5	1	-	-	-	-	-	-	-	-	-	7
Whitman	480	-	-	-	2	-	-	-	-	-	-	-	-	-	1	3
Yakima	2,644	-	-	-	3	-	-	-	1	-	-	1	-	-	-	5
All WA Births	**80,698[c]**	**6**	**-**	**10**	**140**	**13**	**5**	**2**	**15**	**2**	**-**	**1**	**9**	**3**	**11**	**217**

[a] includes 1 infant born in Benton County and resides out of state; [b] includes 1 infant born in Snohomish County and resides out of state ; [c] includes 14 infants with an unknown county of birth

Infants Detected with Newborn Screening Disorders by Infant's Reported Race and Ethnicity

County	Births	Amino acid disorders	Biotinidase deficiency	Congenital adrenal hyperplasia	Congenital hypothyroidism	Cystic fibrosis	Fatty acid oxidation disorders	Galactosemia	Hemoglobinopathies	Organic acid disorders	Mucopolysaccharidosis type I	Pompe	Severe combined immunodeficiency	Spinal muscular atrophy	X-linked adrenoleukodystrophy	All Infants Detected
Asian	9,367	1	-	-	28	-	1	-	3	-	-	-	-	-	1	34
Black	3,991	-	-	-	4	-	-	-	9	-	-	-	1	-	-	14
Native American	1,150	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
White	49,654	5	-	4	52	12	4	1	-	-	-	1	4	3	8	94
Other	6,641	-	-	2	9	-	-	-	-	-	-	-	2	-	-	13
Multiple	5,053	-	-	4	17	1	-	-	2	-	-	-	2	-	1	27
Unknown	4,842	-	-	-	30[a]	-	-	1	1	2[b]	-	-	-	-	1	35
Total	**80,698**	**6**	**-**	**10**	**140**	**13**	**5**	**2**	**15**	**2**	**-**	**1**	**9**	**3**	**11**	**217**

[a] includes 1 infant of an unknown race with CH who resides out of state; [b] includes 1 infant of unknown race with an organic acid disorder who resides out of state

Ethnicity	Births	Amino acid disorders	Biotinidase deficiency	Congenital adrenal hyperplasia	Congenital hypothyroidism	Cystic fibrosis	Fatty acid oxidation disorders	Galactosemia	Hemoglobinopathies	Organic acid disorders	Mucopolysaccharidosis type I	Pompe	Severe combined immunodeficiency	Spinal muscular atrophy	X-linked adrenoleukodystrophy	All Infants Detected
Hispanic	20,063	1	-	1	27	1		-	1	1	-	-	-	1	-	33

Follow-Up Status of Infants Detected with Severe Newborn Screening Disorders

Follow-Up	Amino acid disorders	Biotinidase deficiency	Congenital adrenal hyperplasia	Congenital hypothyroidism	Cystic fibrosis	Fatty acid oxidation disorders	Galactosemia	Hemoglobinopathies	Organic acid disorders	Mucopolysaccharidosis type I	Pompe	Severe combined immunodeficiency	Spinal muscular atrophy	X-linked adrenoleukodystrophy	Total
Referred to medical specialist – (i.e., pediatric endocrinologist, hematologist, or comprehensive clinic)	6	-	10	139[a]	13	4	-	15	2	-	-	2	3	11	205
Followed by primary care provider, with some consultation from specialist	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Infant died or Lost to Follow-Up	-	-	-	1[b]	-	-	-	-	-	-	-	-	-	-	1
Total	**6**	**-**	**10**	**140**	**13**	**4**	**-**	**15**	**2**	**-**	**-**	**2**	**3**	**11**	**206**

[a] includes both mild and severe CH cases; [b] includes 1 micropreemie with CH who expired on day of life 50

Age at which Treatment Began for Infants Detected with Severe Newborn Screening Disorders

Disorder	Number of Infants	Age Treatment Began (Days)	
		Median	Range
Amino acid disorders	6	7	6-24
Biotinidase deficiency	-	-	-
Congenital adrenal hyperplasia	10	11.5	3-74
Congenital hypothyroidism	138[a]	20[a]	2-136[a]
Cystic fibrosis	13	15	7-31
Fatty acid oxidation disorders	4	13.5	3-26
Galactosemia	-	-	-
Hemoglobinopathies	8	10.5	3-18
Organic acid disorders	2	124.5	33-216
Mucopolysaccharidosis type I	-	-	-
Pompe	-	-	-
Severe combined immunodeficiency	2	25.5	1-50
Spinal muscular atrophy	3	21	9-32
X-linked adrenoleukodystrophy	11	48	25-115
Total	**197**		**1-216**

[a] includes both mild and severe CH cases

WASHINGTON STATE BOARD OF HEALTH

Date: March 13, 2024

To: Washington State Board of Health Members

From: Kate Dean, Environmental Health Subcommittee Chair

Subject: Request for Delegated Rulemaking Authority – WAC 246-272A-0110, Table I, Category 2.

Background and Summary:

The Department of Health (Department) is requesting the delegation of rulemaking authority from the State Board of Health (Board) to update the small on-site sewage system rule regarding eligibility requirements for proprietary treatment products, WAC 246-272A-0110, Table I, Category 2, to align with industry and subject matter expert recommendations.

RCW 43.20.050(3) authorizes the Board to adopt rules concerning on-site sewage systems (OSS) with design flows less than three thousand five hundred gallons a day. WAC 246-272A-0110, Table I, Category 2 lists the core testing requirements for Category 2 proprietary treatment products for small on-site systems.

At the January 2024 Board meeting, the Board adopted the permanent rule for chapter 246-272A WAC. The permanent rule identifies EPA Method 1664 as the testing protocol for Category 2 treatment products. The protocol tests the ability of the product to treat oil and grease but does not include a test for organic sewage strength and suspended solids.

The Board may delegate any of its rulemaking authority to the Department under RCW 43.20.050(4). Board Policy number 2000-001 further outlines conditions and circumstances for “Considering Delegation of Rules to Department of Health.” Delegated rulemaking authority would allow the Department to revise WAC 246-272A-0110, Table I, Category 2 to incorporate the necessary testing requirements.

Joining us today from the Department’s Office of Wastewater Management is Roger Parker, the OSS Technical Assistance Lead. He will discuss the Department’s request for delegated rulemaking authority.

(continued on the next page)

Recommended Board Actions:

The Board may wish to consider, amend if necessary, and adopt one of the following motions:

The Board delegates to the Washington Department of Health rulemaking authority to amend WAC 246-272A-0110, Table I, Category 2 to incorporate the necessary testing requirements for Category 2 treatment products.

Or

The Board denies the Department's request to delegate rulemaking authority to amend WAC 246-272A-0110, Table I, Category 2.

Staff

Andrew Kamali

Board Authority

RCW 43.20.050

Powers and duties of state board of health—Rule making—Delegation of authority—Enforcement of rules.

(1) The state board of health shall provide a forum for the development of public health policy in Washington state. It is authorized to recommend to the secretary means for obtaining appropriate citizen and professional involvement in all public health policy formulation and other matters related to the powers and duties of the department. It is further empowered to hold hearings and explore ways to improve the health status of the citizenry.

In fulfilling its responsibilities under this subsection, the state board may create ad hoc committees or other such committees of limited duration as necessary.

(2) In order to protect public health, the state board of health shall:

(a) Adopt rules for group A public water systems, as defined in RCW 70A.125.010, necessary to assure safe and reliable public drinking water and to protect the public health. Such rules shall establish requirements regarding:

(i) The design and construction of public water system facilities, including proper sizing of pipes and storage for the number and type of customers;

(ii) Drinking water quality standards, monitoring requirements, and laboratory certification requirements;

(iii) Public water system management and reporting requirements;

(iv) Public water system planning and emergency response requirements;

(v) Public water system operation and maintenance requirements;

(vi) Water quality, reliability, and management of existing but inadequate public water systems; and

(vii) Quality standards for the source or supply, or both source and supply, of water for bottled water plants;

(b) Adopt rules as necessary for group B public water systems, as defined in RCW 70A.125.010. The rules shall, at a minimum, establish requirements regarding the initial design and construction of a public water system. The state board of health rules may waive some or all requirements for group B public water systems with fewer than five connections;

(c) Adopt rules and standards for prevention, control, and abatement of health hazards and nuisances related to the disposal of human and animal excreta and animal remains;

(d) Adopt rules controlling public health related to environmental conditions including but not limited to heating, lighting, ventilation, sanitary facilities, and cleanliness in public facilities including but not limited to food service establishments, schools, recreational facilities, and transient accommodations;

(e) Adopt rules for the imposition and use of isolation and quarantine;

(f) Adopt rules for the prevention and control of infectious and noninfectious diseases, including food and vector borne illness, and rules governing the receipt and conveyance of remains of deceased persons, and such other sanitary matters as may best be controlled by universal rule; and

(g) Adopt rules for accessing existing databases for the purposes of performing health related research.

(3) The state board shall adopt rules for the design, construction, installation, operation, and maintenance of those on-site sewage systems with design flows of less than three thousand five hundred gallons per day.

(4) The state board may delegate any of its rule-adopting authority to the secretary and rescind such delegated authority.

(5) All local boards of health, health authorities and officials, officers of state institutions, police officers, sheriffs, constables, and all other officers and employees of the state, or any county, city, or township thereof, shall enforce all rules adopted by the state board of health. In the event of failure or refusal on the part of any member of such boards or any other official or person mentioned in this section to so act, he or she shall be subject to a fine of not less than fifty dollars, upon first conviction, and not less than one hundred dollars upon second conviction.

(6) The state board may advise the secretary on health policy issues pertaining to the department of health and the state.

[**2021 c 65 § 37**; **2011 c 27 § 1**; **2009 c 495 § 1**; **2007 c 343 § 11**; **1993 c 492 § 489**; **1992 c 34 § 4**. Prior: **1989 1st ex.s. c 9 § 210**; **1989 c 207 § 1**; **1985 c 213 § 1**; **1979 c 141 § 49**; **1967 ex.s. c 102 § 9**; **1965 c 8 § 43.20.050**; prior: (i) **1901 c 116 § 1**; **1891 c 98 § 2**; RRS § 6001. (ii) **1921 c 7 § 58**; RRS § 10816.]

Washington State Board of Health
Policy & Procedure

Policy Number:	**2000-001**
Subject:	**Considering Delegation of Rules to Department of Health**
Approved Date:	**November 8, 2000 (Revised August 13, 2014)**

Policy Statement

In some instances, the Washington State Board of Health may determine it is appropriate to delegate its authority for rulemaking to the Department of Health (RCW 43.20.050). The Board and the Department recognize the need to balance both broad constituent participation and administrative efficiency when making decisions about any rule delegation. For this reason, the Board and the Department have agreed on certain policy considerations to assist Board members in their decisions related to rule delegation.

The Board's decision to delegate a specific rule will be made on a case-by-case basis. The Board will determine the breadth of the delegation, which may range from specific aspects of a single rule section to a broader body of regulatory authority, such as an entire chapter of rules. Each Board delegation is for a single rulemaking process unless specified in an approved motion to be a continuing delegation until rescinded. Once a rule has been delegated, the Department will keep the Board informed about the rule making process through periodic progress reports. The Board may rescind its delegation at any time.

When considering delegation of authority to modify or adopt a rule, the Board may consider the following:

- The extent to which the proposed rule revision is expected to include editorial and/or grammatical changes that do not change the substance of the rule;
- The extent to which the proposed rule seeks to adopt federal requirements in which the state has little or no discretion;
- The extent to which the substance and direction of the proposed rule is expected to have broad public and professional consensus;
- The extent to which the proposed rule may make significant changes to a policy or regulatory program; and

- The extent to which the rule revision process would benefit from the Board's role as a convener of interested parties.

Procedure

When the Board receives a request from the Department to delegate authority for rulemaking, the Executive Director will review the request compared with the above policy considerations. The Executive Director will prepare or direct staff to prepare a recommendation for the Board to consider at its next most convenient meeting. The Executive Director will consult with the Board Chair and members of any appropriate policy committee to formulate the recommendation. The Board will then act on the request, which may include delegating authority to the Department as requested or otherwise specifying the rulemaking authority it delegates.

If the Board is not scheduled to meet again within two months and the Department justifies a pressing need to begin rulemaking, the Board's Chair may call a special meeting of the Board to consider the request. The Executive Director will send the request for delegation to all Board members prior to the meeting.

STATE OF WASHINGTON

DEPARTMENT OF HEALTH

DIVISION OF ENVIRONMENTAL PUBLIC HEALTH

PO Box 47820 ○ Olympia, Washington 98504-7820

(360) 236-3000 ○ TTY Relay Service: (800) 833-6388

March 13, 2024

TO: Michelle Davis, Executive Director
Washington State Board of Health

FROM: Lauren Jenks, Assistant Secretary
Division of Environmental Public Health

SUBJECT: State Board of Health Rule Making Authority Delegation Request-
WAC 246-272A-0110, Proprietary treatment products, Onsite Sewage Systems

The Department of Health (department) is requesting delegation of rule-making authority from the State Board of Health (board) to change product testing requirements in WAC 246-272A-0110, Table I, Category 2.

Changes to the rule under this delegation request, if approved, will be limited to adding NSF/ANSI 40 testing to category 2.

Category 1 products treat residential-strength sewage from homes. The rule requires that Category 1 products are tested under NSF/ANSI 40 -Residential Wastewater Treatment Systems (versions dated between January 2009 and May 31, 2021). This protocol tests the product's capacity to treat organic sewage strength ($CBOD_5$) and suspended solids (TSS).

Category 2 products treat high-strength sewage from restaurants and other facilities that generate high levels of oil and grease. Prior to the recent rule revision, the rule required testing for Category 2 products under the EPA/NSF Protocol for the Verification of Wastewater Treatment Technologies/EPA Environmental Technology Verification (April 2001). This protocol tested for organic sewage strength ($CBOD_5$) and suspended solids (TSS), as well as oil and grease. EPA archived this testing protocol in 2013. Most laboratories have stopped testing for it. This has created a barrier for manufacturers to register new Category 2 products. None have been registered for several years.

During the recent rule revision, the department recommended updating the Category 2 product testing requirements to EPA Method 1664, Revision B (February 2010). This method tests the product's capacity to treat oil and grease. This recommendation, however, neglected to assure that Category 2 products have also been tested for organic sewage strength ($CBOD_5$) and suspended solids (TSS). A manufacturer provided formal comment highlighting this oversight and recommending Category 2 products instead be tested with NSF/ANSI 40 -Residential Wastewater Treatment Systems (versions dated between January 2009 and May 31, 2021).

The department has determined that Category 2 products should be tested by both EPA Method 1664, Revision B (February 2010) and NSF/ANSI 40 -Residential Wastewater Treatment Systems (versions dated between January 2009 and May 31, 2021). This will ensure the product is tested for organic sewage strength ($CBOD_5$), suspended solids (TSS), and oil and grease.

The department believes that most existing products that manufacturers are interested in registering as Category 2 products have already undergone testing under NSF/ANSI 40 -Residential Wastewater Treatment Systems (versions dated between January 2009 and May 31, 2021). Likewise, manufacturers are expected to test most new products under NSF/ANSI 40 Residential Wastewater Treatment Systems (versions dated between January 2009 and May 31, 2021). Therefore, adding it as a testing requirement is not expected to impact most manufacturers.

The department anticipates completing this rule making by December 31, 2024.

Conformance with the State Board of Health Delegation Criteria:

The board's policy (Policy Number 2000-001) for Considering Delegation of Rule to the Department of Health provides the following elements for consideration*:*

The extent to which the proposed rule revision is expected to include editorial and/or grammatical changes that do not change the substance of the rule:

- The department does not anticipate editorial or grammatical changes to the rule.

The extent to which the proposed rule may make significant changes to a policy or regulatory program.

- The proposed rule revision would add a testing requirement for Category 2 products, which treat high-strength sewage.

The extent to which the rule revision process would benefit from the board's role as a convener of interested parties.

- The department does not anticipate any controversy or opposition to the rule change because most manufacturers already test their products under NSF/ANSI 40 -Residential Wastewater Treatment Systems (versions dated between January 2009 and May 31, 2021).
- The department will keep interested parties informed through email updates sent to everyone that has signed up for our rule updates and all known manufacturers.

For additional information, please contact Jeremy Simmons at 360.236.3346 or jeremy.simmons@doh.wa.gov.

Washington State Department of HEALTH

Request for Rulemaking Authority Delegation: WAC 246-272A-0110, Table I, Category 2

State Board of Health Meeting

March 13, 2024

Presenter

Roger Parker

OSS Technical Assistance Lead

Wastewater Management Section

Division of Environmental Public Health

Office of Environmental Health and Safety

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www.doh.wa.gov

@WADeptHealth

Scope of Rulemaking - Potential Change

Testing Requirement for Proprietary Treatment Products – WAC 246-272A-0110, Table I	
Treatment Component/Sequence Category	Required Testing Protocol
Category 2 Designed to treat effluent anticipated to be greater than treatment level E. (Such as at restaurants, grocery stores, mini-marts, group homes, medical clinics, residences, etc.)	EPA Method 1664, Revision B (February 2010), and NSF/ANSI 40—Residential Wastewater Treatment Systems (versions dated between January 2009 and May 31, 2021)

Testing Protocols/Method Requirements

- EPA/NSF Protocol for the Verification of Wastewater Treatment Technologies/ EPA Environmental Technology Verification (April 2001) –**ARCHIVED**
  - Organic Sewage Strength ($CBOD_5$)
  - Suspended Solids (TSS)
  - Oil and Grease
- EPA Method 1664, Revision B (February 2010)
  - Oil and Grease
- NSF/ANSI 40—Residential Wastewater Treatment Systems (January 2009 - May 31, 2021)
  - Organic Sewage Strength ($CBOD_5$)
  - Suspended Solids (TSS)

SBOH Delegation Considerations

- Most manufacturers already have tested, or plan to test, their products under NSF/ANSI 40—Residential Wastewater Treatment Systems (versions dated between January 2009 and May 31, 2021).
- Few manufacturers are interested in registering products under Category 2.
- Adding it as a testing requirement for Category 2 products is expected to impact few, if any, manufacturers.
- No expected controversy or opposition from interested parties.
- The department will notify all interested parties by email, provide the proposed rule language to interested parties, and post information about the rulemaking on the department's rulemaking webpage.

To request this document in another format, call 1-800-525-0127. Deaf or hard of hearing customers, please call 711 (Washington Relay) or email civil.rights@doh.wa.gov.

WASHINGTON STATE BOARD OF HEALTH

Swinomish Indian Tribal Community (SITC)

Jennifer La Pointe,
SITC General Manager and Tribal community member

didgʷálič wellness center

Beverly Keyes, DNP, RN

Beverly Keyes, DNP, RN, is the Chief Executive Officer of didgʷálič Wellness Center.

Dr. Keyes held Senior leadership roles in Washington state at rural and urban hospitals and Skagit Valley College. She has been an active member of Skagit County serving on many boards including the Population Health Trust Advisory Committee. Dr. Keyes held a governor-appointed position on the Skagit EMS Executive Board. She also served as a La Conner Council Member and Planning Commissioner where she developed working relationships with the Swinomish Tribe.

In 2019, Dr. Keyes came to the didgʷálič Wellness Center as a primary care consultant, transitioned to practice administrator, and in 2021 became CEO.

Dr. Keyes is honored to serve the Swinomish Tribal Indian Community.

SVC | Skagit Valley College

dəxʷx̌ayəbus-Dental Therapy Program

Dr. Cheyanne E. Warren, Program Director

Dr. Cheyanne Warren is the Dental Therapy Program Director. She was born and raised in Vermont and completed all of her undergraduate and post-graduate education in Virginia. She holds a Bachelor of Science degree from James Madison University (JMU), a Master's in Biochemistry from Virginia Commonwealth University (VCU), followed by her dental degree from VCU. After completing her Advanced Education in General Dentistry (AEGD) residency at VCU, she and her husband returned to Vermont to begin practicing general dentistry.

Dr. Warren began her clinical career at a community health center in Plainfield, Vermont where she had the opportunity to supervise and mentor General Practice Residency (GPR) students. Simultaneously, she began supervising dental hygiene students at Vermont Technical College (the only dental education program in the state of Vermont) as they performed clinical procedures.

In May of 2017, Dr. Warren transitioned from practicing general dentistry and became the Dental Therapy Program Director at Vermont Technical College (VTC). In her capacity as the Dental Therapy Program Director at VTC, she was tasked with creating a CODA accredited hygiene-based dental therapy curriculum for the State. While at VTC, Dr. Warren began supervising dental externs from the University of New England School of Dentistry to prepare for the dental therapy clinical education, expand community access to care, and increased the interprofessional collaboration between all oral health providers.

Dr. Warren values community service and has volunteered in dental service projects including Mission of Mercy in Virginia, provided care at a free dental clinic in Charlottesville, VA and Internationally with Hands for Honduras before the pandemic shut down in 2020. She also served on the Vermont Oral Health Collation, is currently on the executive committee of the National Collation of Dentists for Health Equity and is passionate about increasing quality oral health access. Her research background has solidified a firm belief that sound evidence should dictate oral health practice across all providers.

Dr. Warren and her family live in Bellingham and enjoy being a part of the incredible community that surrounds Skagit Valley. In her spare time, you will find her exploring the great outdoors while running, skiing, hiking, biking and fly fishing. Dr. Warren is committed to ensuring our students are prepared to provide exceptional oral healthcare in their communities and further develop this incredible educational model in order to expand access to oral health further then we thought possible.

SWINOMISH DENTAL

Dr. Rachael Hogan, Dental Director

Dr. Rachael Hogan is a general dentist and the Dental Director of the Swinomish Tribal Dental Clinic in LaConner, WA where she helps lead the effort to address the oral health crisis in Indian County and supervised the first Alaska trained dental therapist in the Lower 48 States. Dr. Hogan is a strong advocate for well-rounded dental teams utilizing primary oral health providers and allowing all staff to work at the top of their scope. She recognizes the importance of holistic care delivered with cultural humility, evidence based clinical excellence and diversifying the dental profession. Dr. Hogan is also the acting director of dəxʷx̌ayəbus, a developing Dental Therapy Education Program at Skagit Valley College in Western Washington which was initiated by the Swinomish Tribe.

Dr. Hogan completed her dental education at Marquette School of Dentistry in 2002. After graduation, she moved closer to her Alaska roots to settle and fulfill a National Health Service Corp obligation in Bellingham, Washington. Prior to being recruited to Swinomish, Dr. Hogan worked for more than 10 years for Sea Mar, one of the largest non-profit community health clinics in the Pacific Northwest. During that time, her passion for access to care issues introduced her to the ADA's Diversity in Leadership Program, the Steering Committee of the Whatcom County Oral Health Coalition and she was the Membership Chair for the Mount Baker District Dental Society. Dr. Hogan has volunteered at Migrant Camps, Smile Mobile and Project Homeless Connect. She piloted programs such as the Placksmakin' Preschoolers, a Volunteer Hygiene Program and a symposium on Prenatal Oral Health. She encourages dental students to consider public health dentistry as a career and mentors externs yearly. When Dr. H is not in the office she is chasing her four active kids and rock star husband.

Date: March 13, 2024

To: Washington State Board of Health Members

From: Paj Nandi, Board Member

Subject: Pro-Equity Anti-Racism Plan and Playbook

Background and Summary:

The COVID-19 pandemic highlighted disparities that impact Washington State communities in different ways, often leading to inequitable outcomes. The Washington State Pro-Equity Anti-Racism Plan and Playbook, also known as the PEAR Plan and Playbook, is a way to keep Washington a great place to live, learn, work, play, and stay. Some Washingtonians have questioned the legitimacy of State Government due to decisions made without them. Others question the government's effectiveness because it is not delivering services that meet their needs. Some do not trust state government because of its history of oppression and marginalization.

The Governor's Executive Order 22-04 implements the Washington State Pro-Equity Anti-Racism Plan and Playbook. It requires that all state agencies, including boards and commissions, implement a PEAR Plan to bridge opportunity gaps. The PEAR Plan and Playbook is an approach that drives systemic change, aims to dismantle oppressive systems, and promotes equity across all of society. The PEAR Plan states that agencies will bridge opportunity gaps by reducing disparities, including racial and ethnic disparities, statewide and across state government.

The Washington State Office of Equity was tasked with the creation of the PEAR Plan and Playbook. The Office of Equity is also tasked with gathering data from each state agency on the effectiveness of the PEAR Plan. They will also provide technical support in the creation of a plan. Every September, state agencies must provide data to the Office of Equity, as well as submit updated plans. This year, the Board will be completing their initial PEAR strategic plan.

The Board will need to create a PEAR strategic plan within our sphere of influence, capacity, and authority. The information provided will provide the Board with general background information on the PEAR Plan, requirements of the PEAR Plan, and guidance on how to complete a PEAR strategic plan. The Board will also learn about the work that has been on-going to support pro-equity efforts. This is an opportunity for the Board to begin discussion on PEAR strategies that can reduce disparities and

(Continued on the next page)

bridge gaps with communities. This is an informational briefing and requires no formal action by the Board.

Staff

Ashley Bell

To request this document in an alternate format or a different language, please contact the Washington State Board of Health, at 360-236-4110 or by email at wsboh@sboh.wa.gov TTY users can dial 711.

PO Box 47990 • Olympia, WA 98504-7990
360-236-4110 • wsboh@sboh.wa.gov • sboh.wa.gov

STATE OF WASHINGTON
OFFICE OF GOVERNOR JAY INSLEE

EXECUTIVE ORDER 22-02

ACHIEVING EQUITY IN WASHINGTON STATE GOVERNMENT

WHEREAS, achieving equal opportunity is foundational to the story of America, and each of us bears the responsibility to stand up and keep this unalienable right in reach for all Washingtonians. Each person in this state deserves a fair chance to live life to the fullest, regardless of race, ethnicity, creed, color, national origin, citizenship or immigration status, sex, honorably discharged veteran or military status, sexual orientation, or the presence of sensory, mental, or physical disability; and

WHEREAS, in 1998, Washington state voters passed Initiative 200 (I-200), now codified as RCW 49.60.400, which reads that "The state shall not discriminate against, or grant preferential treatment to, any individual or group on the basis of race, sex, color, ethnicity, or national origin in the operation of public employment, public education, or public contracting."; and

WHEREAS, in response to the passage of I-200, the then-sitting governor issued Directive 98-01 to state agencies to implement the initiative. Subsequent court decisions and legal guidance have clarified the scope of options available to state agencies to address evident discrimination; and

WHEREAS, Washington is a state of great beauty with an abundance of opportunities, resources, and a growing population that has become increasingly ethnically and racially diverse over the last several decades. Within this beautiful landscape, too many Washingtonians face systemic barriers, discrimination, and inequities that are deep, pervasive, persistent, and prevent them from flourishing and achieving their full potential; and

WHEREAS, state government recognizes and embraces its responsibility to dismantle discrimination and institutional and systemic barriers to fulfill its public service mandate to ensure that all people have full access to opportunities to flourish and live healthy, successful lives. In recent years, Washington state government has taken the following actions:

- Public Contracting – the Subcabinet on Business Diversity was formed in 2015 to improve opportunities for certified firms to contract with Washington state government and directed the Department of Enterprise Services (DES) to conduct a statewide disparity study. In 2019, the findings of the study supported the conclusion that people of color and women do not enjoy equal access to all aspects of state contracting opportunities. There was compelling evidence that the state should remedy the disparities and discrimination happening within state public contracting.

The Subcabinet on Business Diversity has begun to implement many of the recommendations from the disparity study, including but not limited to: a) the development and implementation of an electronic data collection and monitoring system, b) examination of current best practices, c) outreach to state agencies, certified businesses, and diverse-owned businesses that are in industries with low minority utilization by the state, d) improving technical assistance to businesses and agencies, and e) increasing direct buy limits to $40,000 for small and certified firms.

- Public Employment – In 2020, State Human Resources (SHR) Directives 20-02 and 20-03 were issued to all executive branch agencies. SHR Directive 20-02 requires all impacted agencies to: a) update or create diversity, equity, and inclusion (DEI) plans and procedures, b) train recruitment staff on mitigating bias in the job application process, c) set workforce diversity goals, d) conduct regular reviews of agency diversity data by leadership, e) develop pathways and connections with higher education, and f) review the diversity of candidate pools for past job opportunities. SHR Directive 20-03 requires all impacted agencies to create policies for: a) diversity, equity, and inclusion, b) respectful work environment, c) anti-discrimination, harassment, and sexual harassment, and d) reasonable accommodation.

- Equity Office – The Office of Equity was established to: (1) promote access to equitable opportunities and resources that reduce disparities and improve outcomes statewide across state government per its authority in RCW 43.06D.020, (2) support state agencies in our commitment to be an anti-racist government system, (3) serve as a tool to root out racism and other forms of discrimination in state government, and (4) publish and report the effectiveness of agency programs on reducing disparities using input from the communities served by those program.

  The Equity Office is partnering with the public workforce and communities to develop the state's comprehensive equity strategic plan and outcome measures designed to bridge opportunity gaps and reduce disparities.

WHEREAS, in December 2020, I declared that Washington will be an anti-racist state, and committed to take actions that hold our state to that commitment. I proposed an historic equity package of policies and funding that reflects our dedication toward disrupting the harmful systemic cycle of racism and inequity.

NOW, THEREFORE, I, Jay Inslee, Governor of the state of Washington, by virtue of the power vested in me by the Constitution and statutes of the state of Washington, do hereby rescind Directive 98-01 immediately and direct as follows:

1. Public Contracting – As the state agency responsible under chapter 39.19 RCW for developing programs to maximize opportunities for minority- and women-owned businesses in public contracting and procurement, the Office of Minority and Women's Business Enterprises (OMWBE) is charged with the implementation of Executive Order (EO) 22-01. EO 22-01 requires all executive and small cabinet agencies to use the newly developed Tools for Equity in Public Spending. OMWBE will continue to be the lead

agency responsible for implementing the Roadmap to Contracting Equity that was developed in response to the 2019 Statewide Disparity Study.

2. Public Employment – All executive and small cabinet agencies will continue to follow SHR Directives 20-02 and 20-03. The Director of SHR will consult with the Office of Equity to deliver a report to me that reviews and evaluates each agency response to SHR Directives 20-02 and 20-03.

   SHR will proactively address and dismantle oppressive systems and practices in the workplace and build new, equitable systems to achieve a workforce that is representative of the diversity of Washington and practices cultural humility. SHR will deliver to me a strategy to accomplish these objectives by October 2022.

   SHR is further directed to: 1) in consultation with the Governor's Committee on Disability Issues & Employment, review and recommend any updates to EO 13-02 to improve employment opportunities for individuals with disabilities with the State of Washington; and 2) issue a directive to require all cabinet agency employees to complete DES's DEI training.

3. Public Education – The Washington Student Achievement Council is directed to prepare a report describing the differences in patterns of access and success across student subpopulations, the faculty and staff equity demographics at public educational institutions, and the scope and progress of existing programs designed to identify and remedy discrimination in our higher education system. The report will also describe gaps in these programs and additional recommended actions.

   I will also solicit the views of the Superintendent of Public Instruction as to any additional steps needed to identify and address discrimination in our K-12 school system.

4. Public Services – All executive and small cabinet agencies shall identify ways to bolster access to state services by reducing barriers and eliminating inequities in all aspects of agency decision making, including but not limited to, service delivery, program development, policy development, staffing, and budgeting.

The rescission of Directive 98-01 does not alter other state and federal legal requirements applicable to affirmative action measures. As agencies implement this Executive Order, they are directed to consult with the Office of the Attorney General, SHR, and the Office of Equity.

I will convene a cabinet-wide and community summit in October 2022, to report on the state strategy and agency plans, and discuss the progress on implementing this Executive Order.

I recognize the traumatic and long-lasting impacts of discrimination, racism, and oppression. I also recognize that Washington state government has the responsibility and the ability to make a difference for all of us—employees, the people served, and current and future generations of Washingtonians. This order, alone, will not create equity in our state, but this is a necessary next step.

I invite other statewide elected officials, institutions of higher education, agencies of the judiciary, agencies of the Legislature, and boards and commissions to follow the provisions of this Executive Order.

This Order is effective immediately.

Signed and sealed with the official seal of the state of Washington on this 17th day of January, A.D., Two Thousand and Twenty-Two at Olympia, Washington.

By:

/s/
Jay Inslee, Governor

BY THE GOVERNOR:

/s/
Secretary of State

STATE OF WASHINGTON
OFFICE OF GOVERNOR JAY INSLEE

EXECUTIVE ORDER 22-04

IMPLEMENTING THE WASHINGTON STATE PRO-EQUITY ANTI-RACISM (PEAR) PLAN & PLAYBOOK

WHEREAS, the Legislature and I created the Washington State Office of Equity ("Office of Equity") in April 2020 to: (1) promote access to equitable opportunities and resources that reduce disparities and improve outcomes statewide across state government consistent with RCW 43.06D.020; (2) support executive branch state agencies and executive branch boards and commissions ("state agencies") in our commitment to be an anti-racist government system; (3) partner with state employees and communities to develop the state's comprehensive equity strategic plan and outcome measures designed to bridge opportunity gaps and reduce disparities; and (4) publish and report the effectiveness of agency programs on reducing disparities using input from the communities served by those programs; and

WHEREAS, in December 2020, I declared that Washington will be an anti-racist state and committed to take actions that hold our state to that commitment. Washington is a state where all are welcomed and will have the opportunity to thrive regardless of race, ethnicity, creed, color, national origin, citizenship or immigration status, sex, honorably discharged veteran or military status, sexual orientation, or the presence of sensory, mental, or physical disability; and

WHEREAS, determinants of equity are the driving factors that impact the overall quality of life for all Washingtonians. King County established the following 14 determinants of equity: economic justice, state and local practices, jobs and job training, justice systems and laws, health and human services, food systems, environment and natural resources, community and public service, transportation and mobility, community and economic development, and housing and home ownership, early childhood development, and education. I agree that these are appropriate determinants of equity and would also add digital access and literacy. By adding digital access and literacy, which is an issue creating additional divides and gaps between Washingtonians, the state has identified 15 determinants of equity. Eliminating disparities in terms of access, practices and procedures, quality of services, and programs in these 15 determinants of equity correlate to better outcomes for people and a Washington where all can thrive; and

WHEREAS, the Office of Equity gathered the collective wisdom of thousands of community members, state employees, board and commission members, state employees, a host of partners across many sectors, and members of all branches of state government to co-create the state's inaugural five-year Washington State Pro-Equity Anti-Racism (PEAR) Plan & Playbook ("PEAR Plan & Playbook"), Washington's approach for achieving pro-equity and social justice across state government. The PEAR Plan & Playbook is designed to bridge opportunity gaps and reduces disparities so everyone in Washington flourishes and achieves their full potential; and

WHEREAS, the PEAR Plan & Playbook establishes a unified vision of equity for state government, mission, values, and goals, and contains a step-by-step playbook for developing, implementing, and embedding PEAR into every government action across state government**.** It reflects both how we do our daily work and who we are at our core – public servants with a shared desire for promoting equity, justice, access, and belonging for the people we serve and our colleagues who serve them; and

WHEREAS, the PEAR Equity Impact Review (EIR) framework describes a five-step process that blends numerical data and descriptive, community narrative data to inform agency planning, decision-making, and implementation of actions that achieve equitable access to opportunities and resources that reduce disparities and improve equitable outcomes statewide. Conducting an EIR is necessary prior to proposing changes to agency policies, programs, and practices. Our people and environment are both healthy and flourish when we work together with those experiencing inequities to ensure that everyone employed or served by state government is treated with fairness, dignity, honor, and respect; and

WHEREAS, the PEAR framework identifies priority investment in the 15 determinants of equity to achieve outcomes that benefit all tribes, communities, and employees of Washington's ecosystem, and calls for investing more of our state's resources "upstream" to address root causes where the needs are greatest to ensure that individuals in underserved communities have their basic needs met long term in Washington's ecosystem; and

NOW, THEREFORE, I, Jay Inslee, Governor of the state of Washington, by virtue of the power vested in me by the Constitution and statutes of the state of Washington, do hereby order and direct as follows:

1. The Washington State Office of Equity (Office of Equity) is charged with the implementation of Executive Order (EO) 22-04. The Office of Equity will be required to:

    a. Communicate the PEAR Plan & Playbook to state agencies in an effective and accessible way.
    b. Provide templates, toolkits, consultation, guidance, technical assistance, and training necessary for state agencies to develop, implement, and measure the effectiveness of their pro-equity, racial justice, access, and belonging strategic action plans. This support will include:
        - Developing a form (format, content, and frequency) that will serve as each agency's strategic action plan.
        - Creating statewide and agency-specific process and outcome measures to show performance, using outcome-based methodology to determine the effectiveness of agency programs and services on reducing disparities.
        - Convening a team of employees and communities to determine whether the performance measures established accurately measure the effectiveness of agency programs and services in the communities served.
        - Creating an online dashboard to publish statewide and agency-specific plans, performance measures, and outcomes.
        - Establishing a process to report on each agency's performance and a process for each agency to respond.

c. Establish- procedures to hold agencies accountable, which may include conducting performance reviews related to agency compliance with Office of Equity performance measures.
d. Convene a team of employees and communities impacted by state programs and services to develop and publish a report for each agency detailing whether the agency has met the performance measures established and the effectiveness of agency programs and services on reducing disparities, including the agency's strengths and accomplishments, areas for continued improvement, and areas for corrective action.
e. Post statewide and agency-specific plans performance measures and outcomes and Equity Office agency performance review reports on the dashboard, by September 30, 2023, and every year thereafter.
f. Beginning in 2022, develop and submit an annual report to the Legislature and me by October 31, detailing an overview of agency compliance with the Office of Equity's standards and performance measures per RCW 43.06D.040(1)(e)(2).
g. Fulfill all other duties consistent RCW 43.06D.040.

2. All state agencies are charged with the implementation of Executive Order (EO) 22-04. The agency leader is responsible and accountable for achieving agency PEAR outcomes, and these duties include but are not limited to:

a. Developing, implementing, and reporting on progress of the PEAR Strategic Action Plan.
b. Gathering data, helping to improve communications, and updating (or recommending, where required) policies, and educating employees about ways to create a PEAR culture.
c. Establishing and delegating authority to the PEAR Team, reporting directly to executive leadership, comprised of agency executive leaders, the agency equity officer, employees, and external customers, partners, and experts for key business lines to assist the agency leader in achieving these goals.
d. Providing agency PEAR Team's contact information to the Office of Equity by **April 30, 2022**.
e. Partnering with individuals, groups, and communities impacted by agency programs or services to complete an initial EIR by **August 1, 2022**, to determine agency baseline.
f. Based on the results of the EIR, completing a PEAR Strategic Action Plan Template due to the Office of Equity by **September 1, 2022**; updated plans are due every year thereafter.
g. Implementing agency PEAR Strategic Action Plans, beginning **September 1, 2022**.
h. Preparing and submitting a PEAR Annual Performance Report to the Office of Equity by **September 1, 2023**, and every year thereafter.
i. Utilizing quarterly performance review process as best practice to monitor progress towards agency PEAR Strategic Action Plan goals.
j. Preparing and submitting a response to reports published by the Office of Equity on the agency's PEAR Strategic Action Plan performance. The agency's response must include the agency's progress on performance, the agency's action plan to address areas for improvement and corrective action, and a timeline for the action plan per RCW 43.06D.040(1)(e)(ii).
k. Providing executive-level support and resources needed to fulfill requirements under this Executive Order.
l. Requesting and receiving consultation, guidance, technical assistance, and training from the Office of Equity as needed to implement this Executive Order.

All state agencies will be evaluated under the framework set by the PEAR Plan & Playbook. I will hold all leaders of state agencies accountable for the effectiveness of your services and programs on reducing disparities, using input from the communities served by your organizations; however, as is true of all Executive Orders, nothing in this Order creates a private right of action. The Office of Equity will be resourced to develop and deliver technical assistance, consultation, and capacity-building services to assist you every step of the way.

I am excited to roll up my sleeves alongside you, today, to create a PEAR ecosystem in Washington state – one that bears fruits of peace, prosperity, and possibility for all, now and for generations to come.

I invite other statewide elected officials, institutions of higher education, agencies of the judiciary, agencies of the Legislature, and other boards and commissions to follow the provisions of this Executive Order.

This Order is effective immediately. Signed and sealed with the official seal of the state of Washington on this 21st day of March, AD, Two Thousand and Twenty-Two, at Olympia, Washington.

By:

/s/
Jay Inslee, Governor

BY THE GOVERNOR:

/s/
Secretary of State

PRO-EQUITY ANTI-RACISM PLANNING

Ashley Bell, MPA
Equity and Engagement Manager
Washington State Board of Health

WASHINGTON STATE BOARD OF HEALTH

Agenda

- Pro-Equity and Anti- Racism [PEAR]
- PEAR Plan Requirements
- Current SBOH Work
- PEAR Plan Timelines

PRO-EQUITY ANTI-RACISM

Introducing the PEAR Plan

WASHINGTON STATE
BOARD OF HEALTH

Executive Orders

- **Executive Order 22-02**
  - Achieving Equity in Washington State Government

- **Executive Order 22-04**
  - Implementing the Washington State Pro-Equity Anti-Racism (PEAR) Plan & Playbook
  - Office of Equity and state agencies are charged with implementation

What is PEAR?

- Pro-Equity Anti-Racism, or PEAR
- Drives systemic change, aiming to dismantle oppressive systems and promote equity in all facets of society
- Recognizes that systems of oppression are the upstream sources of all our inequities, and therefore, addressing these systems is crucial to creating a more equitable world

Why does PEAR exist?

- Some Washingtonians question the legitimacy of state government because decisions are consistently made without them
- Some question state government’s effectiveness because it is not delivering services that meet their needs
- Some do not trust state government because of its history of oppression and marginalization

What does PEAR do?

- Bridges opportunity gaps and reducing disparities statewide and across state government, to keep Washington a great place to live, learn, work, play, and stay
- Invest where the needs are the greatest to address upstream, root cause, issues that perpetuate systemic inequities
- Creates meaningful impact to the determinants of equity
    - Social, economic, geographic, political, and physical conditions that determine equity conditions

15 Determinants of Equity

Equity in State & Local Practices

Early Childhood Development

Quality Education

Parks, Recreation & Natural Resources

Healthy Built & Natural Environments

Transportation & Mobility

Equity in Jobs & Job Training

Health & Human Services

Food Systems

Economic Justice

Strong, Vibrant Neighborhoods

Housing & Home Ownership

Community & Public Safety

Equity in Justice Systems & Laws

Digital Equity[3]

How does PEAR work?

- Act on the 15 Determinants of Equity by focusing, creating a PEAR Strategic Action Plan
- Invest in intentional and meaningful change in our PEAR Service lines
- Embed equity into decision making, which include service delivery, program development, policy development, and budgeting

PEAR Service Lines

- **Leadership, Operations & Services**
- **Plans, Policies & Budgets**
- **Workforce Equity**
- **Tribal Government Relationships**
- **Public Communications & Education**
- Facilities & Systems Improvements
- **Policy Agenda**
- **Building a Racially Just Washington**
- Capacity Building
- Data & Strategy Reporting
- **Engagement & Community Partnerships**

PEAR Ecosystem Goals and Outcomes

- Reduce disparities in public contracting, public education, public employment, and public services
- Improve outcomes that benefit all tribes, communities, and employees of Washington’s PEAR ecosystem
- Enable all people in Washington to flourish and achieve their full potential, embody pro-equity anti-racism values, and enjoy peace, prosperity, and possibility now and for generations to come

Implement PEAR Framework

1. Implement a pro-equity, anti-racism framework in partnership with relevant communities and organizations
2. Embrace continuous learning, growing, and pivoting
3. Consistently assess your equity impact
4. Make values driven, data informed upstream investments
5. Be transparent, accountable, and operate with urgency

PEAR PLAN REQUIREMENTS

Engagement and Assessments

WASHINGTON STATE BOARD OF HEALTH

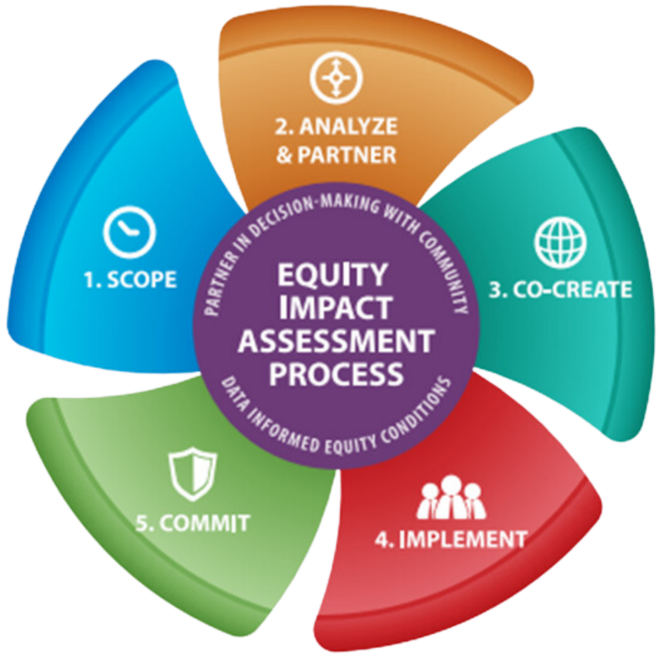
Develop a PEAR Team

- Executive Order 22-04 directs state agencies to create a PEAR team that consist of:
  - Board Sponsor, Executive Leaders, Diversity Professional, Staff, Community members, Partners, Experts for Business Lines
- The PEAR team is responsible for assisting the Board of Health achieve PEAR outcomes and the goals
- Without a strong team in place, there is a greater likelihood of gaps in the work

Conduct an Equity Impact Assessment

- Must be completed prior to creating a PEAR Strategic Action Plan

- The five-step equity impact assessment (EIA) process that uses
  - Numerical (quantitative) data
  - Community voices (qualitative) data



Develop and Implement PEAR Strategic Action Plan

- This plan is unique to the State Board of Health and is informed by our equity impact assessment
- The investments are guided by the determinants of equity and are designed to serve the impacted communities
- Currently, we have begun work in these areas:
  - Community Newsletter, Community Compensation, Equity Assessments, Language Justice, Meeting Accessibility and Accommodations, Scoping Document

Track and Report Performance

- Prepare and submit a PEAR Annual Performance Report to the Office of Equity each year to demonstrate performance
- Use outcome-based methodology to determine the effectiveness of agency programs and services on reducing disparities
- Receive and take into consideration community feedback on whether the performance measures established accurately measure the effectiveness of agency programs and services in the communities served
- Assess and refine our plan as needed based on our performance and community need

CURRENT SBOH WORK

Equity Projects

WASHINGTON STATE BOARD OF HEALTH

Community Compensation

- Removes barriers to co-creating policies with Community
- Promotes equitable policy development by establishing, sustaining, and growing relational partnerships
- Moves us from a transactional culture to a relational one
- Begins to create trust in the community

Community Engagement

- Focuses on finding and building relationships with Community
- Quarterly Community Newsletter
- Gift cards for participation that is not covered by community compensation
- Engagement scoping for rules work so that outreach is intentional and meaningful

Language Justice

- Interpreter services that use best practices
- Translation services
- Culturally and Linguistically Appropriate Standards
- Workgroup to explore strategies that promote language justice

Access for All

- Digital equity
- Meeting accessibility
- Reasonable accommodations review for participating in public meetings
- Plain Talk language

PROPOSED TIMELINES

Implementation Phases

WASHINGTON STATE BOARD OF HEALTH

Integration Phases

Phase 1: Compliance

- Establish PEAR Teams
- Conduct Assessment
- Develop PEAR Strategic Action Plan
- Annual Performance Report

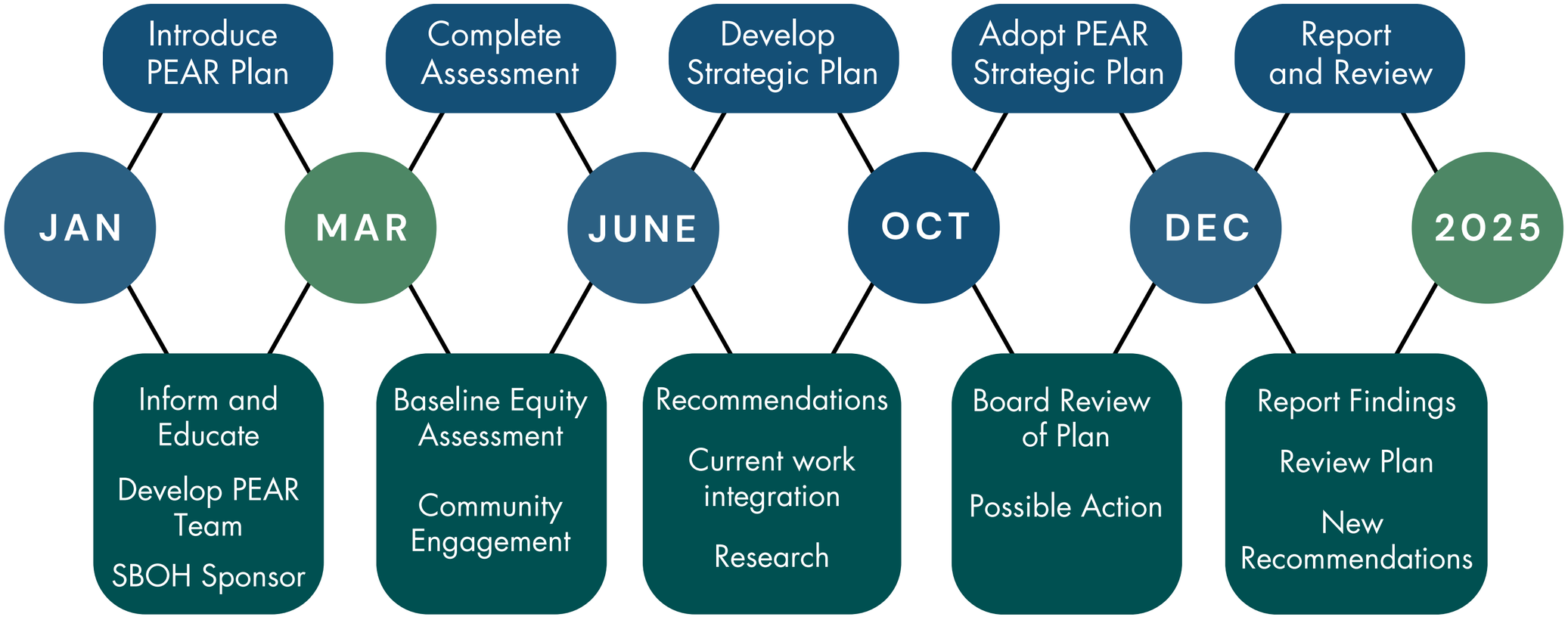
Phase 2: Transformation

- Embrace community partnership and center community voice

Phase 3: Accountability

- Respond to statewide and Board specific process and performance measures
- Foster continued growth and movement towards equity and justice for all

Proposed timeline



QUESTIONS OR COMMENTS?

Ashley Bell, Equity and Engagement Manager

WASHINGTON STATE BOARD OF HEALTH

ACCESSIBILITY AND THE AMERICANS WITH DISABILITIES ACT (ADA)

- The Washington State Board of Health (Board) is committed to providing information and services that are accessible to people with disabilities. We provide reasonable accommodations, and strive to make all our meetings, programs, and activities accessible to all persons, regardless of ability, in accordance with all relevant state and federal laws.
- Our agency, website, and online services follow the Americans with Disabilities (ADA) standards, Section 508 of the Rehabilitation Act of 1973, Washington State Policy 188, and Web Content Accessibility Guidelines (WCAG) 2.0, level AA. We regularly monitor for compliance and invite our users to submit a request if they need additional assistance or would like to notify us of issues to improve accessibility.
- We are committed to providing access to all individuals visiting our agency website, including persons with disabilities. If you cannot access content on our website because of a disability, have questions about content accessibility or would like to report problems accessing information on this website, please call (360) 236-4110 or email wsboh@sboh.wa.gov and describe the following details in your message:
    - The nature of the accessibility needs
    - The URL (web address) of the content you would like to access
    - Your contact information

We will make every effort to provide you the information requested and correct any compliance issues on our website.

WASHINGTON STATE BOARD OF HEALTH

EQUITY INITIATIVES

Around the State

WASHINGTON STATE BOARD OF HEALTH

Office of Equity

- Promote access to equitable opportunities and resources that reduce disparities and improve outcomes
- Support state agencies in our commitment to be an anti-racist government system
- Serve as a tool to root out racism and other forms of discrimination in state government
- Publish and report the effectiveness of agency programs on reducing disparities by using input from communities served by these programs

Equity in Public Contracting

- Executive Order 22-01
- Business diversity
  - Root cause analysis to determine participation of minority, women, and veteran-owned businesses in state contracting
- 2019 Washington State Disparity Study
  - Recommended several race-neutral remedial actions agencies could take to accomplish greater equity in state contracting activities
- Equity in Public Spending

Public Employment

- State Human Resources (SHR) Directive 20-02
  - Update or create diversity, equity, and inclusion plans and procedures
  - Train recruitment staff on mitigating bias in job application process
  - Set workforce diversity goals, conduct regular reviews of agency diversity data, and develop pathways and connections with higher education
  - Review the diversity of candidate pools for past job opportunities
- State Human Resources (SHR) Directive 20-03
  - Create policies for diversity, equity, and inclusion
  - Respectful work environment
  - Anti-discrimination, harassment, and sexual harassment
  - Reasonable Accommodations

Foundational DEI Training

- State Human Resources (SHR) Directive 23-01
- Must meet standards for employee diversity, equity, and inclusion (DEI) training and development
- Every state employee is required to take training grounded in statewide foundational competencies that promote diversity, equity, and inclusion to support workplace culture change and service delivery improvements
    - Who We Are: A Chronicle of Racism in America

Other State Directives

- Executive orders and directives related to workforce diversity, equity, and inclusion
  - Veterans
  - Persons with Disabilities in State Government
  - LGBTQ Inclusion and Safe Places
  - Tolerance, diversity and inclusiveness for Immigrants
  - Washington State Business Resource Groups

Department of Health Initiatives

- Workforce Pathways Program
  - Mentorship and Externship
  - Professional Development and EDI Training
  - Community Investments and Funding
- Equitable Rulemaking
- Community Collaborative
- Access
  - Cultural Appropriateness
  - Interpretation and Translations
- Office of Inclusion, Belonging and Well-being

Washington State

Pro-Equity Anti-Racism (PEAR) Plan & Playbook

2022–2027 Version 1.0

OFFICE OF EQUITY
Washington State
EQUITY and JUSTICE for ALL

Please email Access@equity.wa.gov to request communication or language services free of charge, such as interpreters, written information in other languages, or other formats (such as large print, Braille, audio, video, electronic) to access this document.

Please email your questions about the PEAR Plan & Playbook to PEAR@equity.wa.gov

Table of Contents

I. Acknowledgments

We acknowledge our ancestors, our elders, and those who began the quest for equity, social justice, access, and belonging for *all* people long before the state of Washington put forth this five-year **Washington State Pro-Equity Anti-Racism (PEAR) Ecosystem Plan & Playbook ("PEAR Plan & Playbook")**. We recognize and honor the commitments and sacrifices made by so many champions for equity in Washington state government, in our communities, in our region, across the nation, and worldwide.

Special thanks to Representative Mia Gregerson, Representative Melanie Morgan, and Senator Manka Dhingra for sponsoring and championing legislation that created the Office of Equity, located in the Office of the Governor, and to the Office of Equity Task Force for laying the foundation for this work: Governor's Interagency Council on Health Disparities Task Force Staff: LinhPhung Huynh, Project Manager Esmael López, Community Engagement Coordinator Hannah Fernald, Administrative Coordinator, Christy Curwick Hoff, former Manager, and Co-Chairs Benjamin Danielson (Co-chair) Jan Olmstead (Co-chair), RaShelle Davis (former Office of the Governor), Washington State Diversity, Equity & Inclusion Council, Sharon Ortiz and Laura Lindstrand (former Human Rights Commission), Elizabeth Gordon (Governor's Committee on Disability Issues and Employment) and Mandeep Kaundal (alternate), Lisa van der Lugt and alternates Rex Brown and Marika Barto (Office of Minority and Women's Business Enterprises), Maria Siguenza (Commission on Hispanic Affairs), Ed Prince (Commission on African American Affairs), Toshiko Hasegawa and alternate Carrie Huie Pascua (Commission on Asian Pacific American Affairs), Manny Santiago, Omar Santana, and Allison Spectator (LGBTQ Commission/Community), Michelle Gonzalez and Marie Vela (WA State Women's Commission), Craig Bill and alternate Mystique Hurtado (Governor's Office of Indian Affairs), Representative Mia Gregerson, Representative Melanie Morgan, Senator Manka Dhingra, Representative Jeremie Dufault and alternate Alec Regimbal.

We are forever grateful to all of you who invested your time, brilliance, and energy in developing models that Washington state could replicate, customize, and scale for use, especially Dr. Matias Valenzuela (former Director, Office of Equity and Social Justice in King County, Washington), Dr. Markisha Webster (former Director of the Office of Equity and Human Rights for the City of Portland), the Honorable Dr. Janice Underwood (former Chief Diversity Officer of the Commonwealth of Virginia, Office of the Governor), and the Government Alliance on Race and Equity (GARE) for leading as regional and national trailblazers for advancing equity and racial justice.

"We give thanks with a grateful heart."

To each of you who completed an equity readiness organizational baseline assessment, participated in listening sessions, shared your lived experiences with us, helped to finalize Executive Order 22-04, or spoke up during the Governor's Community and State Agency Roundtable, we hope you see and hear your voice in this PEAR Plan & Playbook. We are here because of you.

ADEFUA Cultural Education Workshop

Arts Commission

Asian Pacific Islander Coalition

Association of Washington Cities

Attorney General's Office: Kristin Lamson, Suzanne LiaBraaten

Leadership Team

Altavia Jones (OFM)

Ayanna Colman (ESD)

Blacks United In Leadership & Diversity (BUILD)

Black Education Strategy Round Table

Board of Accountancy

Career Connect Washington Statewide Team

Centro Latino

Charter School Commission

Childhood Obesity Prevention Coalition (COPC)

Children/Families Ombuds Stakeholder

Commissioner Cami Feek (ESD)

Conference of Minority Transportation Officials (COMTO)

Criminal Justice Training Commission

Daniya Baisubanova (Arts Commission)

Delta Sigma Theta Sorority, Inc. – Tacoma Alumnae Chapter

Denise Ross (Puget Sound Partnership)

Department of Corrections (DOC) Executive Strategy Team and Indeterminate Sentence Review Board (ISRB)

Department of Natural Resources

Deputy Directors Group 1: Jessica Todorovich, Heather Bartlett, Catherine Mele, Kendrick Stewart, David Puente, Jr., Wendi Gunther, Annette Meyer, Elizabeth Smith, Cami Feek (former Director), Annette Meyer, Cheryl Sullivan-Colglazier (former OFM employee)

Disability Inclusion Network Business Resource Group (DIN)

Diversity Center of Washington

Dr. Erica Hernandez Scott (PESB)

Carrie Basas (former Education Ombuds)

Education Ombuds Stakeholders

Educational Opportunity Gap Oversight and Accountability Committee

Emerald Cities

Energy Facility Site Evaluation Council

Environmental & Land Use Hearings Office (ELUHO)

Evette Jasper (former DCYF employee)

Front & Centered

Gender and Justice Commission

Gonzaga University

Good Shepherd Youth Outreach

Governor's Committee on Disability Issues & Employment

Governor's Executive Order 22-04 Roundtable Participants: Josephine Tamayo Murray, Yazmin Aguilar, Karen Vargas, Wendy Taliaferro, Faduma Ahmed, Nicole Franklin, john miller, Clinton Johnson, Ricardo Ibarra, Jan Olmstead, Dr. Ben Danielson, Cami Feek, Lisa Brown, Manny Santiago, Marcus Glasper, Russ Olsen

Governor's Executive Team

Governor's Office of Indian Affairs

Health and Human Services group

Helping Human Systems & The Athena Group

Information Technology Services Division (ITSD) of OFM

Interagency Committee of State Employed Women (ICSEW)

Interagency Protected Class Network

Jessica Zinda (DSHS)

Joana Ramos and the Washington State Coalition for Language Access (WASCLA)

Joanne Lee (DES)

Kitsap Equity Race & Community Engagement (ERACE)

Larry Delgado (while at DES)

Latino Leadership Network (LLN)

Laura Bradley (OAH)

Marika Barto (while at OMWBE)

Mark Adreon (former Department of Services for the Blind employee)

Microsoft Corporate External Legal Affairs Team: Jiam Ma, Sami Bailey, & Joanne Market

Minority Veterans of America

Nicholas Vann (DAHP)

Northwest Harvest

Northwest Museum of Arts and Culture

Office of Equity and Community Partnership-Public Health, Seattle & King County

Office of Financial Management (OFM) Forecasting Team

Office of the Commissioner of Public Lands

Pacific Lutheran University Executive Team

Pacific Northwest University of Health Services

Pierce County Community Engagement Task Force Leaders (PCCETF)

Pollution Liability Insurance Agency (PLIA)

Professional Educator Standards Board (PESB)

Puget Sound Partnership (PSP) (Laura Blackmore, Larry Epstein, Stephanie Suter, Melissa Schutten)

Rainbow Alliance and Inclusion Network (RAIN)

Results Washington

Robin Vazquez (HCA)

Rodney McAuley & Charlene Kay, Leaders in Spokane

Scott Nicholson (while at OFM)

Secretary of State Executive Team

Serve Washington

State Parks Commission

State Treasurer's Office

Statewide DEI Council and State Human Resources

Statewide Deputy Director Group 1

Statewide Deputy Director Group 2

Superior Court Translation Commission

Supreme Court Commission on Children in Foster Care

Theresa Powell (DSHS)

Tiffany Lamoreaux & Patricia Hayden

Utilities and Transportation Commission (UTC)

Veteran's Employee Resource Group (VERG)

Vicki Lowe (DOHi)

Washington Center for Deaf and Hard of Hearing Youth Executive Leadership Team

Washington Bar Association

Washington State Business Resource Leaders

Washington State Deaf Employees Meeting

Washington Department of Services for the Blind

Washington State Diversity, Equity & Inclusion council

Washington Federation of State Employees (WFSE)

Washington Immigrant Network (WIN)

Washington Office of Superintendent of Public Instruction (OSPI) Superintendent and Executive Team

Washington State Board for Community and Technical Colleges

Washington State Board of Pilotage Commissioners

Washington State Governor's Office for Regulatory Innovation & Assistance (ORIA)

Washington State Investment Board

Washington State LGBTQ Commission

Washington State Transportation Commission (WSTC)

Washington State Women's Commission

Washington Student Achievement Council (WSAC)

Washington Traffic Safety Commission

Washington Workforce Association (WWA)

We Are One America

Wendy T and Trillium Employment Services

Western Washington University

Workforce Training and Education Coordinating Board

Washington Recovery Group (WRG)

II. Foreword

"...equity and justice for all, now and for generations to come."

The year 2020 ushered in a decade of both historic challenge and historic opportunity.

- January: COVID-19 swept across the earth and nearly every aspect of our lives.
- March: Blacks, Latinos, and people fully fluent in their native language experienced significantly higher COVID-19 hospitalization and death rates than Whites.
- April: States began to reopen the economy.
- May: The world watched Mr. George Floyd's murder by police officers responding to a call from a store clerk claiming that he had paid for cigarettes with a counterfeit $20 bill.

Millions protested and demanded racial justice worldwide and here in Washington state. Racism, they said, is the real public health crisis that must be addressed because the distribution of privilege and the distribution of burden, based on one's membership in a particular social identity group, continues to be uneven and unjust across society.

We remember the year 2020 as a time of historic opportunity. The Washington State Office of Equity, located in the Office of the Governor, was established in April 2020 to promote equitable access to opportunities and resources that reduce disparities across state government and improve outcomes statewide (RCW 43.06D.020). The social justice movement of 2020 demanded that government admit and repair the harm it caused to so many people. What was considered "normal" before the pandemic—to serve and privilege some at the expense and erasure of others—was no longer acceptable to people worldwide or in Washington state. People across the state mobilized to hold state leaders accountable for ending disparities, especially racial and ethnic disparities, in all aspects of state agency decision-making.

We are excited to present the Washington State Pro-Equity Anti-Racism (PEAR) Ecosystem Plan & Playbook ("PEAR Plan & Playbook"). Co-created with input from thousands of state employees and Washingtonians, it recognizes that our people and environment are both healthy and flourish when we work together with those experiencing inequities to ensure that every person who works in, contracts with, or visits a state agency for assistance or services is welcomed, receives procedural and outcome fairness, and is treated with dignity, honor, and respect.

It is time for action. Join us as Washington state leads the way in transforming government to work in a way that achieves equity and justice for all, now and for generations to come.

What are you prePEARed to do in 2022 and beyond?

–Washington State Office of Equity Team
Office of the Governor

III. Preface

"Everyone is different. Everyone belongs here." ***–Unknown***

As the inaugural Director of the Washington State Office of Equity, located in the Office of the Governor, I am honored to present the *Washington State Pro-Equity Anti-Racism (PEAR) Ecosystem Plan & Playbook*.

The premise of the PEAR ecosystem is that each and every state employee, no matter their title or position, wants to bring their A-game and authentic self to work, to be seen, heard, and valued as they team up with their colleagues to deliver superior services in a way that affirms the humanity, dignity, and value of every person they encounter. Everyone is different. Everyone belongs here.

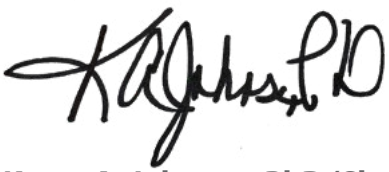
What follows is a description of why this historic work is necessary, how the PEAR Plan & Playbook was created, who created the plan, and how the PEAR Ecosystem will advance equity and justice for all across the state of Washington, now and for generations to come.

We believe the PEAR strategy will disrupt longstanding injustice and inequity and create sustainable change, innovation, and productivity statewide and across state government, giving Washington a competitive advantage for becoming the first Belonging state in the nation, the number one "state of choice" to live, learn, work, own a business, play, and stay.

The PEAR Plan & Playbook positions Washington as a national leader in partnering with communities to transform state government to work for everyone, and as a model for other public and private sector organizations to follow, especially those with whom we seek voluntary partnership.

Therefore, it is with great enthusiasm that I present *Washington's Pro-Equity Anti-Racism (PEAR) Ecosystem Plan & Playbook* and online tools to help Washington and its state-supported agencies, as well as other public and private sector organizations, achieve visible progress in equity, justice, access, and belonging.

Equity & Justice for all,



Karen A. Johnson, PhD (She/Her/Beloved)
Director, Washington State Office of Equity, Office of the Governor

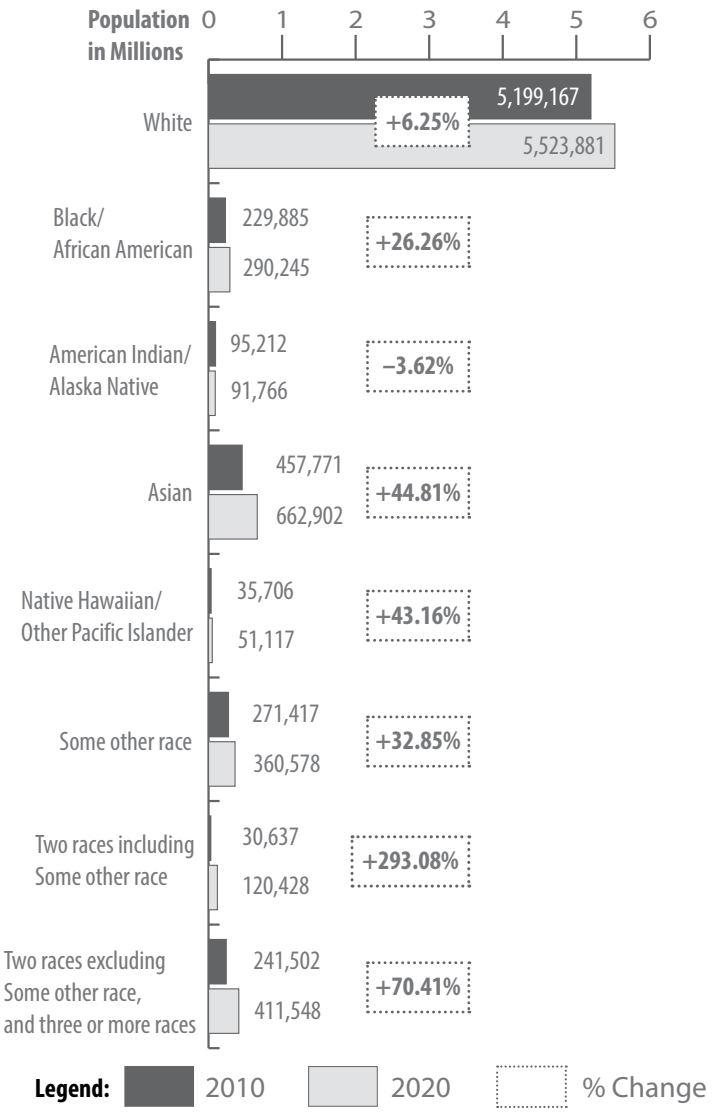
IV. Background: Why? How? So What?

Why? The legislature established the Office of Equity because:

1. They found that the diversity of Washington's population has increased over the last several decades.

Change in race and ethnicity of Washington residents from 2010 to 2020

Source: U.S. Census Bureau American Community Survey, 2010 and 2020 5-year Estimates Detailed Tables, last accessed 12/22/2022.



The graph at left compares the Washington State population of eight racial groups between 2010 and 2020.

The population of people who identify as:

White alone increased from 5,199,167 in 2010 to 5,523,881 in 2020, an increase of 6.25 percent.

Black or African American alone increased from 229,885 in 2010 to 290,245 in 2020, an increase of 26.26 percent.

American Indian/Alaska Native alone decreased from 95,212 in 2010 to 91,766 in 2020,
a decrease of 3.62 percent.

Asian alone increased from 457,771 in 2010 to 662,902 in 2020, an increase of 44.81 percent.

Native Hawaiian/Other Pacific Islander alone increased from 35,706 in 2010 to 51,117 in 2020,
an increase of 43.16 percent.

Some other race alone increased from 271,417 in 2010 to 360,578 in 2020, an increase of 32.85 percent.

Two races including Some other race increased from 30,637 in 2010 to 120,428 in 2020,
an increase of 293.08 percent.

Two races excluding Some other race, and three or more races increased from 241,502 in 2010 to 411,548 in 2020, an increase of 70.41 percent.

2. As the demographics of our state change, they found that:

1

People from historically and currently marginalized groups still do not have the same opportunities to experience health, wealth, and well-being as their nonmarginalized counterparts.

2

Inequities based on race, ethnicity, gender, and other characteristics continue to be deep, pervasive, and persistent, and they come at a great economic and social cost.

3

Work happening in agencies to address the disparate outcomes faced by people from historically and currently marginalized groups is fragmented across state government.

How? Listening and learning

Between May and September 2021, state agencies and the Office of Equity conducted baseline equity organizational readiness assessments and listened to thousands of community members and state employees to better understand their priorities for the state's first five-year equity strategic plan, designed to bridge opportunity gaps and reduce disparities, including racial and ethnic disparities, statewide and across state government. You can view the data dashboards that present the results from these efforts in the Online Toolkit.

What we heard from community members and state employees

The following is a collection of quotes from nine different people who responded to the listening surveys. This is a very small sampling of the feedback we received.

"Go to the people. Listen early (before the process is designed or decisions are made) rather than late and listen often. We need to get this right."

"Explore sustainability from a community perspective: how to build learning structures and culture."

"Do not create splitting, pitting, or divisive environments for people impacted by inequities."

"Agencies need to **understand differences in cultural values** when co-creating with communities."

"Build trust and relationship by **hiring people from impacted communities."**

"Hire staff who look like communities served and **ensure that they are at the decision-making table."**

"Set truth and reconciliation tables to **lay a solid foundation of trust to own harm and begin healing."**

"Take the time necessary to be relational instead of rushing this in the transaction of work."

"Intentionally make time to **hear the stories from the community about the harm caused by government** and own the pain caused."

So What?

We incorporated all of this input to collaboratively create *Washington's Pro-Equity Anti-Racism (PEAR) Ecosystem Plan & Playbook*, a statewide strategy that calls for state agencies and communities to work together to achieve equity and justice statewide and across Washington's 100+ state-supported agencies.

V. PEAR Plan & Playbook Design Team

Washington's Pro-Equity Anti-Racism (PEAR) Ecosystem Plan & Playbook was developed in partnership with a team of expert consultants who also created an online toolkit and data maps for use by state agencies and other stakeholders across the state to collectively measure progress toward broad goals that advance equity and justice in Washington.

PEAR Plan & Playbook Design Team

Karen A. Johnson, Ph.D.
Director
WA State Office of Equity

Nicole Franklin
Enhanced Interactions,
PEAR Strategist, Team Convener, Project Manager

john miller
Collective Brilliance
Washington's PEAR Playbook

Ricardo Ibarra, Trio Group
Listening Sessions and Equity Baseline Dashboards, PEAR Plan & Playbook PDF and web versions

Clinton Johnson, Northstar of GIS
Equity GIS visualizations, performance dashboards, and online PEAR destination & playspace

"Teamwork makes the dream work." *–John Maxwell*

PEAR Orientation Sessions

Carolyn Cole
Assistant Director
Equity, Access & Belonging

Megan Matthews
Assistant Director
Shared Power Design

Former Office of Equity Team Members:

Cynthia "Cindy" Varley

LaTasha "Tasha" Tisdel

VI. Executive Summary

The Washington State Pro-Equity Anti-Racism (PEAR) Ecosystem Plan & Playbook outlines the framework and tools that Washington state agencies will use to create a PEAR ecosystem in which all Washingtonians have full access to the opportunities, power, and resources they need to flourish and achieve their full potential.

PEAR Ecosystem Goals

1. Reduce disparities in public contracting, public education, public employment, and public services (Executive Order 22-02).
2. Improve outcomes that benefit all tribes, communities, and employees of Washington's PEAR ecosystem (Executive Order 22-04).

Strategies to help state agencies to achieve the PEAR Ecosystem Goals include:

1. Implement a pro-equity, anti-racism framework in partnership with relevant communities and organizations
2. Embrace continuous learning, growing, and pivoting
3. Consistently assess your actions' equity impact
4. Make values driven, data informed, upstream investments
5. Be transparent, accountable, and operate with urgency

This document contains:

- Definitions and Abbreviations (Section VII) commonly used in this document.
- Introduction (Section VIII) describing what it will take for Washington state to transform from its current state to a Pro-Equity Anti-Racism (PEAR) ecosystem (future state).
- Washington's PEAR Ecosystem Framework (Section IX) described.
- Washington State's PEAR Ecosystem Strategic Plan (Section X) including vision, mission, values, goals and overall strategies, outcomes, and 2022-2027 implementation roadmap (Section XI).

"Everything seems impossible until it's done." *–Nelson Mandela*

Online Toolkit

The Online Toolkit (available on the Office of Equity website) will help facilitate each state agency's implementation of Washington's PEAR Ecosystem Plan & Playbook. Every leader is expected to fully leverage all of the online resources. The resources provided are adaptable and can be tailored to meet the needs of each agency or organization. The contents of the Online Toolkit are listed below.

- The ***Washington State Pro-Equity Anti-Racism (PEAR) Ecosystem Plan & Playbook*** (this document)
- **Easy as 1-2-3:** PowerPoint decks, recorded trainings, and tools provided during PEAR Team Orientation sessions, including guidance for establishing a PEAR Team, completing an initial Equity Impact Assessment and PEAR Strategic Action Plan, and developing a quarterly performance tracker.
- **The Equity Impact Assessment (EIA) tools:** Tools that can be used prior to proposing changes to agency policies, programs, and practices, including budgets and agency request legislation, to assess the potential impact on communities that historically have been marginalized and institutionally oppressed.
- **Language Access guides:** Guides that include best practices and resources for providing language services (written translation, spoken language interpreting services, and sign language interpreting services) to individuals requiring language access to agency programs, activities, and services.
- **Relational Partnership Guide:** Relationships are the foundation of establishing impactful teams that can produce deliverables. The Relational Partnership Guide provides guidance for building relationships with community to help establish your PEAR Teams and build and grow your community network.
- **Tribal government relationship guides:** Guides that include information about the legal framework and requirements for the government-to-government relationship between tribes and the state of Washington and resources to assist agencies in fulfilling their legal obligations as required by Chapter 43.376 RCW.

More resources, including an entire section on the development of the **Statewide Universal Access & Belonging Plan**[1], will be added as they become available.

VII. Definitions & Abbreviations

It is important to have a shared vocabulary in equity and social justice work. Below is a short list of common terms and abbreviations used throughout this document, along with their meanings. A more comprehensive "Glossary of Terms" intended to advance education and awareness of institutional and structural racism and to assist in formulating appropriate language for any formal or informal conversations about race, racism, proposed executive action, or upcoming legislation will be provided in the online toolkit.

Anti-Racism
Anti-racism is a process of actively identifying and opposing racism. The goal of anti-racism is to challenge racism and actively change the policies, behaviors, and beliefs that perpetuate racist ideas and actions.

"Anti-racism is rooted in action. It is about taking steps to eliminate racism at the individual, institutional, and structural levels." Source: Verywell Mind

Belonging
The Office of Equity uses john a. powell's definition of belonging. Belonging calls for something more than Inclusion and Equity, yet also includes them in meaningful ways.

Belonging is both objective and subjective.

- It can be quantified and measured, but it is also a perception found in the eye of the beholder. In this respect, Belonging, unlike both Equity and Inclusion, contains a psychological component — an affective component, which shapes the way social groups regard whatever it is they are regarding: an institution, a city, or even society writ large.
- If members of a social group feel as if they belong, then belonging exists. But if they do not, despite being included and having few tangible resource inequities or other disparities between groups, then belonging is lacking.

A core element of belonging: the expressive or communicative message that a group belongs.

- It can be expressed explicitly, through representation, or by signaling that members of a particular group are welcome in a particular space, institution, or community.
- It can also be expressed implicitly, as when accommodations are made, such as when special food or holidays are provided for.

Belonging is perceptual and tangible; it is a feeling and a practice. Belonging requires more than accommodation; it also demands agency. Belonging is realized fully when included groups have more than a voice — they are actually able to reshape the institution together with existing stakeholders.

"If my colleagues had the right language, it would make conversations a thousand times easier." –David Baboolall (they/them)

BIPOC

Black, Indigenous, and People of Color

DE&I
Diversity, Equity & Inclusion

Diversity
Building diversity in our state agencies is only a starting point. Diversity is defined broadly as any difference whatsoever, all of the characteristics that make individuals unique. It is used to describe the various combinations of group/social differences (e.g., race/ethnicity, class, gender, gender identity, sexual orientation, country of origin, and ability, as well as cultural, political, religious and other affiliations) and human differences (e.g., personality, learning style, and life experiences). Our working definition of diversity is to foster a work environment of belonging for every employee, recognizing and effectively utilizing their talent, skills, and perspectives to create a unified and high-performance workforce.

Ecosystem
The biological community of living beings, communicating with the physical environment and other nonliving components. It can also be defined as the chain of communication or interaction between the living organisms and their environment

Equity
The most important construct among DE&I, equity, refers to the creation of opportunities for historically underrepresented populations to have equitable access to equitable opportunity. Equity is also the process of allocating resources, programs, and opportunities to employees, customers, and residents to address historical discrimination and existing imbalances. Therefore, equity requires an organizational commitment that all employees, customers, and residents will be provided equitable access to opportunities, resources, and the ability to fully contribute to the agency's mission and goals.

The work of the Office of Equity must be guided by the following principles of equity per RCW 43.06D.020:

- Developing, strengthening, and supporting policies and procedures that distribute and prioritize resources to those who have been historically and are currently marginalized, including tribes;
- Eliminating systemic barriers that have been deeply entrenched in systems of inequality and oppression; and
- Achieving procedural and outcome fairness, promoting dignity, honor, and respect for all people.

Historical(ly)
This term refers to a 10-year or longer trend at a given department, agency, organization, or state.

PEAR
Pro-equity, anti-racism (see Pro-Equity)

Institutional Racism
The policies and practices within and across institutions that, intentionally or not, produce outcomes that chronically favor or place one racial group at a disadvantage. The overlapping and intersectionality of personal characteristics, including race, color, national origin, ethnicity, religion, gender, sex, sexual orientation, gender identity, and disability, determine the degree of disadvantage. Examples of institutional racism can be found in school disciplinary policies in which students of color are punished at much higher rates than their white counterparts; in the criminal justice system; and within many employment sectors where day-to-day operations, as well as hiring and firing practices, significantly impact workers of color in a negative manner.

PEAR Ecosystem
Recognizing the interconnectedness between human systems and nature systems, our working definition of the PEAR ecosystem is finding and fostering a microclimate for change:

- Community is the guiding light
- Interconnected system of PEAR Values, PEAR Service Lines, and PEAR Determinants of Equity
- Outcomes: All people in Washington flourish and achieve their full potential, embody pro-equity, anti-racism values, and enjoy peace, prosperity, and possibility now and for generations to come.

Pro-Equity
"…[T]he proactive way of doing equity work... the knowledge that we live in a society permeated by racism and bigotry... combat or control... in every action..." Source: Caroline Hill.

Relational Partnership
Empathy-centered collaboration between government and people groups who have been excluded and marginalized by government decisions and actions… to undo harm and advance pro-equity anti-racism (PEAR) outcomes.

Structural Racism
A system in which public policies, institutional practices, cultural representations, and other norms work in various, often reinforcing, ways to perpetuate racial group inequity. It identifies dimensions of our history and culture that have allowed privileges associated with "whiteness" and disadvantages associated with "color" to endure and adapt over time. Structural racism is not something that a few people or institutions choose to practice. Instead, it has been a feature of the complex social, economic, and political systems in which we all exist.

Underrepresented
This term refers to populations, of employees, for example, that are disproportionately lower in number relative to their number in the national/state population.

VIII. Introduction

"The wrong first question is what do we need to do? The right first question is who do we need to become?"

–Benjamin McBride

Washington state will transform from its current state to a Pro-Equity Anti-Racism (PEAR) ecosystem (future state).

Current state:

Some Washingtonians question the legitimacy of state government because decisions are consistently made without them.

Some Washingtonians question whether state government is effective because it is not delivering services that meet their needs. People feel devalued and often cannot access services.

Some Washingtonians do not trust state government because of its history of oppression and marginalization. People are left behind, hopeless, homeless, frustrated, and disconnected.

Future state:

Implementing *Washington's Pro-Equity Anti-Racism (PEAR) Ecosystem Plan & Playbook* (Executive Order 22-04) is a critical dimension of the state's pursuit of bridging opportunity gaps and reducing disparities, including racial and ethnic disparities, statewide and across state government, to keep Washington a great place to live, learn, work, play, and stay.

To remain the number one place to live in the nation and to become a Belonging state, our state government must be able to implement a pro-equity, anti-racism (PEAR) approach that goes beyond meeting government mandates and legal compliance; it is about recognizing that a state workplace culture of equity, justice, access, and belonging produces a competitive business advantage and return on investment with regard to performance, outcomes, and learning. It is about state agencies and communities that are traditionally left out and left behind working together toward reaching our broad, shared goal of achieving equity here: especially in public contracting, public employment, public education, and access to public services (Executive Order 22-02).

The role of leadership is to create a PEAR culture rooted in equity, justice, access, and belonging to ensure that all people in Washington flourish and achieve their full potential, embody pro-equity anti-racism values, and enjoy peace, prosperity, and possibility now and for generations to come.

Success requires leaders to 1) formally assess their own biases and personal experiences in order to listen, learn, and lead employees in this transformative work, 2) be willing to exhibit exemplary leadership behaviors and implement the processes necessary for attaining both short- and long-term PEAR goals, and 3) prioritize on-going learning opportunities designed to meet the needs of the people we serve and the people who serve them.

Successful equity and belonging reform will involve ongoing experimentation, assessment, and innovation, most of which will challenge historical policies and practices that have presented barriers to achieving equity statewide and across state government, especially for those facing persistent inequities and injustice.

Formalized mechanisms of assessment will serve to hold leaders accountable for increasing and supporting equity and making a belonging environment an agency priority.

PEAR Champions, such as the agency head, the agency-level equity officer, and members of the agency PEAR Team or PEAR Team Advisory Group will play leading roles in promoting and sustaining an organizational culture that values and supports PEAR outcomes. Yet, achieving equity is everyone's work. Applying equity considerations to every law, rule, policy, program, practice, procedure, and interaction both at agency headquarters and in the field requires the collective action of every employee throughout state agencies and Washington communities.

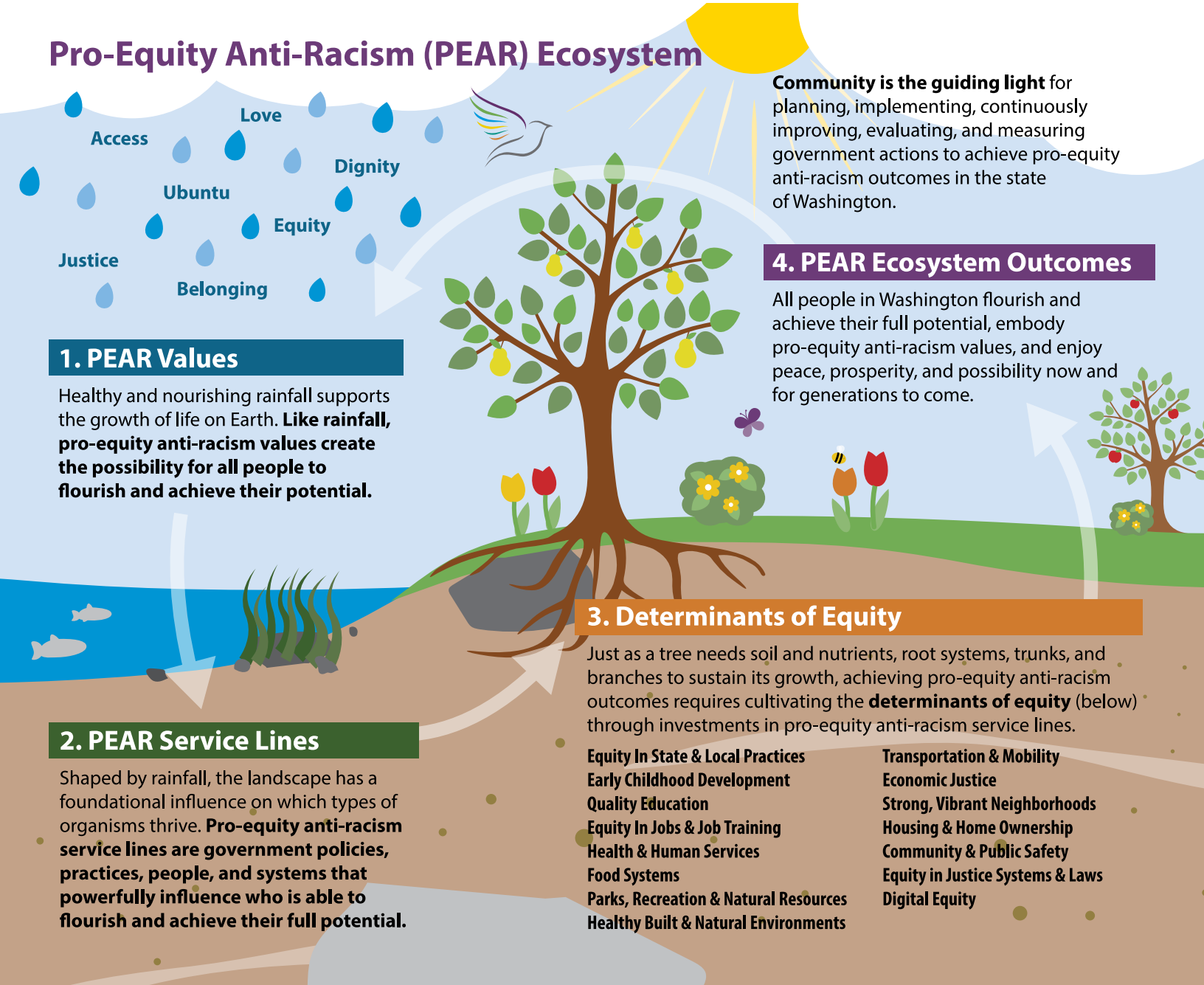
"No phenomenon can be isolated, but has repercussions through every aspect of our lives. We are learning that we are a fundamental part of nature's ecosystems."

–Arthur Erickson

IX. Washington's PEAR Ecosystem Framework

"…The PEAR framework prioritizes investments in 15 determinants of equity to achieve outcomes that benefit all tribes, communities, and employees of Washington's ecosystem, and calls for investing more of our state's resources "upstream" to address root causes where the needs are greatest to ensure that individuals in underserved communities have their basic needs met long term in Washington's ecosystem." –Executive Order 22-04

Pro-Equity Anti-Racism (PEAR) Ecosystem



Community is the guiding light for planning, implementing, continuously improving, evaluating, and measuring government actions to achieve pro-equity anti-racism outcomes in the state of Washington.

1. PEAR Values

Healthy and nourishing rainfall supports the growth of life on Earth. **Like rainfall, pro-equity anti-racism values create the possibility for all people to flourish and achieve their potential.**

4. PEAR Ecosystem Outcomes

All people in Washington flourish and achieve their full potential, embody pro-equity anti-racism values, and enjoy peace, prosperity, and possibility now and for generations to come.

2. PEAR Service Lines

Shaped by rainfall, the landscape has a foundational influence on which types of organisms thrive. **Pro-equity anti-racism service lines are government policies, practices, people, and systems that powerfully influence who is able to flourish and achieve their full potential.**

3. Determinants of Equity

Just as a tree needs soil and nutrients, root systems, trunks, and branches to sustain its growth, achieving pro-equity anti-racism outcomes requires cultivating the **determinants of equity** (below) through investments in pro-equity anti-racism service lines.

- Equity In State & Local Practices
- Early Childhood Development
- Quality Education
- Equity In Jobs & Job Training
- Health & Human Services
- Food Systems
- Parks, Recreation & Natural Resources
- Healthy Built & Natural Environments
- Transportation & Mobility
- Economic Justice
- Strong, Vibrant Neighborhoods
- Housing & Home Ownership
- Community & Public Safety
- Equity in Justice Systems & Laws
- Digital Equity

Pro-Equity Anti-Racism (PEAR) Ecosystem

Image description for the previous page: A bright sun peeks through white clouds above a fruitful pear tree in the center of the image. The tree's roots are shown extending into the soil which provides stability and nutrients. To the left of the tree, a body of water is home to fish and aquatic plants. Flowers and other plants are visited by a bee and a butterfly as they grow from a green field to the right of the pear tree.

Text: Community is the guiding light for planning, implementing, continuously improving, evaluating, and measuring government actions to achieve pro-equity anti-racism outcomes in the state of Washington.

1. PEAR Values (rain feeding the soil)

Healthy and nourishing rainfall supports the growth of life on Earth. Like rainfall, pro-equity anti-racism values create the possibility for all people to flourish and achieve their potential.

Values coming out of clouds through rain:
Access, Justice, Ubuntu, Love, Equity, Dignity, Belonging

2. PEAR Service Lines (soil providing nutrients for tree)

Shaped by rainfall, the landscape has a foundational influence on which types of organisms thrive. Pro-equity, anti-racism service lines are government policies, practices, people, and systems that powerfully influence who is able to flourish and achieve their full potential.

3. Determinants of Equity (fruitful tree)

Just as a tree needs soil and nutrients, root systems, trunks, and branches to sustain its growth, achieving pro-equity, anti-racism outcomes requires cultivating the determinants of equity (below) through investments in pro-equity, anti-racism service lines:

- Equity In State & Local Practices
- Early Childhood Development
- Quality Education
- Equity In Jobs & Job Training
- Health & Human Services
- Food Systems
- Parks, Recreation & Natural Resources
- Healthy Built & Natural Environments
- Transportation & Mobility
- Economic Justice
- Strong, Vibrant Neighborhoods
- Housing & Home Ownership
- Community & Public Safety
- Equity in Justice Systems & Laws
- Digital Equity

4. PEAR Ecosystem Outcomes (flows back into clouds)

All people in Washington flourish and achieve their full potential, embody pro-equity, anti-racism values, and enjoy peace, prosperity, and possibility now and for generations to come.

Determinants of Equity[2]

Washington will achieve PEAR ecosystem outcomes by cultivating and measuring state agencies' impact on **15 Determinants of Equity**, the social conditions that everyone in Washington needs to flourish and achieve their full potential:

Equity in State & Local Practices

Early Childhood Development

Quality Education

Equity in Jobs & Job Training

Health & Human Services

Food Systems

Parks, Recreation & Natural Resources

Healthy Built & Natural Environments

Transportation & Mobility

Economic Justice

Strong, Vibrant Neighborhoods

Housing & Home Ownership

Community & Public Safety

Equity in Justice Systems & Laws

Digital Equity[3]

Pro-Equity Anti-Racism (PEAR) Service Lines

"This is the interrelated structure of reality."
–Reverend Dr. Martin Luther King, Jr.

Washington state agencies will cultivate 15 Determinants of Equity by focusing PEAR Strategic Action Plan Investments in PEAR Service Lines, which are 11 aspects of agency decision making, including service delivery, program development, policy development, and budgeting (RCW 43.06D.040), in order to achieve PEAR outcomes.

The 11 PEAR Service Lines are:

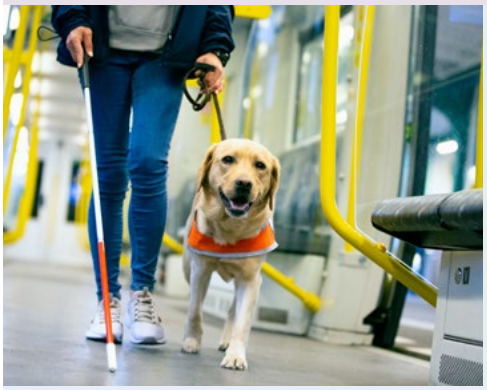
Leadership, Operations & Services: Advance PEAR practices and systems at all levels of state government through transparent and accountable organizational development and adaptive change agent leadership.

Plans, Policies & Budgets: Incorporate PEAR values into plans, policies, and budgets to meet the needs of employees and the communities we serve, eliminating disparities where the needs are greatest.

Workforce Equity: Develop a PEAR organizational culture by building a diverse (including racially and ethnically diverse) and culturally responsive pipeline for employees at all levels and create opportunities for each employee to bring their full self to work and feel welcomed, supported, and valued.

Tribal Government Relationships[4]: Invest in Tribal governments and enterprises and Tribal organizations that progressively inform our state's PEAR decision-making lens and cultivate equitable, racially just and accessible participation with recognition of the unique histories of Tribes and American Indian/Alaska Native people, their connection to traditional territories, and the significance of the connection between the land and cultural ways of life practiced since before our larger nation was founded.

Public Communications & Education: Advance our state's capacity to better communicate and educate our communities and employees in ways that are equitable, racially just, accessible, and cultivate a sense of belonging.



Facilities & Systems Improvements: Design and develop facilities improvements, public works projects, and business diversity programs that center the values and priorities of our employees and the communities we serve.

Policy Agenda: Address root causes of disparities through policies, practices, and systems to end disparities, including racial and ethnic disparities, and improve outcomes statewide across state government, particularly in hiring and promotions, state spend for public works, goods and services (including client services), procurement, and access to services.

Building a Racially Just Washington: Use PEAR strategies and tools to eliminate racial inequities and improve outcomes for all racial groups, with an intentional focus on places where the needs are greatest.

Capacity Building: Intentionally develop organizational capacity to support the implementation of the PEAR framework in all agency-decision making.

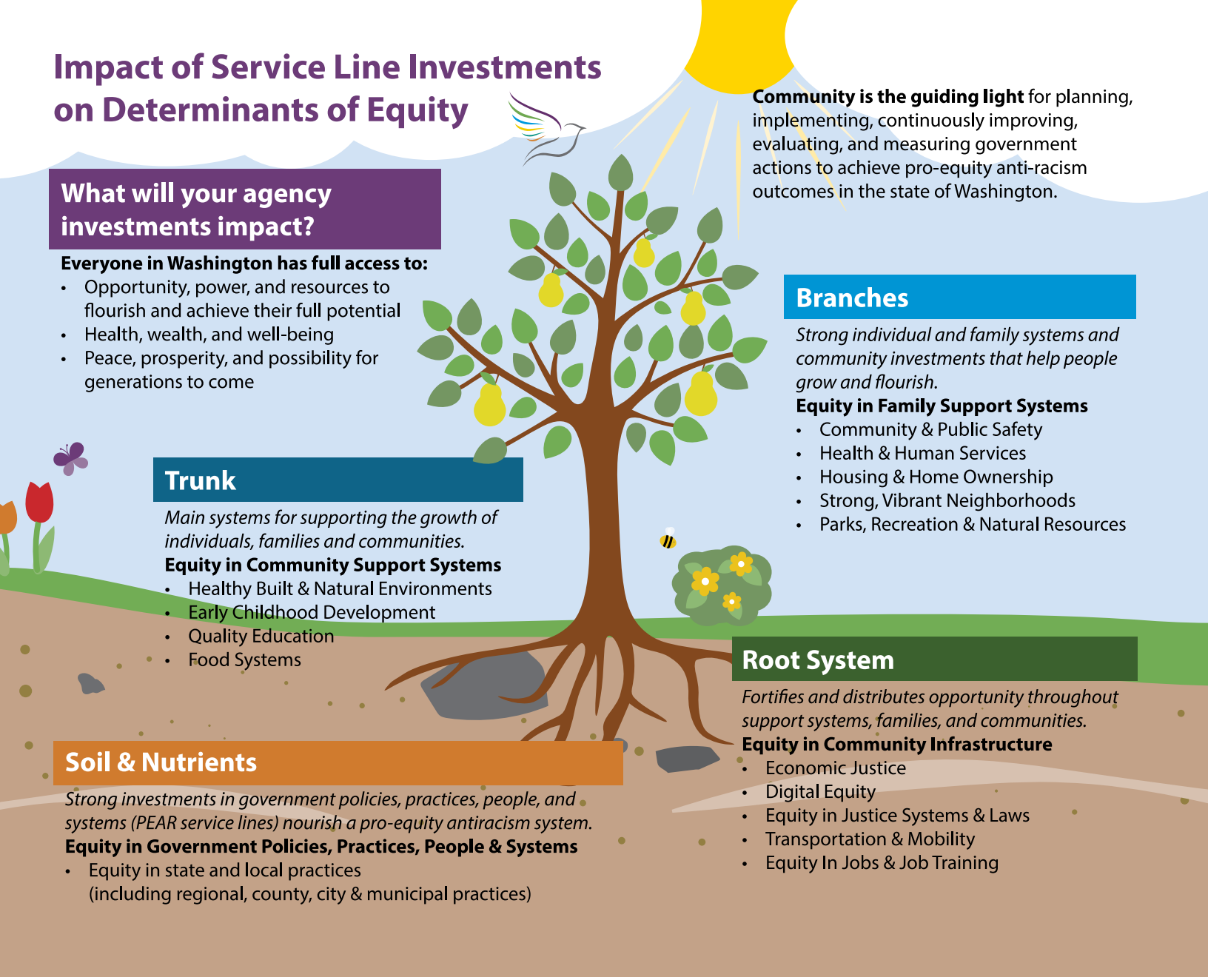
Data & Strategy Reporting: Invest in data and strategy reporting systems to ensure that we drive equitable outcomes by investing where the needs are greatest and hold state agencies accountable for eliminating disparities in their business lines.

Engagement & Community Partnerships: Build partnerships with communities and employees that inform and support Washington state's PEAR ecosystem.

Consultation and Technical Assistance

Within these 11 PEAR Service Lines, the Office of Equity's Pro-Equity Consultants will provide consultation and technical assistance to help agencies identify:

- PEAR priorities
- PEAR Strategic Action Plan Investments
- PEAR organizational habits
- PEAR intended outcomes
- PEAR performance measures



Impact of Service Line Investments on Determinants of Equity

Image description: A bright sun peeks through white clouds above a fruitful pear tree in the center of the image. The tree's roots are shown extending into the soil which provides stability and nutrients. Flowers and other plants grow from a green field surrounding the pear tree.

Text: Community is the guiding light for planning, implementing, continuously improving, evaluating, and measuring government actions to achieve pro-equity, anti-racism outcomes in the state of Washington.

What will your agency investments impact?

Everyone in Washington has full access to:

- Opportunity, power, and resources to flourish and achieve their full potential
- Health, wealth, and well-being
- Peace, prosperity, and possibility for generations to come.

Equity in Community Support Systems (Trunk)

Main systems for supporting the growth of individuals, families and communities.

- Healthy Built & Natural Environments
- Early Childhood Development
- Quality Education
- Food Systems

Equity in Government Policies, Practices, People & Systems (Soil & Nutrients)

Strong investments in government policies, practices, people, and systems (PEAR service lines) nourish a pro-equity, antiracism system.

- Equity in state and local practices (including regional, county, city & municipal practices)

Equity in Community Infrastructure (Root System)

Fortifies and distributes opportunity throughout support systems, families, and communities.

- Economic Justice
- Digital Equity
- Equity in Justice Systems & Laws
- Transportation & Mobility
- Equity In Jobs & Job Training

Equity in Family Support Systems (Branches)

Strong individual and family systems and community investments that help people grow and flourish.

- Community & Public Safety
- Health & Human Services
- Housing & Home Ownership
- Strong, Vibrant Neighborhoods
- Parks, Recreation & Natural Resources

"And while I stood there, I saw more than I can tell, and I understood more than I saw; for I was seeing in a sacred manner the shapes of things in the spirit, and the shape of all shapes as they must live together like one being."

—Black Elk, Black Elk Speaks

X. Washington State's PEAR Ecosystem Strategic Plan (2022-2027)

A. PEAR Ecosystem Vision and Mission

Vision

Everyone in Washington has full access to the opportunities, power, and resources they need to flourish and achieve their full potential.

Mission

Promote equitable access to opportunities, power, and resources across government that reduces disparities and improve outcomes statewide.

B. PEAR Ecosystem Values

Values are basic and fundamental beliefs that guide or motivate our attitudes or actions. They help us to determine what is important to us. Values are the motive behind purposeful action.[5]

The following values reflect the common themes that surfaced during the listening sessions conducted by the Office of Equity and state agencies between May and September 2021.

Access: Creating and supporting barrier-free design, standards, systems, processes, and environments so that all individuals, regardless of ability, background, identity, or situation, can participate in, use, and enjoy the benefits of: employment, programs, services, activities, communication, facilities, electronic information technology, and business opportunities.

Belonging: Values and practices that ensure no person is left out of our circle of concern. Belonging means more than just having access, being seen, or feeling included. It means that every member of society has a meaningful voice, that their well-being is considered, and that they can participate in the design of political, social, and cultural structures.

Dignity: We respect the sacred nature of each individual's personhood. We honor the worth due each person by virtue of their existence as a human being. Human lives have an unimpeachable value simply because they are human, and therefore deserving of a baseline level of respect. That baseline requires more than the absence of violence, discrimination, and authoritarianism. It means giving individuals the freedom to pursue their own happiness and purpose.

Equity: Systemic, full, and true access to opportunities, power, and resources that allow all people to achieve their full potential and thrive.[6] Our actions and decisions will be guided by the following principles of equity (RCW 43.06D):

- Equity is not equality. Equity requires developing, strengthening, and supporting policies and procedures that distribute and prioritize resources to people in identified groups who have been historically and currently are marginalized, including tribes;
- Equity requires the elimination of systemic barriers that have been deeply entrenched in systems of inequality and oppression; and
- Equity achieves procedural and outcome fairness, promoting dignity, honor, and respect for all people.

Justice: We make or do right that which has been done wrong. We embody what love looks like in action.

Love: Sometimes defined as a strong affection for another arising out of kinship or personal ties.[7] Love requires us to:

- **Fumble Forward:** The idea that we are each on a journey. We recognize that while we are on this journey, we are doing the best we can with the tools, conditions, and knowledge we have. We will have compassion and care for one another as we grow.
- **Stay committed; stay open; stay adaptive:** Our collective willingness to embrace the concept that words matter and that the labels we ascribe to ourselves are not simply ways of being "politically correct," they are validations of our humanity. We create and support belonging by expressing love to one another and treating others as they want to be identified and treated. We will check our fear-based decisions to ensure a better future for all is achieved.
- **Be humble:** We own our stories, points-of-view, successes, and mistakes. We admit we do not know everything, in fact no one does, and that instead, we all have something learn from one another. We acknowledge there are things we do not know so we can approach each other with love.

Ubuntu: A South African (Nguni Bantu) term meaning "humanity," often translated as "I am because we are," stresses the importance of the interconnectedness of humanity. We recognize that our destinies are linked and we need each other to survive.

Apply PEAR Values to Agency Work

State agencies are encouraged to tailor the descriptions for the values listed above in a way that guides their agency's PEAR work.

Example use of PEAR Values
Below is an example of how the Washington State Office of Equity describes the PEAR values in all of its job announcements:

We Value

- **Access:** Barrier-free environments so everyone can participate.
- **Belonging:** The right to participate in all aspects of society with acceptance, attention, and support from members of the society, providing the same to others.
- **Dignity:** We honor the sacred nature of each individual's personhood.
- **Equity:** Acknowledging systemic inequalities by developing, strengthening, and supporting policies and procedures that distribute and prioritize resources to people in social identity groups who have been historically and currently are marginalized to ensure everyone has access to the same opportunities, power, resources, and outcomes to achieve equality.
- **Justice:** Treating people fairly. To make right. What love looks like in public (Cornel West).
- **Love:** A selfless and giving act of the will. We seek to out-give and out-serve the other.
- **Ubuntu:** I am because we are. We are interconnected.

C. PEAR Ecosystem Goals & Overall Strategies

The PEAR goals and overall strategies below guide the delivery of state goods, services, policies, and practices so all Washingtonians can participate, prosper, and achieve their full potential. The Office of Equity will partner with state agencies and communities to create an annual report to the Governor and Legislature. The report will include agency strengths and accomplishments made on PEAR expectations and the effectiveness of agency programs and services on reducing disparities, including the agency's action plan to address areas for continued improvement and a timeline for the action plan.

Overall PEAR Ecosystem Goals

Reduce disparities in public contracting, public education, public employment, and public services. –Executive Order 22-02

Improve outcomes that benefit all tribes, communities, and employees of Washington's PEAR ecosystem. –Executive Order 22-04

Overall PEAR Ecosystem Strategies

1.Implement a pro-equity, anti-racism framework in partnership with relevant communities and organizations
Partner with others to intentionally name and address implicit and explicit bias and all levels of racism, particularly against people who are seen and treated as Black, Indigenous, or People of Color.

2. Embrace continuous learning, growing, and pivoting
Build organizational capacity and infrastructure to continuously learn, improve, and make adjustments to sustain meaningful policy and systems change that achieves equitable policies, practices, and outcomes.

3. Consistently assess your equity impact
Understand and acknowledge your agency's equity impact to inform agency planning, decision-making, and action steps when changing policies, programs, and practices that perpetuate inequities and when developing new policies and programs that perpetuate equity.

4. Make values driven, data informed upstream investments
Identify and target root causes of opportunity gaps and disparities and prioritize the people who have traditionally been excluded to improve outcomes that benefit all.

5. Be transparent, accountable, and operate with urgency
Create and maintain a long-term commitment to change and help others to see the benefit to them for acting immediately. Build public trust and accountability for sustaining equity through values-driven, data-informed decision-making and outcome tracking.

D. PEAR Ecosystem Outcomes

All people in Washington flourish and achieve their full potential, embody pro-equity anti-racism values, and enjoy peace, prosperity, and possibility now and for generations to come.

XI. 2022-2027 PEAR Implementation Strategy

"For tomorrow belongs to the people who prepare for it today."
–African Proverb

The Office of Equity and all state agency leaders are responsible and accountable for implementing Executive Order 22-04, "Implementing the Washington State Pro-Equity Anti-Racism (PEAR) Plan & Playbook."

State Agency Leaders are directed to:

1. Develop a PEAR Team;
2. Conduct an agency Equity Impact Assessment;
3. Develop and implement PEAR Strategic Action Plan; and
4. Prepare and submit a PEAR Annual Performance Report to the Office of Equity each year to demonstrate performance, using outcome-based methodology to determine the effectiveness of agency programs and services on reducing disparities, taking into consideration community feedback on whether the performance measures established accurately measure the effectiveness of agency programs and services in the communities served.

Implementation Phases

The Washington State Office of Equity will provide consultation, technical assistance, and resources to facilitate state agency implementation of Washington's PEAR Ecosystem in three phases.

PHASE 1

Phase 1: Compliance

Support agencies in meeting Executive Order 22-04 to apply a pro-equity, anti-racism lens in all aspects of decision-making (RCW 43.06D.040 (1)(a)).

1. Establish PEAR Teams
2. Conduct Equity Impact Assessment
3. Develop PEAR Strategic Action Plans
4. Produce PEAR Annual Performance Report

PHASE 2

Phase 2: Transformation

Champion agency transformation that seeks and embraces community partnership, centering the voice of people impacted by state programs and services in all we do (RCW 43.06D.040 (1)(a)).

Within the 11 PEAR Service Lines, the Office of Equity's Pro-Equity Consultants will provide consultation and technical assistance to help agencies identify:

- PEAR priorities
- PEAR strategic action plan investments
- PEAR organizational habits
- PEAR intended outcomes
- PEAR performance measures

PHASE 3

Phase 3: Accountability

Establish statewide and agency-specific process and performance measures that foster continued growth and movement towards equity and justice for all, measuring outcomes and impact (RCW 43.06D.040 (1)(a)).

Implementation Roadmap

Year 1

- Agency leaders establish their PEAR Teams to create agency PEAR Strategic Action Plans.
- Office of Equity holds PEAR Team Orientation sessions and provides technical assistance to agency leaders and their PEAR Teams.
- Agencies complete the PEAR Readiness Checklist (Available in Online Toolkit).
- Agencies complete a Baseline Equity Impact Assessment (EIA) of key business lines to identify where the needs are greatest and root causes of disparities (Available in Online Toolkit).
- Based on the results of the EIA, agencies complete a PEAR Strategic Action Plan Template (Available in Online Toolkit) and submit to the Office of Equity by September 1, 2022.
- Implementation of agency PEAR Strategic Action Plans begins September 1, 2022.
- Office of Equity helps establish a statewide PEAR Team and PEAR Team Advisory Group to create the Statewide Universal Access & Belonging Plan.
- Office of Equity prepares and submits a report to the Governor and Legislature by October 31, 2022, and every year thereafter.

Years 2–4

- Agencies continue to conduct EIAs prior to proposing changes to agency policies, programs, and practices.
- Continued implementation of agency PEAR Strategic Action Plans.
- Office of Equity continues to provide technical assistance to agency leaders and their PEAR Teams.
- Agencies partner with Office of Equity to conduct quarterly reviews of PEAR Strategic Action Plan performance.
- PEAR Strategic Action Plans are adjusted as needed to achieve PEAR outcomes and goals.
- Updated PEAR Strategic Action Plans are submitted to the Office of Equity annually.
- Agencies prepare and submit agency PEAR Annual Performance Reports to the Office of Equity.
- Agencies prepare and submit responses to reports published by the Office of Equity on agency PEAR Strategic Action Plan performance.
- Statewide PEAR Team completes the Statewide Universal Access & Belonging Plan. Begin implementation and performance monitoring.

Year 5

- Agencies prepare and submit a Five-Year PEAR Performance Report to the Office of Equity that summarizes PEAR Strategic Action Plan performance since September 1, 2022.
- Office of Equity prepares and submits a Five-Year PEAR performance report to the Governor and Legislature.
- Office of Equity leads the development of the 2028 - 2033 PEAR Plan & Playbook.

XII. Conclusion

"If you want to go fast, go alone. If you want to go far, go together."
–African Proverb

Advancing a state government and statewide culture and reality in which each and every person belongs, matters, and has what they need to be successful requires time, commitment, and active engagement of every person who plays any role in state government.

The *Washington PEAR Ecosystem Plan & Playbook* will be successful only if everyone assumes responsibility and a role in our collective impact. Thus, *if* we embrace the *PEAR Ecosystem Plan & Playbook* mission to promote equitable access to opportunities, power, and resources across state government that reduces disparities and improves outcomes statewide, achieve equitable access and fairness in public contracting, public education, public employment, and public services (Executive Order 22-02), and invest upstream to address root causes of inequities where the needs are greatest to achieve outcomes that benefit all tribes, communities, and employees of Washington's PEAR ecosystem (Executive Order 22-04), *then* we will position Washington to be truly an equitable Belonging state, the number one place where all people in a U.S. state flourish and achieve their full potential, embody pro-equity, anti-racism values (access, belonging, dignity, equity, justice, love, Ubuntu), and enjoy peace, prosperity, and possibility now and for generations to come.

We will go far because we will go together.

XII. References

1 Per its authorizing statute Chapter 43.06D RCW, the Office of Equity will help lead the development of a stand-alone Statewide Universal Access & Belonging Plan. The plan will support enterprise-wide investments in the following initial focus areas:

- Language access & belonging
- Disability access, accessibility & belonging
- Digital communications as it relates to language and disability access, accessibility & belonging
- Age discrimination (over 40) in employment
- Plain talk

The Plan will identify policies, procedures, and practices, and required legislation, including but not limited to:

- A sustainable statewide testing and certification system for spoken and sign language interpreters, and translators that all Washington state offices, agencies, departments, and commissions can use;
- Strategies for centering community voice and creating barrier-free access to and delivery of government services; and
- Guidance and technical assistance for Washington state offices, agencies, departments, and commissions to provide language, accessibility, and communication services.

2 In 2015, King County, Washington, identified 14 Determinants of Equity, the social conditions that each of us need to thrive, by which future progress toward becoming a fair and just community could be measured. Because these 14 social conditions are the same conditions that "everyone in Washington needs to flourish and achieve their full potential," the Office of Equity will adapt and focus on the same 14 social conditions, and identify key performance indicators to measure the state's progress toward becoming an equitable and just state.

3 In 2020, the Office of Equity added Digital Equity as a 15th PEAR Determinant of Equity. For purposes of the PEAR Plan & Playbook, digital equity includes, but is not limited to:

- Websites, applications, and other online content accessibility;
- Accessible digital documents and communications;
- Equitable access to:
  - Affordable, robust, reliable wi-fi/internet/broadband services and devices that meet the needs of the user;
  - Quality technical support;
  - Applications and online content designed to enable and encourage self-sufficiency, participation, and collaboration; and
  - Digital literacy training.
- Pro-equity in emerging digital technologies, such as facial recognition and artificial intelligence.

4 Tribal governments are sovereign nations with authority to govern their own people, lands, and resources. Tribal governments as sovereign nations have a unique relationship with the state and federal governments that is not based upon race. Federally recognized Tribes have the right and authority to regulate activities on their land independent from state government. However, tribes and the state frequently collaborate and cooperate in areas of mutual interest through consultation and government-to-government protocols as required by Chapter 43.376 RCW. It is of the utmost importance that Tribal sovereign nation status is honored throughout this process.

Tribes share responsibility for the health, safety, and welfare of their citizens and their communities with state and federal governments. Tribal Members, American Indians and Alaska Natives are dual citizens, as members of their Tribe and the State of Washington. Both governments share the responsibility to ensure equity and justice for AI/AN Washingtonians.

5 What are Values? - Ethics Sage

6 Equity vs Equality - What's the Difference? Milken Institute School of Public Health - The George Washington University

7 Love Definition & Meaning - Merriam-Webster

Washington State PEAR Plan & Playbook

2022–2027 Version 1.0

Access additional resources and trainings:

http://equity.wa.gov

OFFICE OF EQUITY
Washington State
EQUITY and JUSTICE for ALL

WASHINGTON STATE BOARD OF HEALTH

Date: March 13, 2024

To: Washington State Board of Health Members

From: Mindy Flores, Board Member

Subject: State Health Report Community Panel

Background and Summary:

RCW 43.20.100 requires the Washington State Board of Health (Board) to develop a State Health Report for the Governor by July 1 of each even-numbered year. The report includes “suggestions for public health priorities for the following biennium and such legislative action as it deems necessary.”

The State Health Report is not intended to describe or capture the state of health in Washington. The report is also not designed to inventory everything community groups, local health agencies, and state agencies are currently doing to address the health needs and priorities of communities across Washington. There are far too many initiatives and projects to capture in just one document. Instead, this report highlights recommended policy directions for the Governor’s consideration for the next legislative cycle.

The Board is organizing two community panels to help inform the 2024 State Health Report. In this first panel, the Board will hear directly from community representatives from the state's western side. These community panels are an opportunity for Board Members to hear the stories, experiences, and health priorities of different communities. The Board is particularly interested in hearing how topics identified for the 2024 State Health Report and related public health policies impact communities across the state. Information that panelists share during this discussion will help the Board align its State Health Report topics and recommendations with the needs of Washingtonians and other future work.

The March panel consists of four members representing organizations with deep relationships within communities and who have lived, or professional expertise related to the topic areas for the next State Health Report. The panelists also have an understanding of public health issues faced by communities in Washington, especially communities that historically have been institutionally underserved, overburdened, or disproportionately impacted by the social and structural determinants of health. The panelists include:

- Amanda Shi, Manager of Research and Evaluation, Tubman Center for Health and Freedom

(continued on the next page)

- Dominique Horn, Community Mobilization Coordinator, Southwest Washington Accountable Community of Health
- Molly Parker, Family Health Provider and Chief Medical Officer for Population Health, Jefferson Healthcare
- Nyka Osteen, Innovation Director, North Sound Accountable Community of Health

Today's informational briefing involves no formal Board action. Board staff will inform panel participants how their shared insights influenced the final report.

Staff

Molly Dinardo
Hannah Haag

2024 State Health Report

COMMUNITY PANEL

March 13, 2024

WASHINGTON STATE BOARD OF HEALTH

WASHINGTON STATE BOARD OF HEALTH

State Health Report (SHR)

- Requirement under Washington law (RCW 43.20.100).
- The State Board of Health (Board) must submit every two years (even-numbered years).
- Highlights suggestions for public health priorities and policy recommendations.
- Not intended to describe the state of health in Washington.
- Legislative report to highlight policy directions for the Governor's Office.

State Health Report
COMMUNITY PANELS

An opportunity for Board Members to hear directly from community members and organizations about how different issues and public health policies impact communities.

The Board has a special interest in hearing about:

- Maternal and Pregnant Person Health
- Health Justice and Culturally Appropriate Care
- Substance Use Prevention, Treatment, and Response
- Data Equity

Topic Selection

2024 State Health Report topics of interest were selected based on:

- The Board's authority granted by the Legislature
- Health Impact Review (HIR) completed by Board staff
- Past State Health Report topics and recommendations
- Feedback and interests provided by interested parties, and community members during Board rulemaking projects and related work
- Feedback and interests provided by Board Members

Reminder of Board Authority

- General Powers and Duties
- Rulemaking Authority
- Health Equity Work
- Consultation and Integration with the Department of Health
- Role in the Governmental Public Health System

Panel Structure and Agenda

- Panel Introduction
- Introductions and Overviews from Panelists
- Brief Break: Reflection and Processing Time
- Questions from Board Members and Panel Facilitators
- Discussion of Mutual Learnings
- Next Steps

Expectations and Norms

We hope for…

- Curiosity
- Deep listening
- A focus on connections
- Active participation
- An opportunity to learn together

We commit to…

- Understanding that panelists do not represent the entire community
- Creating a safe, respectful space for diverse experiences
- Learning from what we hear
- Being aware of our words: Avoid using idioms, acronyms, and phrases that others can misunderstand.
- Staying on topic and minding the time
- Equitable participation: Be mindful of how much space you are taking up in the discussion

Guiding Questions

- How do the topic(s) of Maternal and Pregnant Person Health, Health Justice and Culturally Appropriate Care, Substance Use, and Data Equity impact your community?
- Are there specific public health policies in these topic areas that are impacting your community (whether positive or negative)?
- Could you share a story with us that illustrates this impact?
- Are you engaged in any projects, efforts, community mobilization, etc., related to these topics or policy areas? If yes, could you provide examples?
- What is the most important thing for the Board to know about one or more of these topic areas in your community?

For Reflection

- What themes and connections are you hearing from panelists?
- Can these themes and connections be turned into public health policy?
- Does the Board have a role in what you are hearing? If not, what entity might have a role?
- Did you hear anything today that surprised you?
- What more do you hope to learn?
- Is there information we are missing?

Panelist Introductions

- **Amanda Shi**
  Manager of Research and Evaluation, Tubman Center for Health and Freedom
- **Dominique Horn**
  Community Mobilization Coordinator, Southwest Washington Accountable Community of Health
- **Molly Parker**
  Family Health Providers and Chief Medical Officer for Population Health, Jefferson Healthcare
- **Nyka Osteen**
  Director of Innovation, North Sound Accountable Community of Health

Break and Reflection

- What themes and connections are you hearing from panelists?
- Can these themes and connections be turned into public health policy?
- Does the Board have a role in what you are hearing? If not, what entity might have a role?
- Did you hear anything today that surprised you?
- What more do you hope to learn?
- Is there information we are missing?

Board Member Questions

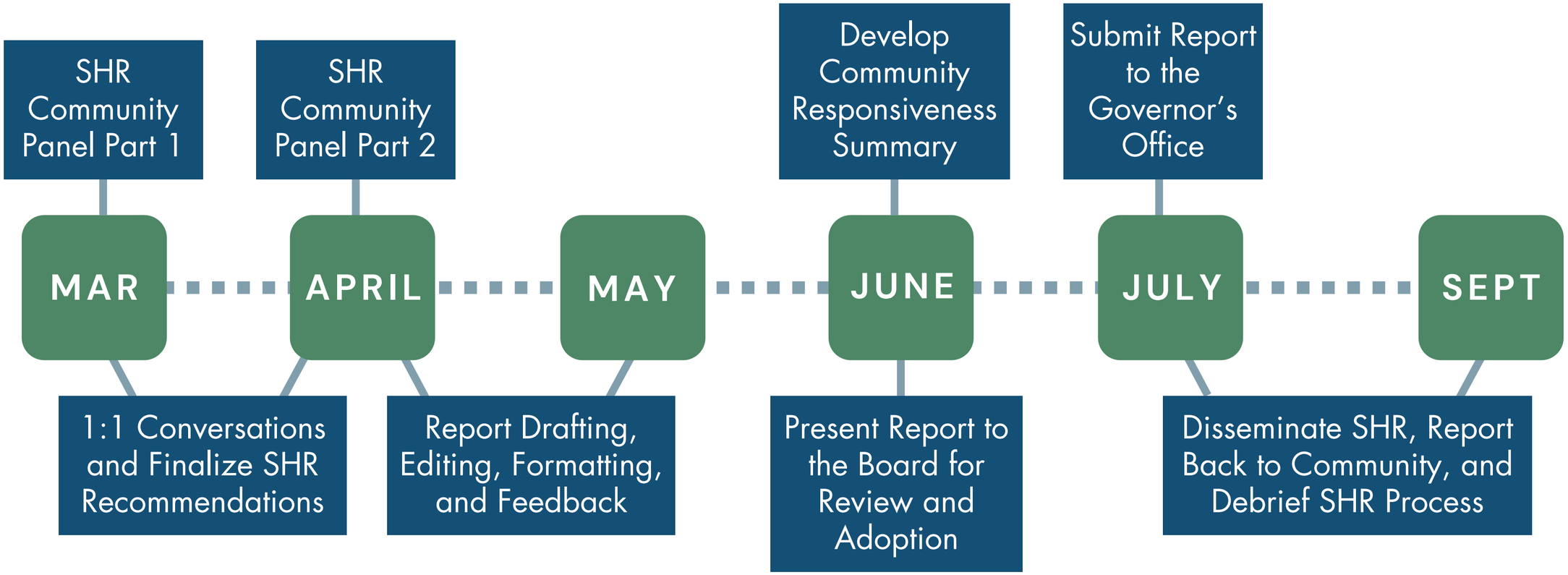
We hope for…

- Curiosity
- Deep listening
- A focus on connections
- Active participation
- An opportunity to learn together

We commit to…

- Understanding that panelists do not represent the entire community
- Creating a safe, respectful space for diverse experiences
- Learning from what we hear
- Being aware of our words: Avoid using idioms, acronyms, and phrases that others can misunderstand
- Staying on topic and minding the time
- Equitable participation: Be mindful of how much space you are taking up in the discussion

Next Steps and Timeline



WASHINGTON STATE BOARD OF HEALTH

THANK YOU

To request this document in an alternate format, please contact the Washington State Board of Health at 360-236-4110, or by email at **wsboh@sboh.wa.gov** | TTY users can dial 711

ACCESSIBILITY AND THE AMERICANS WITH DISABILITIES ACT (ADA)

- The Washington State Board of Health (Board) is committed to providing information and services that are accessible to people with disabilities. We provide reasonable accommodations, and strive to make all our meetings, programs, and activities accessible to all persons, regardless of ability, in accordance with all relevant state and federal laws.
- Our agency, website, and online services follow the Americans with Disabilities (ADA) standards, Section 508 of the Rehabilitation Act of 1973, Washington State Policy 188, and Web Content Accessibility Guidelines (WCAG) 2.0, level AA. We regularly monitor for compliance and invite our users to submit a request if they need additional assistance or would like to notify us of issues to improve accessibility.
- We are committed to providing access to all individuals visiting our agency website, including persons with disabilities. If you cannot access content on our website because of a disability, have questions about content accessibility or would like to report problems accessing information on our website, please call (360) 236-4110 or email wsboh@sboh.wa.gov and describe the following details in your message:
    - The nature of the accessibility needs
    - The URL (web address) of the content you would like to access
    - Your contact information

We will make every effort to provide you the information requested and correct any compliance issues on our website.

WASHINGTON STATE BOARD OF HEALTH

State Health Report Community Panel
SBOH Public Meeting - March 13, 2024

Amanda Shi

Amanda Shi, MPH, MPA
Research & Evaluation Manager
Tubman Center for Health & Freedom

Amanda Shi, MPH, MPA, is the Manager of Research & Evaluation at the Tubman Center for Health & Freedom (Tubman Health) where she leads the Community Health Research Institute. She graduated from the University of Washington with a BS (public health) and BA (political economy), then with an MPH (health policy & systems) and MPA (leadership & decision-making). As a lifelong Washingtonian, she has lived in Clark, King, and Snohomish County, working as a community advocate, CHW, researcher, and public health practitioner. She is passionate about centering community-directed research priorities, community solutions and equity-based decision-making to address health and health-related social needs in Washington State. She found a home to do this work when she first joined Tubman Health as an American Public Health Association/Kaiser Permanente Community Health Fellow. In her current role, she works collectively with community members and a vibrant team on community design, a process in which community members apply their diverse experiences and strengths to the process of reshaping systems of power that do not serve us, replacing them with community solutions. She loves being a part of building the Tubman Center for Health & Freedom, immersion in nature, leaning into movement and foods from home as medicine, and dreaming with community to reimagine wellness and our collective healing.

Dominique Horn

Dominique Horn
Community Mobilization Coordinator
Southwest Accountable of Community Health

Dominique is a natural community advocate and longtime member of the Vancouver, Washington community who works to impact change and elevate community voice. Dominique is a founding member of the Community Health Advocates and Peer Support Network of Southwest Washington (CHAPS), serves on the community Voice & Equity Committee & the Bridgeview resource center Board. Dominique earned her bachelor's in social work from Eastern Washington University. She enjoys spending time with her family, being in nature, and practicing photography.

SWACH is one of nine organizations leading the state's Healthier Washington Initiative in their regions. These Accountable Communities of Health (ACHs) are building a healthier Washington that meets their communities' unique needs. Southwest Washington Accountable Community of Health brings partners together to create equitable, sustainable systems change that reduces cost and improves whole-person wellness for all. Through our partnerships, we strive to build a healthier future in Southwest Washington - for everyone. Our work covers Clark, Klickitat, and Skamania counties in Southwest Washington. The region represents diverse languages, backgrounds, and lived experiences. Our goal is to ensure that each population in our community has access to the care and wellness they need.

WASHINGTON STATE BOARD OF HEALTH

State Health Report Community Panel
SBOH Public Meeting - March 13, 2024

Molly Parker

Molly Parker, MD, MPH
Population Health
Chief Medical Officer
Jefferson Healthcare

Dr. Molly Parker brings a love of problem solving to her career as family physician and Chief Medical Officer of Population Health at Jefferson Healthcare (JH). From starting Jefferson County's sexual assault nurse examiner program and expanding JH family planning services to coordinating community partners to create a new child care center, Dr. Parker's enthusiasm for solving health gaps in her rural community drives her work.

Dr. Parker grew up on a dairy farm in rural Wisconsin. She obtained degrees in biology, French, and education at Lawrence University and then worked as a molecular biologist Universities of Wisconsin and Washington and the Faculte de Medicine in Paris. She obtained a Master of Public Health degree in Maternal and Child Health at the University of Washington after which she worked as a youth health educator and in diabetes QI for a coalition of community health centers in King County. Dr. Parker completed her medical degree at the UW School of Medicine, family medicine residency training at the 45th Street Clinic and a high-risk obstetrics fellowship at Swedish Medical Center. After practicing family medicine in rural New Zealand and Whatcom County, WA, she settled in Port Townsend, Washington where she has worked for Jefferson Healthcare since 2010. Half of her work week is spent caring for her family practice panel and delivering babies. The other half is spent co-leading the population health department as it tackles improving the local social drivers of health including food insecurity, loneliness, early childhood education, and rural reproductive healthcare access. Dr. Parker continues to find joy in solving community health problems and equity gaps.

Nyka Osteen

Nyka Osteen, MPH
Director of Innovation
North Sound Accountable of Community Health

Nyka Osteen, MPH is the Director of Innovation with North Sound Accountable Community of Health (ACH). She grew up in Mount Vernon, WA and completed her bachelor's degree in microbiology and global health before earning her graduate degree in community-oriented public health from the University of Washington. Nyka has been a part of the North Sound ACH team since 2019, when she started as the Program Evaluation and Data Manager where she utilized her experience working in LGBTQ+ health and program evaluation. Now, as the Director of Innovation she is responsible for creating and supporting opportunities to incubate and foster growth of nontraditional ideas, leveraging partner assets and resources, and identifying key areas for innovative transformation. Having spent her entire childhood navigating the Medicaid system with chronic health conditions, and as an adult navigating these systems for her family who all struggle with chronic illness, disability, and mental health challenges, Nyka is familiar with the complexities and challenges that communities face while simply trying to survive.

WASHINGTON STATE BOARD OF HEALTH

Background on 2024 State Health Report Proposed Topics

Staff worked with State Board of Health (Board) Members to identify potential topics to include in its next State Health Report. Topics of interest identified for the report include:

- Maternal and Pregnant Person Health
- Health Justice and Culturally Appropriate Care
- Data Equity
- Substance Use, Prevention, Treatment, and Response
- Environmental Justice and Climate Change
- School Environmental Health and Safety
- Continuing Investments in the Public Health System (Foundational Public Health Services or FPHS)

These topics were selected based on:

1) The Board's authority granted by the Legislature,
2) Health Impact Reviews (HIRs) completed by Board staff,
3) Past State Health Report topics and recommendations,
4) Feedback and interests that Board Members, interested parties, and community members expressed during Board rulemaking projects and related work.

The Board recently convened two panels, one in November 2023 focusing on Environmental Justice and Climate Change and another in January 2024 focusing on Indoor Air Quality. Materials for these panels are available on the Board's meeting webpage.

The Board's next two panels will focus on Maternal and Pregnant Person Health, Health Justice and Culturally Appropriate Care, Substance Use, and Data Equity.

This handout includes brief definitions of these topics and background on the Board's authority and work in these areas.

Please note that this information has been compiled by Board staff and is meant to provide a high-level educational overview of these topics. It does not represent the Board's position or understanding of these topics.

Maternal and Pregnant Person Health

Why this topic?

The term "maternal health" typically refers to a person's physical, mental, emotional, and social health and well-being before, during, and after pregnancy.[1,2] However, as our understanding of the social determinants of health and their impacts on population health have evolved, the definition of maternal health shouldn't be limited to a particular

stage of life. An example of a framework that looks at maternal health more holistically is the life course or life cycle framework.

A life-course framework considers the impact that biological, social, environmental, and behavioral risk and protective factors have on an individual's health throughout their lifetime and how they interact and can contribute to health inequities across generations.[3,4] A life course framework considers the broad range of factors that impact a person's health. It also acknowledges that maternal, infant, and community health are intertwined. Supporting and promoting maternal health provides a strong foundation for population health.

While the Board's authority is limited to certain areas within maternal and pregnant person health, it is also charged with providing statewide leadership in developing and promoting policies that improve population health in Washington.

The Washington State Board of Health is part of Washington's Governmental Public Health System. Maternal, Child, and Family Health (MCFH) is a core service (or foundational program) within Foundational Public Health Services (FPHS).[5] MCFH is an essential public health service that must be provided to all residents in Washington, and it is a shared state and local responsibility. The Board's role in the system is specific to specifying the list of conditions for the screening of congenital disorders.[5] However, as a partner in the governmental public health system, it's important to acknowledge that the Board's role may extend beyond this (e.g., making policy recommendations, supporting state and local partners in their work, and completing Health Impact Reviews on legislation related to this topic).

Key Items to Highlight on this Topic:

- The Board's specific authority related to this topic includes:
    - Defining and adopting rules for testing all newborns in Washington for rare but treatable congenital disorders (RCW 70.83.050). These rules are under Chapter 246-650 WAC.
    - Adopting rules to establish standards, criteria, and timelines for screening and diagnostic tests for prenatal diagnosis of congenital disorders during pregnancy (RCW 48.21.244) (RCW 48.44.344) (RCW 48.46.375). The Board's rules also establish the standards that certain health insurance providers must follow when determining the medical necessity of screenings and diagnostic procedures. These rules are under Chapter 246-680 WAC.
- Between 2018 and 2024, Board staff have completed five Health Impact Reviews (HIRs) related to maternal and pregnant person health.
- In 2018, Substitute Senate Bill 6219 (SSB 6219) directed the Governor's Interagency Council on Health Disparities (Council) to conduct a literature review on disparities in access to reproductive healthcare in Washington State and to propose recommendations to reduce those disparities. Board Staff, specifically

the Health Policy Analyst team, led this research on behalf of the Council. The literature review identified 45 unique barriers to reproductive health access in Washington, grouped into three categories: Economic, Structural, or Social. The final report included 14 recommendations and was informed by the literature review findings, conversations with key informants, and reports authored by state agencies and community-based organizations.

Health Justice and Culturally Appropriate Care

Why this topic?

The term health justice builds on the concepts of health equity and social justice. It is broadly defined as "both a community-led movement for power building and transformational change and a community-oriented framework for health law scholarship."[6] Health justice focuses on the role that systemic factors, such as laws, policies, and institutions, play in creating, perpetuating, and dismantling health inequities within the healthcare and public health systems, and beyond. Health justice aims to recognize and build the power of individuals and communities directly impacted by health inequities to create and sustain conditions that support health and justice.[6,7]

Examples of conditions and factors contributing to health inequities and preventing progress toward health justice include barriers to providing and accessing culturally and linguistically appropriate services (CLAS). The goal of CLAS is to provide effective, equitable, understandable, and respectful quality care and services that are responsive to a person's diverse cultural health beliefs and practices, preferred languages, health literacy, and other communication needs.[8,9] Research has revealed the persistent gap in the provision of culturally and linguistically appropriate care and the impact it has on equity and health outcomes.[10,11] For example, the lack of culturally and linguistically appropriate care in the U.S. impacts the quality of care delivery for patients with limited English proficiency (LEP) by increasing time to treatment, reducing the quality of patient-provider communication, and increasing the length of hospitalization stays.[12–14]

The Board has the authority to serve as a public forum for policy and rulemaking development. The Board has also committed to promoting health equity and addressing racism as a public health crisis. The topics of health justice and promoting culturally and linguistically appropriate services are foundational to these efforts. Additionally, while health justice and culturally appropriate care are not explicitly core programs or foundational capabilities within FPHS, they are integral to providing and supporting foundational public health services across Washington.

Key Items to Highlight on this Topic:

- While the Board doesn't have explicit statutory authority related to health justice or culturally appropriate care, these topics are integral to our work. To meaningfully engage communities and ensure they are involved in this work,

particularly those who historically have been institutionally underserved and are disproportionately impacted by social determinants of health, the Board must work to remove systemic barriers to participation. This includes, but is not limited to:
    - Providing critical and timely public health information in culturally and linguistically appropriate formats,
    - Creating materials that are easy to understand,
    - Offering translation and interpretation support for meetings,
    - Having materials developed in alternative formats,
    - Identifying and creating meeting spaces that are accessible to community members.
- The U.S. Department of Health and Human Services' Office of Minority Health (OMH) developed national CLAS Standards to advance health equity, improve quality of services, and work toward eliminating health disparities. Any organization or agency can implement CLAS Standards to provide responsive services to the diverse population it serves. The Council was the first state entity to initiate work on language access in Washington and make recommendations to agencies for adopting CLAS standards in their work. The Council has also developed training and resources for agencies to learn about CLAS standards.
- The Board's 2022 State Health Report had several recommendations related to health justice and culturally appropriate care. These recommendations included removing barriers to health care insurance and improving access to culturally and linguistically appropriate health services.

Data Equity

Why this topic?

Data is an essential component of public health. Public health programs, their funders, program managers, and community partners all rely on data to make decisions about where resources are needed and should be allocated. However, to be a useful tool, data must accurately reflect communities and incorporate considerations of personal data privacy, data sovereignty, and prevent the misuse and misrepresentation of data that can cause harm to communities and individuals.

Data equity can be broadly defined as "a set of principles and practices to guide anyone who works with data...to use a lens of justice, equity, and inclusivity."[15] This equity lens should be applied when considering data collection, interpretation, distribution, and sharing.[15,16] It also challenges people and programs working with these data to consider the ways in which data can create and reinforce stereotypes, create stigma, exacerbate existing systemic inequities, or otherwise create harm, even if unintentional.

Data, specifically disaggregated data, are essential to achieving health equity. Disaggregated data can be broken down and analyzed by key demographic categories

such as age, race, ethnicity, sex, gender, disability, income, and veteran status.[17] Disaggregated data can also reveal inequities across and within groups and are instrumental for public health efforts to prevent and control diseases and conditions. These data also offer clearer indicators of community health and well-being, provide perspective into who is accessing public health programs, and whether services reach institutionally underserved or underrepresented communities.

Data are fundamental to making visible the longstanding inequities in the health care and public health system and their impacts on communities, particularly Black and Indigenous communities, and communities of color. Collecting these data in greater detail is essential to identifying and eliminating health inequities, undoing institutional racism, and advancing equity within public health and the broader governmental system. In addition, respect must be given to Tribal sovereignty, including data sovereignty. Tribes are sovereign nations that own the rights to their own stories and data. Governmental entities may only collect Tribal data with Tribal approval, consultation, and guidance.

While data equity and data disaggregation are not explicitly named as core programs or foundational capabilities within FPHS, data are a foundational component across all core programs and capabilities within Foundational Public Health Services (FPHS), from vital records and communicable diseases to assessment and policy development. Public health services cannot be effective without disaggregated data. Additionally, disaggregated data allows public health and governmental entities to provide more tailored, culturally relevant, linguistically appropriate, and effective services to communities. The Board's statutory authority is limited to certain areas within data equity, specifically data disaggregation for race, ethnicity, language, and other key demographic reporting in specific Board rules (notifiable conditions and vital statistics). However, as a partner in the governmental public health system, the Board has the opportunity to provide input, support, and recommendations on this topic.

Unfortunately, the governmental public health system is limited in the data it can collect. In many instances, governmental entities must follow federal statistical standards set by the Office of Management and Budget (OMB). This impacts how data can be collected, analyzed, and reported at the state and local levels. The Federal Office of Management and Budget (OMB) established the current minimum standards for collecting race and ethnicity data in 1997. The OMB standard consists of two reporting categories for ethnicity (Hispanic or Latino, Not Hispanic or Latino) and five for race (American Indian or Alaska Native, Asian, Black or African American, Native Hawaiian or Other Pacific Islander, and White). OMB does permit additional granularity where it is supported by sample size and if the additional detail can be aggregated back to the minimum standard set of race and ethnicity categories.

Until OMB revises its standards to require the uniform collection of detailed disaggregated data across federal, state, and local public health and governmental

agencies, the Washington governmental public health system will continue to face challenges in achieving data equity and health equity more broadly.

Key Items to Highlight on this Topic:

- The Board's authority related to this topic includes:
    - Adopting rules for the prevention and control of infectious and non-infectious diseases (RCW 43.20.050[2][f]). This includes establishing rules for notifiable conditions in Washington. The Board shares this authority with the Department of Health, which has the authority to establish requirements for some notifiable conditions within Chapter 246-101 WAC.
    - Adopting rules related to the statistical information to be collected on the confidential section of Washington State live birth and fetal death certificates (RCW 70.58A.020). Specifically, the Board has authority over data items related to birth and the manner of delivery necessary for statistical study (WAC 246-491-029).
- Recently, the Board adopted revisions to the Notifiable Conditions rule, Chapter 246-101 WAC. As part of the recent revisions, the Board included the requirement for reporting patient-identified race, ethnicity, and preferred language based on community feedback (WAC 246-101-011). These updated rules went into effect on January 1, 2023, and include 4 reporting categories for the patient's ethnicity (OMB standard plus "patient declined to respond" and "unknown"), 72 reporting categories for the patient's race (categories include and reaggregate to the OMB standard plus "other race", "patient declined to respond", and "unknown"), and 50 categories for the patient's preferred language.
- The Board's 2022 State Health Report had several recommendations related to the topic of data equity. These recommendations included improving public health's response to health inequities through data reform. In April 2023, the Board and Council submitted comments on the OMB's Initial Proposals for Updating Race and Ethnicity standards (SPD 15). The OMB Interagency Workgroup are reviewing feedback and comments on the proposal to put together final recommendations for revising OMB's race/ethnicity statistical standards for the Chief Statistician of the U.S. The OMB has a goal to revise SPD 15 by Summer 2024.

Substance Use, Prevention, Treatment, and Response

Why this topic?

Substance use is broadly defined as "the use of selected substances, including alcohol, tobacco products, drugs, inhalants, and other substances that can be consumed, inhaled, injected, or otherwise absorbed into the body."[18] These substances may also have the potential to cause dependence or other detrimental effects. If recurring substance use becomes harmful to a person's health and well-being and or they are

unable to control or stop their use of these substances, substance use can turn into substance use disorder (SUD). The CDC defines SUD as "a cluster of cognitive, behavioral, and physiological symptoms indicating that [an] individual continues using the substance despite harmful consequences."[19]

According to findings from the most recent National Survey on Drug Use and Health (NSDUH), more than 1 in 6 people in the U.S. aged 12 or older reported having a substance use disorder in the past year (SUD).[20] Substance use disorders are a pervasive public health issue in the U.S. and will continue to be until the root causes of the issue are addressed. Social and political determinants of health, such as economic instability, lack of affordable housing, high costs and inaccessibility of health and social services, experiences with systemic racism and generational trauma, and targeted product marketing, are all examples of factors that can contribute to and exacerbate substance use disorders. When discussing the topic of substance use, prevention, treatment, and response, it's essential to look at the topic holistically, and look at the range of conditions and factors that cause individuals to use and rely on substances.

Related to the topic of substance use, the Board's work has primarily focused on preventing the use of tobacco, nicotine, and vapor products (also known as commercial tobacco products), particularly among youth. In addition, the Board has supported policy recommendations related to the opioid crisis in Washington, and Board staff have completed Health Impact Reviews (HIRs) on opioid use disorder and alcohol concentration.

While the topic of substance use, prevention, treatment, and response isn't explicitly a core program or foundational capability within FPHS, it is a component of chronic disease, injury, and violence prevention, which is a core FPHS program. As a foundational program, chronic disease, injury, and violence prevention focuses on data, planning, and coordination of core programming and additional importance services (AIS). Additionally, the emphasis of this core FPHS program is *prevention*.

Key Items to Highlight on this Topic:

- The Board doesn't have explicit statutory authority related to the topic of substance use, prevention, treatment, and response. However, in 2019, the Board was directed by the Governor's Office to use its emergency rulemaking authority to ban the sale of all vapor products and flavors in Washington during an outbreak of e-cigarette or vaping associated lung injury (EVALI).[21] The emergency rule went into effect on October 10, 2019, for 120 days. When this emergency rule expired in March 2020, the Board subsequently adopted a second emergency rule but instead of a ban on all vapor products, just those containing vitamin E acetate. This is because vitamin E acetate was identified as the substance in products linked to the EVALI outbreak. The second emergency rule was also in place for 120 days. The Board then directed staff to begin the permanent rulemaking process to permanently ban vitamin E from all vapor

products in WA. This rule, WAC 246-80-012, was implemented by LCB in collaboration with the Board.

- Between 2018 and 2024, Board staff have completed twelve Health Impact Reviews (HIRs) related to substance use. One of the HIRs was on Engrossed House Bill 1074 (Chapter 15, Laws of 2019) which raised the minimum age of purchase for tobacco and vapor products in Washington to 21 years (also known as Tobacco 21).
- The Board's 2022 State Health Report included a recommendation to decrease youth use of tobacco, nicotine, and vapor products (also known as commercial tobacco products). Since its 2018 State Health Report, the Board has included a recommendation related to decreasing the use of commercial tobacco products. Past reports have also included recommendations related to addressing the opioid crisis in Washington.

To request this document in an alternate format or a different language, please contact the Washington State Board of Health at 360-236-4110
TTY users can dial 711
Requests can be made by email at **wsboh@sboh.wa.gov**

WASHINGTON STATE BOARD OF HEALTH

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Tubman Center for Health & Freedom

CENTERING COMMUNITY SOLUTIONS

VISIONS FOR OUR WELLNESS

March 2024

Tubman Health - Who We Are

Community - Directed Research & Design

The Importance of Starting with Community Design

- Visioning with providers and patients from community to **intentionally build from the ground up to sustain and support our healers**.
- Creating a community-designed model of care **centering culturally-appropriate, relational medicine**.
- Piloting of **responsive provider arrangements** within multi-disciplinary, integrative care teams.

Dreaming & Visioning for Our Care

Think of the Last Time You Went to a Clinic…

What was the experience like?

What did you see, hear, smell when you walked into the clinic?

How were you greeted?

Dreaming & Visioning for Our Care

What Care Can Be…

What Care Can Be

Atmosphere	Duality of privacy/openness with options to choose either/both (circular openness specifically)
	Engagement and interaction of multiple senses to create ease
Familiarity	Visual expression and community-based art that represents diverse experiences within the community
	Personalization and tailoring integrated into mixed use décor
Inclusivity & Accessibility	Consideration of intergenerational needs and accessible connection with each other in the space
	Different spaces for different needs / spaces meeting different needs

What Care Can Be

Natural Elements	Draping greenery that creates vibrancy
	Multi-use community garden, for grounding, for medicine, & for food
	Hydration of the body and the environment
	Natural and filtered lighting that inspires relaxation and safety
Sacred Space	Dedicated spaces for prayer and honoring of the ancestors
	Educational spaces that feature books, journaling, and other printed media
Spatial Flow	A range of comfortable places to sit & stop for a range of bodies
	Natural funneling that invites connection and slows down the pace
	Places to be held and loved, by self and by others

What Care Can Be…and Should Be

Whole Person Care from Birth to End of Life

- Truly culturally appropriate care recognizes:
  - Our bodies cannot be treated in parts and pieces
  - Mental, physical, emotional, and spiritual wellbeing are all interconnected and deeply related
  - The connection between self, family, and community
  - Healthcare does not happen in a silo
  - Ancestral medicines (CAM) work and are preferred
  - We are each experts in our own bodies

What Care Can Be…and Should Be

Health Justice

- Healing our healers – nourishing the strengths that already exist in our communities
- Looking into the systems and structures that govern how healthcare is provided – addressing utilization-focused incentives
- Access to our data & building community capacity to direct research priorities & carry out the research

North Sound ACH

North Sound ACH

Presented to Washington State Board of Health

by Nyka Osteen

March 13, 2024

Land Acknowledgement

We acknowledge, with humility, that the land of the North Sound ACH region today is the territory of the People of the Salish Sea. Their presence is imbued in the waterways, shorelines, valleys, and mountains of the traditional homelands of the Coast Salish People, since time immemorial.

Visit https://native-land.ca/ to learn more about the Indigenous land where you live, work, and play.

North Sound ACH

Transformation of Purpose

2017 Mission

North Sound ACH exists for the health benefit of North Sound residents through the creation and/or facilitation of policies, strategies and programs that improve health.

2023 Purpose

North Sound ACH exists to create a just and inclusive culture and the necessary conditions for all community members to thrive.

North Sound ACH

Leading with Belonging and Love

North Sound ACH intentionally focuses on how we relate to and work with each other. We are rooted in equity, belonging, and love, and are creating a practice of bridging in which our team approaches colleagues and partners within this paradigm.

North Sound ACH

Leading with Belonging and Love

Building upon systems thinking, targeted universalism, and belonging with the incorporation of well-being, vital conditions, grief, and love.

North Sound ACH

Supporting Culturally Responsive COVID-19 Response

- Culturally appropriate food boxes to farmworker families
- Vaccine and testing clinics for farmworkers
- Distributed over $2.5 million of PPE and other supplies

North Sound ACH

Current and Future Work

- **Birth Equity** - March 20 Learning Session
- **Jail Reintegration** - Supporting individuals leaving jail via community-based organizations led by those with lived experience
- **Care Coordination Hub** - Culturally responsive care coordination through CHWs, peer navigators, etc.
- **Opioid Use Disorder** - Stigma reduction education in rural communities
- **Data Equity** - Measuring well-being using Indigenous Indicators, Vital Conditions for Health, and Cantril's Ladder

North Sound ACH

North Sound ACH

"Partners working in concert to make a difference."

Vision:

North Sound Accountable Community of Health (North Sound ACH) exists to create a just and inclusive culture and the necessary conditions for all community members to thrive.

About North Sound ACH:

The North Sound region is home to more than one million people across Island, San Juan, Snohomish, Skagit, and Whatcom counties — urban and rural settings spanning across mountains, farmlands, urban settings, and islands in the Salish Sea. Our region is also on the traditional homelands of the Coast Salish who have inhabited this land since time immemorial: Lummi Nation, Nooksack Tribe, Upper Skagit Tribe, Samish Indian Nation, Swinomish Indian Tribal Community, Stillaguamish Tribe of Indians, Tulalip Tribes, and Sauk-Suiattle Indian Tribe.

North Sound ACH was established in 2015 to foster collaborative learning, planning, and decision-making, crossing traditional jurisdictional boundaries, and looking upstream to tackle issues that impact health, believing that people in the region are more connected than separate.

What is an ACH?

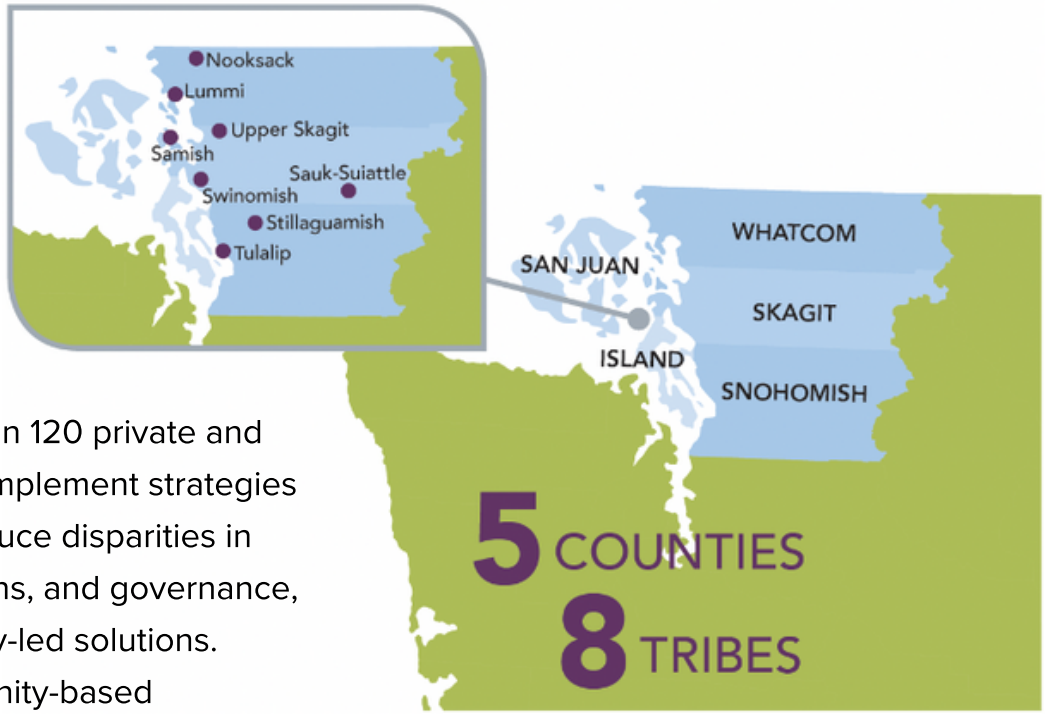
Nine ACHs cover Washington state, each dedicated to serving a specific region. ACHs share a common approach to improving the health of their communities and transforming healthcare delivery, by collaborating with diverse partner organizations across sectors to create and invest in innovative and sustainable community-led solutions.

Washington's Accountable Communities of Health (ACH), a national model, is an integral part of Washington's Medicaid transformation efforts.

Contact Us! NorthSoundACH.org Team@NorthSoundACH.org

What We Do

At North Sound ACH,



we work alongside more than 120 private and public sector partners – to implement strategies that advance equity and reduce disparities in systems operations, decisions, and governance, investing in local, community-led solutions. Partners range from community-based organizations, schools, public health agencies, behavioral health clinics, fire and EMS, county governments, and health systems.

- We honor tribal sovereignty and learn from their experience in holistic, intergenerational approaches to well-being, healing, and stewardship.
- The work is place-based and centered around community and lived experience.
- We use targeted universalism as a framework and reject zero-sum approaches to advancing equitable well-being and addressing systemic racism.
- Belonging is both a vital condition and a practice. We endeavor to ensure everyone can see themselves in the ongoing process of co-creating equitable well-being.

NorthSoundACH.org
Team@NorthSoundACH.org
About the Collaborative Action Network
Newsletter Sign Up

North Sound ACH

Scan QR code to save and view the links!

Our Priorities

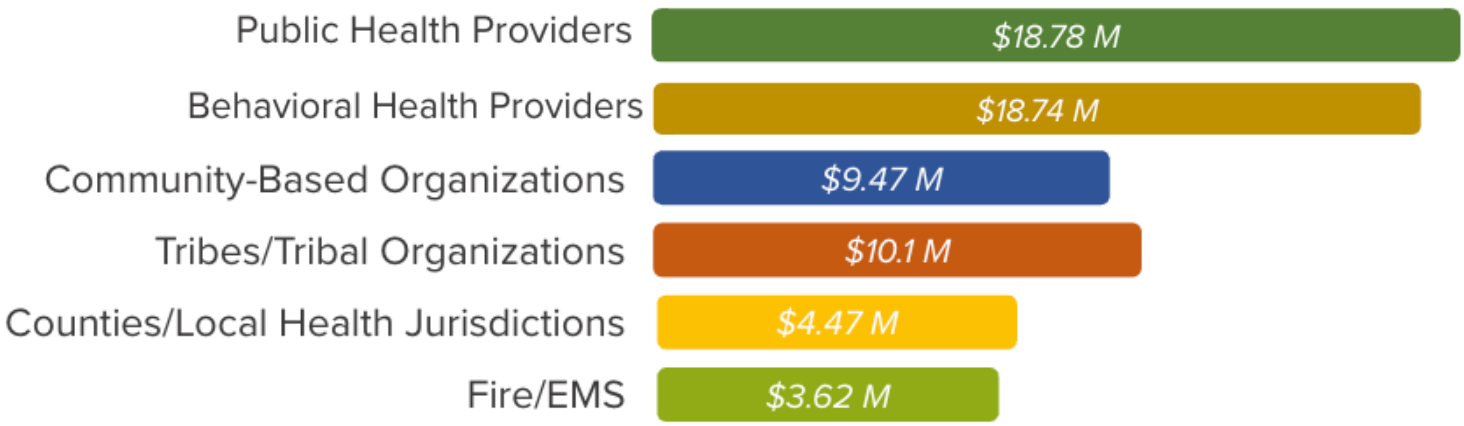
01 **Tribal and Equity Learning**
All contracted partners take part in learning opportunities, resulting in further opportunities to partner, increased understanding and demand for equity.

02 **Beyond Medicaid, Beyond Health Care**
North Sound ACH is committed to long term, sustainable change, leveraging the opportunity that MTP 2.0 brings, and building other resources to support the work of community partners.

03 **Relationship Building**
The Operational focus is on building the capacity of partners and deepening relationships, which continues to result in new and stronger collaborations

04 **Commitment to Equity**
Agreement to this commitment occurs at all levels – operations, governance and with contracted partners. Learning, capacity building and strategic recruitment uses an eye toward equity and social justice.

Medicaid Waiver Funds Invested in Regional Partners from 2016-2023



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North Sound ACH

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Molly Parker, MD, MPH

she/her

Jefferson County

Jefferson Healthcare

Jefferson Healthcare


Indian Reservation
Strait of Juan De Fuca
Hoko River
112
Ozette Lake
Sol Duc River
Lake Crescent
Lake Crescent
101
Dungeness River
Strait of Juan De Fuca
WHIDBEY ISLAND
Skagit Bay
Saratoga Passage
CAMANO ISLAND
Tulalip Reservation
Possession Sound
Puget Sound
Elwha River
Bogachiel River
OLYMPIC NATIONAL PARK
Hoh River
WASHINGTON
OLYMPIC NATIONAL PARK
Dosewallips River
Clearwater River
Queets River
Hood Canal
BAINBRIDGE ISLAND
Seattle
Bremerton
Boeing Field/King County International Airport
Quinault Reservation
Quinault Lake
Lake Cushman
Quinault River
VASHON ISLAND
Des Moines
Federal Way
Snohomish County Airport (Paine Field)
Edmonds
405
Shoreline
Burien
3

JEFFERSON COUNTY

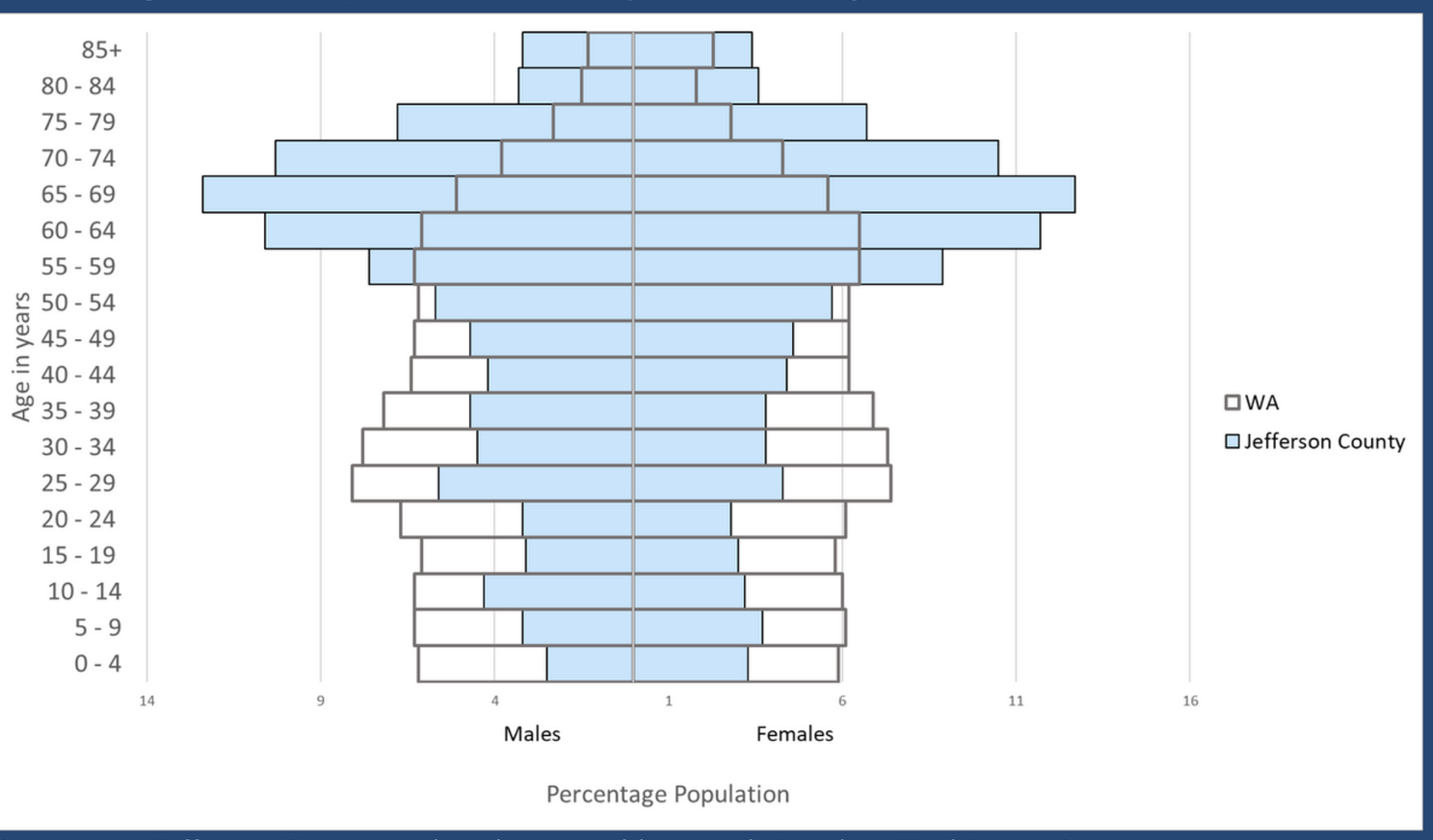
COMMUNITY PROFILE

POPULATION: 31,825

OVERVIEW*

Jefferson County is located at the western-most edge of the state of Washington. With a **population of 31,825** in 2020, Jefferson County has a **median age of 59**. The region is economically suppressed, with a population of **13.6% living below the poverty line** (compared to 10.2% in Washington State).

Population in Jefferson County vs. Washington State (2020)*



On average, Jefferson County tends to have an older population than Washington State.

As of 2020*...

4.9%
of the population in Jefferson County immigrated there

and

5.0%
speak a language other than English at home

Compared to 14.5% and 20.0%, respectively, in WA

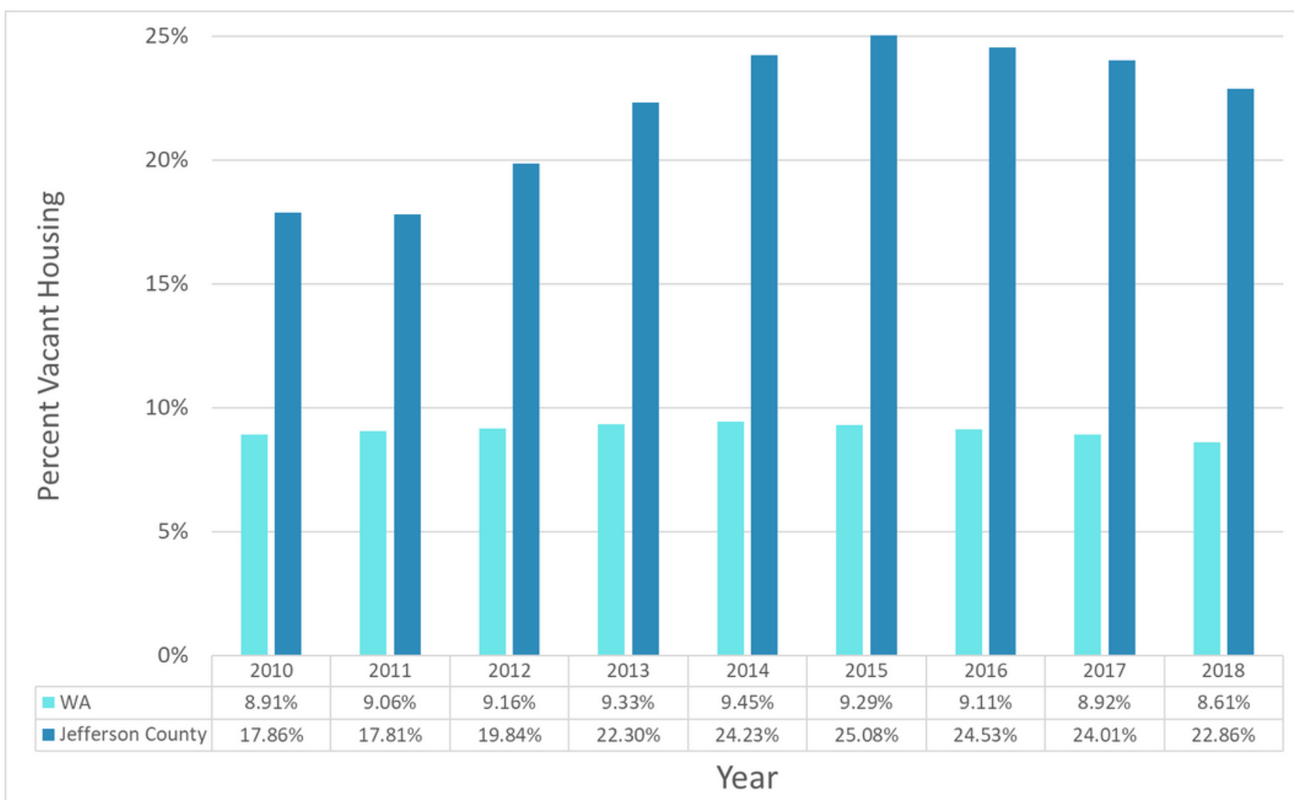
$362,300
Median housing price (in 2020 dollars)*

$57,693
Median household income (in 2020 dollars)*

81 years
life expectancy of the population in Jefferson County (compared to 80.3 years in WA)***

25.6%
of children under the age of 18 are living in poverty (compared to 12.6% in WA)*

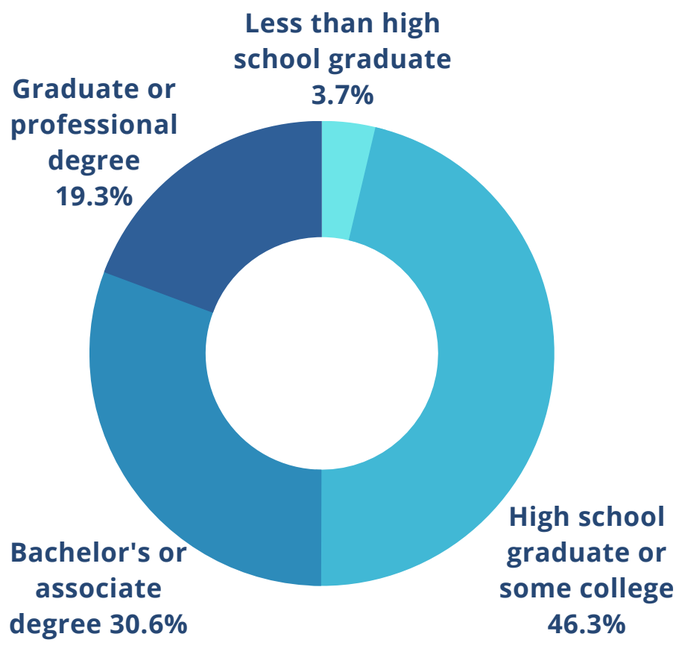
Percent of Vacant Housing in Jefferson County vs. Washington State*



	2010	2011	2012	2013	2014	2015	2016	2017	2018
WA	8.91%	9.06%	9.16%	9.33%	9.45%	9.29%	9.11%	8.92%	8.61%
Jefferson County	17.86%	17.81%	19.84%	22.30%	24.23%	25.08%	24.53%	24.01%	22.86%

On average, Jefferson County tends to have a greater percentage of vacant housing units than Washington State.

Of the population 25 years and over (2020)*...



Of the population of workers 16 years and over...

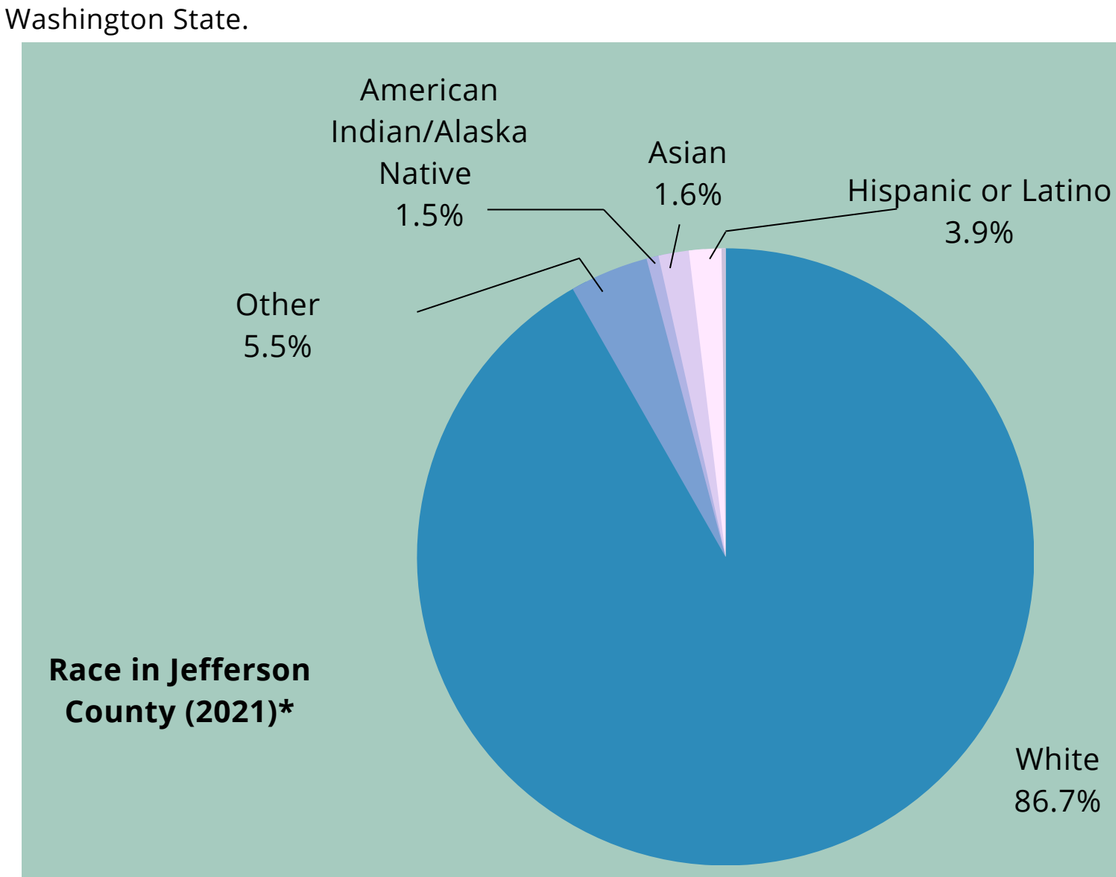
9.4%
Unemployed
(8.4% in WA)^

6.5%
Disabled
(8.0% in WA)*

40.2%
Working (full or part time)
(60.6% in WA)*

36.7%
Retired
(21.5% in WA)*

Race in Jefferson County (2021)*



Data: *United States Census Bureau (Census Designated Places), **Epic, JH Tableau Server, ***Washington State Employment Security Department, ****Institute for Health Metrics and Evaluation, ^County Health Rankings and Roadmaps

Ensuring Access to Robust Maternity Care in Rural Communities

Ensuring a comprehensive maternal health program not only requires the support of our community, but also those in office. Jefferson Healthcare is committed to providing high quality, robust obstetrical and maternal healthcare to our community. In order to protect this service, we believe it's integral to support legislation that prioritizes high quality and accessible care, and ensures long-term financial sustainability.

Maternal healthcare is struggling in rural communities across the country

The ability to preserve maternity programs is becoming increasingly difficult in rural areas across the country. Financial costs and staffing shortages, combined with decreasing birth rates, have impacted rural hospitals' ability to keep these programs afloat. A 2020 study from the American Hospital Association found that nearly half of all rural hospitals did not offer OB services, stranding more than 2.2 million child-bearing patients in maternity care deserts nationwide. As Jefferson Healthcare remains focused on protecting its OB program, there are important factors impacting our hospital to keep in mind.

Mirroring national trends, birth rates have been decreasing in Jefferson County. Serving a population with the lowest birth rate in the State, Jefferson Healthcare delivered 96 babies in 2022, and there have been only 23 births in the first five months of 2023. Despite the low number of births, maternity care still requires substantial investment. At Jefferson Healthcare, it's crucial our obstetrical program does everything within its power to mitigate risk and ensure patient safety. The significant cost of required high-level training, in addition to the maintenance of adequate equipment and supplies has caused Jefferson Healthcare's OB program to operate at a deficit for many years, with losses only increasing.

Protecting maternity care is critical

Jefferson Healthcare is committed to maintaining access to the full suite of maternity care services for our community. In order to maintain these services, we need policy solutions that protect the financial sustainability as well as ensure the highest quality and safety for our patients. Specifically, we are supportive of legislation that increases reimbursement rates for low birth volume hospitals to cover the costs for providing care, innovative solutions for competency management, as well as legislation that addresses the maternity services staffing crisis.

For more information please contact Dunia Faulx at dfaulx@jeffersonhealthcare.org

Jefferson Healthcare
jeffersonhealthcare.org

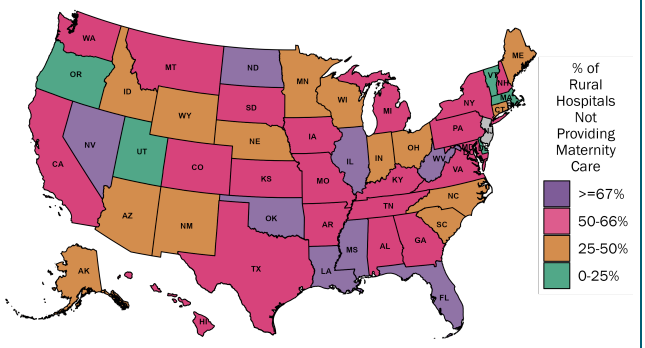
CENTER FOR HEALTHCARE QUALITY & PAYMENT REFORM

THE CRISIS IN RURAL MATERNITY CARE

Most Rural Hospitals in the U.S. No Longer Deliver Babies

Fewer than half (46%) of the rural hospitals in the U.S. currently offer labor and delivery services, and in 8 states, less than one-third do. Over the past decade, more than 200 rural hospitals across the country have stopped delivering babies.

Proportion of Rural Hospitals Without Labor and Delivery Services

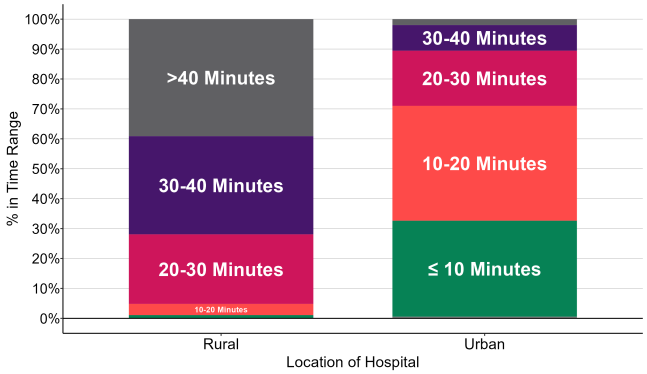


Maternity Care is Far Away for Mothers in Many Rural Communities

If the closest hospital does not offer labor and delivery services, a pregnant woman may have to travel to a different community to deliver her baby. In most urban areas, the travel time to a hospital with labor and delivery services is under 20 minutes, but in rural areas, the travel time is likely to be at least 30 minutes, and it is often 40 minutes or more.

There is a higher risk of complications and death for both mothers and babies in communities that do not have local maternity care services. Women are less likely to obtain adequate prenatal and postpartum care when it is not available locally.

Travel Time from Non-Maternity Care Hospitals to Closest Hospital with Maternity Services

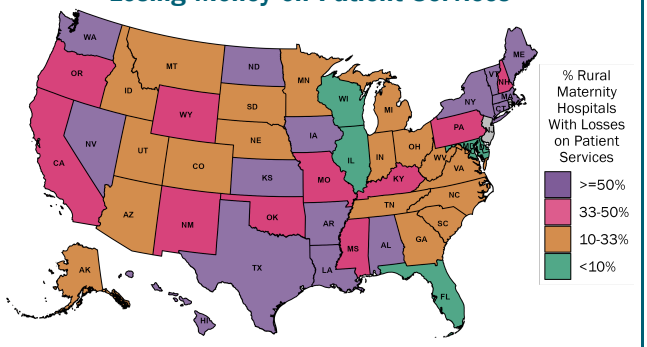


Many More Rural Communities Are at Risk of Losing Maternity Care

Hundreds of additional communities are at risk of losing maternity care because of the financial challenges rural hospitals are facing. Rural hospitals typically lose money on obstetric care, so if a hospital can't make enough money on other services to offset those losses, it may be forced to eliminate maternity care in an effort to keep the hospital from closing entirely.

More than 1/3 of the rural hospitals that still have labor & delivery services have been losing money on patient services, so their ability to continue delivering maternity care is at risk.

Proportion of Rural Maternity Care Hospitals Losing Money on Patient Services

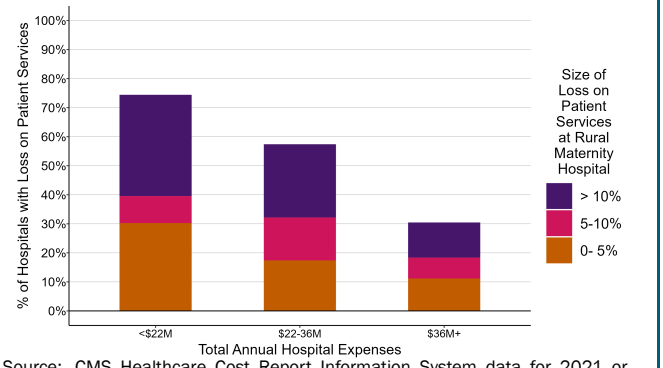


Source: CMS Healthcare Cost Report Information System data for the most recent fiscal year available (2021 or 2022).

Small Communities Are Most at Risk

Smaller rural hospitals are more likely to be losing money on patient care services than larger hospitals, and they are more likely to experience large losses. More than half of small rural maternity care hospitals lost money in 2021-22. In most cases, if these hospitals are forced to eliminate maternity care, community residents would have to travel more than 40 minutes to reach a hospital with obstetric services.

Small Rural Maternity Care Hospitals Are Experiencing the Greatest Financial Problems



Source: CMS Healthcare Cost Report Information System data for 2021 or 2022 at rural hospitals providing labor and delivery services in 2023.

RURAL MATERNITY CARE AT RISK

State	Total Number of Rural Hospitals	Rural Hospitals Without Obstetric (Labor & Delivery) Services			Rural Hospitals Still Providing Obstetric Services		
		% Rural Hospitals without OB Services	Number without OB Services	Median Minutes Driving Time to Hospital with OB Services	Number of Hospitals with OB Services	% with Losses on Patient Services[1]	Median Minutes to Alternative OB Hospital
Connecticut	3	33%	1	31	2	100%	33
Vermont	13	23%	3	34	10	80%	43
Nevada	13	69%	9	58	4	75%	>90
Maine	25	36%	9	48	16	69%	37
Hawaii	12	50%	6	47	6	67%	68
Kansas	104	58%	60	32	44	66%	36
New York	51	55%	28	38	23	61%	44
Arkansas	49	59%	29	39	20	60%	41
Texas	162	57%	93	37	69	58%	40
North Dakota	39	79%	31	58	8	57%	77
Iowa	93	61%	57	31	36	56%	30
Washington	40	50%	20	40	20	55%	47
Louisiana	53	75%	40	37	13	54%	35
Alabama	52	65%	34	36	18	50%	40
Massachusetts	5	20%	1	24	4	50%	76
Oklahoma	78	68%	53	39	25	48%	41
Missouri	57	53%	30	35	27	44%	43
California	56	52%	29	49	27	41%	45
Kentucky	72	56%	40	32	32	41%	35
New Hampshire	17	53%	9	34	8	38%	47
Wyoming	23	30%	7	58	16	38%	53
Oregon	32	22%	7	38	25	36%	45
Pennsylvania	42	60%	25	39	17	35%	41
Mississippi	74	69%	51	35	23	35%	34
New Mexico	28	36%	10	57	18	33%	55
Idaho	30	47%	14	37	16	31%	38
South Carolina	25	48%	12	35	13	31%	42
Montana	55	60%	33	52	22	29%	57
Minnesota	95	46%	44	31	51	27%	29
Alaska	17	35%	6	>90	11	25%	>90
South Dakota	48	60%	29	43	19	25%	54
Tennessee	53	55%	29	33	24	25%	36
Utah	21	5%	1	35	20	25%	37
West Virginia	28	71%	20	44	8	25%	37
Georgia	67	61%	41	37	26	23%	39
North Carolina	53	34%	18	32	35	23%	38
Nebraska	71	45%	32	32	39	21%	29
Michigan	64	50%	32	36	32	19%	42
Indiana	53	38%	20	32	33	12%	30
Ohio	70	47%	33	28	37	11%	29
Arizona	27	48%	13	46	14	10%	68
Colorado	42	52%	22	46	20	10%	47
Virginia	29	66%	19	40	10	10%	49
Illinois	72	71%	51	32	21	10%	34
Wisconsin	75	44%	33	29	42	7%	29
Delaware	2	0%	0		2	0%	26
Florida	21	86%	18	50	3	0%	50
Maryland	4	50%	2	48	2	0%	59
New Jersey	0						
Rhode Island	0						

[1] Percentage of hospitals with OB services that had a negative margin (loss) on (all) patient services in the most recent year available.

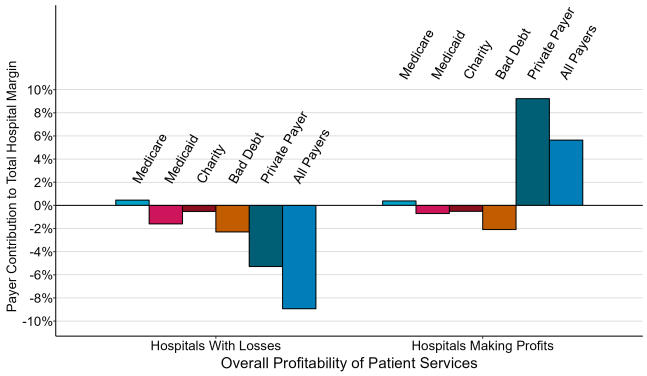
Data current as of May 2023

Losses Are Due to Inadequate Payments from Private Payers

As shown below, the primary reason small rural maternity care hospitals are losing money is that private insurance plans pay them less than what it costs to deliver many types of services to patients, not just maternity care. Although the hospitals are also losing money on uninsured patients and Medicaid patients, the losses from private payers have the biggest impact on their overall profit margins.

Conversely, small rural maternity hospitals that avoid losses are able to do so because their payments from private health plans not only cover the costs of services (of all types) to the patients with private insurance but also offset the hospitals' losses on services to uninsured and Medicaid patients.

Payer-Specific Profits/Losses on Patient Services at Small Rural Maternity Care Hospitals



The bars show the median profit or loss on both maternity and non-maternity services delivered to patients with each type of insurance.

Source: CMS cost report data for 2021-2022 at rural hospitals with less than $35 million in total expenses that deliver obstetric care.

Actions Needed to Preserve and Strengthen Rural Maternity Care

Significant changes in payments from both private and public payers are needed to resolve the financial problems facing rural hospitals before even more maternity care services are lost.

Require That Health Insurance Payments Cover the Actual Cost of Rural Maternity Care

A hospital cannot provide maternity care for its community if private health plans and state Medicaid programs do not pay enough to cover the cost of the services. It is often assumed that low Medicaid payments and uninsured patients are the reasons hospitals lose money on maternity services, but over 40% of births in rural communities are paid for by private health plans, so inadequate payments from private payers also threaten the viability of rural maternity care.

Health plans should be required to pay amounts that cover the cost of: (1) perinatal care services from physicians and midwives; (2) assistance during labor and delivery from appropriately-trained nurses; (3) anesthesia services (such as when C-Sections are needed); and (4) telemedicine assistance from specialists for complex cases. Payment amounts must be higher in communities that have difficulty attracting staff, and payments must also be higher in communities with smaller numbers of births to ensure that revenues cover the fixed costs of services.

In small rural communities, obstetric services will often be delivered by family physicians rather than obstetricians. Since maternity care will only be a portion of the services these physicians provide, health plans must pay adequately for all of the primary care services they deliver, not just perinatal care.

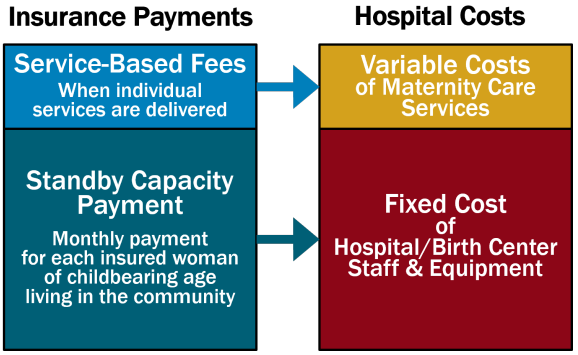
Rural Health Clinics (RHC) can serve as an important way of supporting maternity care as well as primary care services in rural areas. However, Medicare staffing and productivity standards need to be revised so that RHCs are not penalized for hiring family physicians who spend time delivering maternity care. Private insurers should also be required to pay amounts based on the clinic's costs, just as Medicare does for its patients.

Create Standby Capacity Payments to Support the Fixed Costs of Maternity Care

The financial challenges of delivering maternity care are caused not only by the inadequate *amounts* paid by insurance plans, but by the problematic *method* used to pay for services. Currently, a rural hospital is only paid when it actually provides a service. However, a small hospital must be staffed and ready to deliver a baby at all times, even though there will be no deliveries at all on many days. As a result, when there are fewer pregnancies than expected, the hospital will lose money, even if payments would have been adequate for a larger number of births.

A better approach is for private insurers and Medicaid to pay an annual ***Standby Capacity Payment*** to the hospital for each insured woman of childbearing age living in the community. This would provide more predictable revenue to cover the fixed costs of maternity care than a purely fee-based system can. The hospital should still receive Service-Based Fees for individual services, but the amounts should be based on the variable costs of the services. More details on this approach are available in *A Better Way to Pay Rural Hospitals*.

A Better Way to Pay for Maternity Care



Require Adequate Payments for All Services to Prevent Hospital Closures

Even if payments are adequate to cover the cost of *maternity care* services, a rural hospital must also receive adequate payments for other essential services, such as its emergency department, or the hospital may not be able to stay open at all. Many small rural hospitals are at risk of closing completely because of the overall financial losses they have been experiencing. *Rural Hospitals at Risk of Closing* provides more information on this problem and how to address it.

WASHINGTON STATE BOARD OF HEALTH

Date: March 13, 2024

To: Washington State Board of Health Members

From: Patty Hayes – Board Chair

Subject: Petition for Rulemaking –WAC 246-290-220, Group A Public Water Supplies, Drinking Water Materials and Additives

Background and Summary:

The Administrative Procedure Act (RCW 34.05.330) allows any person to petition a state agency for the adoption, amendment, or repeal of any rule. Upon receipt of a petition, the agency has sixty days to either (1) deny the petition in writing, stating the reasons and, as appropriate, offer other means for addressing the concerns raised by the petitioner, or (2) accept the petition and initiate rulemaking.

On February 12, 2024, the Washington State Board of Health (Board) received a rulemaking petition to amend its Group A Public Water Supplies standards (chapter 246-290 WAC), specifically WAC 246-290-220.

The Board has the authority under RCW 43.20.050 to adopt rules for group A public water systems as defined in RCW 70A.125.010. Chapter 246-290 WAC establishes the standards for these water systems related to their design, construction, sampling, management, maintenance, and operation practices. The purpose of these rules is to define basic regulatory requirements and to protect the health of consumers using public drinking water supplies.

WAC 246-290-220 requires Group A public water systems to test and certify for conformance with NSF/ANSI Standards 60 and 61 for:

- treatment chemicals added to public drinking water supplies; and
- public water system components in substantial contact with potable water such as water pipes, tank coatings or liners, and treatment system media.

Washington State, along with most other U.S. states and Canadian provinces, require this certification to ensure the safety of chemicals and products used in public water systems. The testing and certification help to ensure that the additives do not contain materials that can cause a health risk and that the system component materials will not leach chemicals that can cause a health risk.

The petitioner requests that the Board amend WAC 246-290-220 to include a new subsection related to water fluoridation. The petition proposes adding language to the rule that removes the Board's endorsement of fluoride in public water systems and

(continued on the next page)

provides recommendations for reducing fluoride exposure for pregnant mothers, infants, and children under 6 years of age (page 14).

The petitioner included multiple attachments to their petition in support of their position regarding the potential impacts of water fluoridation on fetus, infant, and child health. Among other things, petitioner refers to fluoride as a drug. Note that Washington case law provides that fluorides in public drinking water are not drugs. See the case of *Protect the Peninsula's Future v. City of Port Angeles*, 175 Wn. App. 201, 215 (2013).

I have invited Andrew Kamali, Board Staff, to provide more information about the petition and the Board's options for responding.

Recommended Board Actions:

The Board may wish to consider one of the following motions:

The Board declines the petition for rulemaking to amend WAC 246-290-220 for the reasons articulated by Board Members. The Board directs staff to notify the petitioner of the Board's decision.

OR

The Board accepts the petition for rulemaking to explore the proposed amendment to WAC 246-290-220 to consider additional language related to water fluoridation. The Board directs staff to notify the requestor of its decision and to file a CR-101, Preproposal of Inquiry, to further evaluate the request and possible rule change.

Staff

Andrew Kamali

Washington State Board of Health
Policy & Procedure

Policy Number:	**2005-001**
Subject:	**Responding to Petitions for Rule-Making**
Approved Date:	**November 9, 2005 (revised August 13, 2014)**

Policy Statement

RCW 34.05.330 allows any person to petition a state agency to adopt, repeal, or amend any rule within its authority. Agencies have 60 days to respond. The agency can deny the request—explaining its reasons and, if appropriate, describing alternative steps it is prepared to take—or it must initiative rule-making. If a petition to repeal or amend a rule is denied, a petitioner can appeal the agency's decision to the Governor.

This policy defines who must be notified and consulted when the Board is petitioned, who may respond on behalf of the Board, and whether Board action is required.

- **Board Response**: When the Board receives a written petition for rule-making within its authority that clearly expresses the change or changes requested, the Board will respond within 60 days of receipt of the petition. The response will be made at the direction of the Board. The response will be in the form of a letter from the Chair denying the petition or informing the petitioner the Executive Director has been directed to initiate rule-making.

- **Consideration of the Petition:** The Chair may place a petition for rule-making on the agenda for a Board meeting scheduled to be held within 60 days of receipt of the petition. Alternatively, if the Board does not have a regular meeting scheduled within 60 days of receipt of the petition, or if hearing the petition at the next regular meeting would defer more pressing matters, the Chair shall call a special meeting of the Board to consider the petition for rulemaking.

Procedure

- **Notifications:** Board staff, in consultation with the Executive Director, will respond to the petitioner within three business days acknowledging receipt of the petition and informing the petitioner whether the request is clear. The Executive Director or staff will notify Board members that a petition for rule-making has been received and will be brought to the Board for consideration at the next regularly scheduled board meeting or will be considered at a special meeting. If

no regular meeting is scheduled before the 60-day response deadline, or if the agenda for the regular meeting cannot accommodate the petition, the Executive Director will notify the Chair of the need to schedule a special board meeting for the purposes of considering the petition. Upon Board action on the petition, the Executive Director shall assure Board members receive electronic copies of the final petition response.

- **Appeals:** If a petitioner appeals the Board's decision to deny a petition to the Governor, the Executive Director will inform the Board of the Governor's action on the appeal at the next scheduled Board meeting.

- **Consultation:** The Executive Director and Board staff will gather background information for the Board's use when it considers the petition. In this regard, the Executive Director will consult with the Board member who sponsored the most recent revisions to the rule being challenged or the appropriate policy committee. The Executive Director may also consult with appropriate representatives of the implementing agency or agencies, and may consult with stakeholders as appropriate.

PDF **WAC 246-290-220**

Drinking water materials and additives.

(1) All materials shall conform to the ANSI/NSF Standard 61 if in substantial contact with potable water supplies. For the purposes of this section, "substantial contact" means the elevated degree that a material in contact with water may release leachable contaminants into the water such that levels of these contaminants may be unacceptable with respect to either public health or aesthetic concerns. It should take into consideration the total material/water interface area of exposure, volume of water exposed, length of time water is in contact with the material, and level of public health risk. Examples of water system components that would be considered to be in "substantial contact" with drinking water are filter media, storage tank interiors or liners, distribution piping, membranes, exchange or adsorption media, or other similar components that would have high potential for contacting the water. Materials associated with components such as valves, pipe fittings, debris screens, gaskets, or similar appurtenances would not be considered to be in substantial contact.

(2) Materials or additives in use prior to the effective date of these regulations that have not been listed under ANSI/NSF Standard 60 or 61 may be used for their current applications until the materials are scheduled for replacement, or that stocks of existing additives are depleted and scheduled for reorder.

(3) Any treatment chemicals, with the exception of commercially retailed hypochlorite compounds such as unscented Clorox, Purex, etc., added to water intended for potable use must comply with ANSI/NSF Standard 60. The maximum application dosage recommendation for the product certified by the ANSI/NSF Standard 60 shall not be exceeded in practice.

(4) Any products used to coat, line, seal, patch water contact surfaces or that have substantial water contact within the collection, treatment, or distribution systems must comply with the appropriate ANSI/NSF Standard 60 or 61. Application of these products must comply with recommendations contained in the product certification.

(5) The department may accept continued use of, and proposals involving, certain noncertified chemicals or materials on a case-by-case basis, if all of the following criteria are met:

(a) The chemical or material has an acknowledged and demonstrable history of use in the state for drinking water applications;

(b) There exists no substantial evidence that the use of the chemical or material has caused consumers to register complaints about aesthetic issues, or health related concerns, that could be associated with leachable residues from the material; and

(c) The chemical or material has undergone testing through a protocol acceptable to the department and has been found to not contribute leachable compounds into drinking water at levels that would be of public health concern.

(6) Any pipe, pipe fittings, plumbing fittings, fixtures, solder, or flux used in the installation or repair of a public water system shall be lead-free:

(a) This prohibition shall not apply to leaded joints necessary for the repair of cast iron pipes; and

(b) Within the context of this section, lead-free shall mean:

(i) No more than a weighted average of twenty-five one-hundredths of one percent lead, calculated in accordance with 42 U.S.C. 300g-6 654(d)(2); and

(ii) No more than two-tenths of one percent lead in solder and flux.

(7) Exceptions to the lead-free requirements of subsection (6) of this section include:

(a) Pipes, pipe fittings, plumbing fittings, or fixtures, including backflow preventers, that are used exclusively for nonpotable services such as manufacturing, industrial processing, irrigation, outdoor watering, or any other uses where the water is not anticipated to be used for human consumption; or

(b) Toilets, bidets, urinals, fill valves, flushometer valves, tub fillers, fire hydrants, shower valves, service saddles, or water distribution main gate valves that are two inches in diameter or larger.

[Statutory Authority: RCW **43.20.050** and **70.119A.080**. WSR 17-01-062, § 246-290-220, filed 12/14/16, effective 1/14/17. Statutory Authority: RCW **43.20.050** (2) and (3) and **70.119A.080**. WSR 03-08-037, § 246-290-220, filed 3/27/03, effective 4/27/03. Statutory Authority: RCW **43.02.050** [43.20.050]. WSR 99-07-021, § 246-290-220, filed 3/9/99, effective 4/9/99. Statutory Authority: RCW **43.20.050**. WSR 91-02-051 (Order 124B), recodified as § 246-290-220, filed 12/27/90, effective 1/31/91. Statutory Authority: RCW **34.04.045**. WSR 88-05-057 (Order 307), § 248-54-131, filed 2/17/88.]

Washington State Board and Department of Health
PO Box 47990
Olympia, WA 98504-7990
wsboh@doh.wa.gov

February 12, 2024

Washington Action for Safe Water
Bill Osmunson DDS MPH

Dear Washington State Board of Health (Board) and Department of Health (Department),

RE: PETITION FOR RULE MAKING: WATER FLUORIDATION,

and FORUM ON FLUORIDATION

"Silence in the face of evil is itself evil: God will not hold us guiltless.
Not to speak is to speak.
Not to act is to act."

— Dietrich Bonhoeffer

Fluoridated water is NOT SAFE

The harm is IATROGENIC

Summary

Fluoride is a legend drug when intent of use is to prevent disease. Neither the Board nor Department have experts, procedures, funding, or authorization to determine the highly complex issue of the efficacy, dosage, label or hazard risk of drugs, such as the ingestion of fluoride, the responsibility of the FDA CDER[1]. The Board and Department are charged by the Legislature to write rules to assure safe drinking water, positively and confidently dispelling any doubt that fluoridation is safe. The Board contacted the FDA CDER charged by Congress to determine efficacy of drugs and was informed, requiring FDA CDER approval *"would effectively ban fluoridation.*" The FDA CDER has not, and would not, approve fluoridation due to a lack of one or all of the following: efficacy, dosage, safety, label, GMP[2], pharmaceutical ingredients, doctor's prescription, or patient consent. The Board is in violation of RCW 43.20.050 and other laws, to assure safe drinking water. This petition is focused on a minimum label to protect the development of the most vulnerable, i.e. fetus, infant, and child. However, this petition will not assure the safety of fluoridated public water, but will start to educate the public for their safety.

[1] Food and Drug Administration Center for Drug Evaluation and Research.
[2] Good Manufacturing Practices

The Board's duty is to adopt rules **to assure safety**. The brief summary of evidence presented in this petition will demonstrate the Board cannot assure safety of fluoridation, because fluoridation is:

- **Contributing to over exposure, overdose.**
- **Not Safe due to lack of safety research.**
- **A highly toxic poison, and not being regulated under drug laws.**
- **A legend drug, an illegal drug, because fluoridation lacks:**
    - **FDA CDER NDA approval**
    - **A doctor's prescription**
    - **Individual Patient Consent**
    - **Good Drug Manufacturing Practices**
    - **FDA Manufacturing Oversight and Licensing**
    - **Pharmaceutical grade purity of ingredients**
    - **Dosage control**
    - **Legend of patient instructions and warnings.**
- **A developmental neurotoxin as measured by:**
    - **Lower IQ**
    - **And pilot evidence of ADHD, Miscarriage, Premature Birth, Infant Mortality**
    - **Causes Tooth Damage**
    - **Contributes to Rheumatoid and Osteoarthritic-like Pain**
    - **Contributes to Cancer**
    - **Contributes to Bone Fractures**

- **Contributes to Thyroid Reduction, Diabetes, Obesity**
- **Contributes to Kidney damage**
- **Contributes to Reproductive problems**
- **Contributes to Allergies (overactive immune system)**
- **Contributes to Gastrointestinal disorders**

Alternatives to fluoridation are available for those who want to ingest fluoride, such as:

- **A doctor's prescription for fluoride supplement**
- **Bottled water with fluoride**
- **Avoid careful rinsing of toothpaste**
- **Avoid organic foods**
- **Drink more tea**
- **Drink more wine**
- **Eat more mechanically deboned meat**

The siloed purpose of fluoridation is to give people more fluoride because the Board does not trust people to make the decision for themselves, to take away freedom of choice.

The laws do not charge or permit the Board to approve drugs, nor determine safety to a confidence level of absolute certainty of harm.

The evidence presented does not permit the Board to assure, or be able to **"tell each person in Washington**

state, fluoridation is safe, positively and confidently, dispelling any doubts they may have."

The evidence presented here need only rise to the level of "doubt" in the Board's mind, not absolute confidence of harm. If the Board doubts fluoridation safety, the law requires the Board to at least stop endorsing fluoridation.

The Board should also consider we are not evaluating an and EPA industrial chemical or water purification chemical. This is an unapproved legend drug administered without consent, as a concentration rather than dosage, with known undisputed harm.

"RCW 43.20.050 Powers and duties of state board of health—Rule making—Delegation of authority—Enforcement of rules.

(1) The state board of health shall provide a forum for the development of public health. . ."

(2) In order to protect public health, the state board of health shall: ***(a) Adopt rules . . . to assure safe and reliable public drinking water*** *and to protect the public health."*

The question the Board should focus on in this petition is, **"Can the Board assure the public that fluoridation is safe?"** It is not the Board's charge to determine whether fluoride CAUSES an adverse effect. Confidence of a causality is a higher level of confidence than to **assure safety**.

Nor does **RCW 43.20.050** charge the Board with "weighing the evidence of benefit." The Board's sole charge is to assure safety. In 1975 the FDA CDER determined the evidence of efficacy was incomplete and has not changed their determination. In contrast, the Board claims on their web site fluoridation is effective and is safe, without reservation.

In 2010 we petitioned the Board 19 times to assure safe water and protect the public health. The lack of safety is not new, the evidence of harm is more robust.

However, we agree with the past Board and Department that they must rely on the FDA CDER to determine **efficacy, dosage, safety and label** of substances marketed with INTENT to prevent disease. The complex pharmacology, toxicology, epidemiology and benefit assessor is not in the lap of the Board, but the Board has attempted to assume the role of benefit (efficacy). Without accepting the FDA CDER's advice, the Board cannot assure fluoridation is safe.

The FDA CDER indicated to the Board in 2010, that should the Board accept our 2010 original petition for rule change, in effect requiring FDA CDER approval, would effectively ban fluoridation. The Board at that time, ignored the FDA and the Board did the exact opposite, more confidently promoting and endorsing fluoridation. Examples include the Board's web page and the Department's survey of public opinion on fluoridation.

The fluoridation lobby will push back against this petition. Throughout this petition, their concerns will be briefly addressed at each issue.

The Board needs to carefully review the evidence and assure themselves and the public that fluoridation is positively and confidently, dispelling any doubts they may have that fluoridation is anything but safe for everyone.

In this case the Legislature is reasonably consistent with:

"The Precautionary Principle says that if some course of action carries even a remote chance of irreparable damage to the ecology, then you shouldn't do it, no matter how great the possible advantages of the action may be. You are not allowed to balance costs against benefits when deciding what to do.

The fluoridation lobby will correctly state that the USA has not accepted the PP (Precautionary Principle) as Europe has done; however, the legislature in this case is consistent and raises the standard from PP's "remote chance" of damage, to the Legislature's increased confidence from damage to assure, positively and confidently, dispelling any doubts fluoridation is safe. And further, the PP uses "irreparable" damage rather than the Legislatures more cautious concern of "safe," which would include repairable damage or "aesthetic concern."

The Fluoridation lobby wants proof of harm, the Board is to be positive, confident, dispelling any doubts fluoridation is safe.

CONCLUSION: Some evidence is stronger than other evidence. However, when all streams of the scientific and legal evidence are assembled together and weighed, no reasonable person could assure their child or pregnant daughter that ingesting fluoridated water is safe.

To prove something is harmful, requires a different frame of reference, a different set of facts, different judgment than to assure something is safe. The Legislature has charged the Board to assure safety. For example, we do not demand to see a child's blood, broken bones or death before we determine a playground is unsafe.

More research is always desired, but no excuse for action. We have enough to know fluoridation is not safe.

OUR PETITION FOR RULE CHANGE

Consistent with health and safety issues in Title 246, Title 173, Title 296, WAC 173-340, and WAC 296-62-07521; this petition is made in compliance with RCW 34.05.330 and WAC Chapter 82-05.

This petition is for amendment to WAC 246-290-220

(8) For the safety of the developing fetus, infant, and child, the board no longer endorses the addition of fluoride to public water and recommends reducing fluoride exposure for pregnant mothers, infants and children under 6 years of age.

(a) Pregnant mothers and women planning to become pregnant (within 10 years) should limit fluoride ingestion by usually drinking water and liquids with less than 0.2 mg/L of fluoride, and do not swallow toothpaste;

(b) Care givers of infants should use water as low in fluoride as practical, less than 0.2 mg/L, for making infant formula, juice and drinking, and do not use fluoridated toothpaste.

(c) Carefully supervise children when they are using fluoridated dental products, such as toothpaste, to assure they are not swallowing the toothpaste and are able to spit, rinse and spit, and again rinse and spit without first swallowing. Read and follow the toothpaste label.

Our Point:[3] The intent of this rule change petition[4] is to start protecting the fetus, infants and children from the most significant risks and harm of fluoride exposure.

This petition will begin to protect the fetus, infant, and children from the worst known harm.

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[3] In 2010, we submitted our first rule change, which was denied. The Board mentioned:

"the EH Committee considers much of the discussion in our petitions to make points that go beyond the requested rule changes and are not pertinent to its decision." See Attachment #G We will try to explain the pertinence of each point.

[4] This petition concept is based in principle on the Safe Drinking Water Act which prohibits

-the addition of anything to water to prevent disease in humans, and

-warnings by the FDA CFSAN (Center for Food Safety and Nutrition).

BACKGROUND

We first asked the Board what was the "intent" of adding fluoride to tap water. Even though the Board had hundreds, actually thousands,[5] of documents on the intent of adding fluoride to public tap water, the Board responded that they had no records.

Our point: the intent of use determines jurisdiction.

In 2010, our first petition attempted to turn the very complex task of evaluation and judgment on the many streams of legal, ethical and scientific evidence, over to the authority charged by Congress in the FD&C Act to determine and regulate substances marketed with the intent to . . . "prevent disease."

We then filed our first petition which was denied. The Board misunderstood our petition and ignored the FDA's implied advice.

Relying on unauthorized agencies to do what they are prohibited from doing (EPA in the SDWA) does not "assure safety." The difficult complex task of determining the **efficacy** of

[5] Based on FOI documents responding with over 25,000 pages.

fluoridation,[6] and the **dosage** for that efficacy inclusive of background exposure, along with the vital determination of **safety** at that total exposure which would have efficacy, and a **label** with warnings and caution for intraspecies variations and alternatives is still the responsibility of the Board, in order to assure safety, dispelling doubt of harm.

This petition starts a label to assure the safety for our most vulnerable, but still falls short of assuring safety. As implied by the FDA, to assure safety, to remove doubt, would prohibit fluoridation.

The Board has become clear on intent of use of fluoridation is to prevent (mitigate) dental caries a disease in humans. We agree.

Petitioners are mostly not lawyers, toxicologists, epidemiologists, neurologists, endocrinologists, statisticians, physicians, pharmacists, hazard assessment experts, or risk assessors. We are voters, in effect, your "patients," and we are being harmed. I am a dentist with public health master's degree.

[6] The term fluoridation here will be used to refer to the addition of fluoride to public water with the intent to prevent dental caries (cavities) a disease.

The Board's first denial (Attachment #G) of our request for the Board or water purveyors to apply for FDA CDER (New Drug Application) would have taken the thorny, complex job of determining the safety, dosage, label, GDMP (Good Drug Manufacturing Practices), product purity, and the legal, ethical, and science off the Board's shoulders and placed the task in the lap of the authorized authority, the FDA CDER.

The Board at the time was correct in contacting the FDA, although the dental devices Division was not appropriate. Fluoride is not a dental device used by a dentist. Fluoride is a legend drug. However, the FDA did not advise the Board that FDA approval was not necessary. The FDA *"said if the Board accepted the language proposed in the petition, it effectively would ban public water fluoridation in Washington."*

Our point: The FDA would not approve fluoridation. Without FDA CDER approval, safety cannot assured.

JUDGMENT: REQUIRES EVALUATING ALL "STREAMS OF EVIDENCE"

To assure safety, the Board must consider and weigh multiple streams of evidence, concepts, studies, and disciplines, omitting none.

We always desire more studies. We always want numerous studies exactly the same so they can be precisely compared to increase confidence. We have enough evidence to be confident, fluoridation is not safe for many, most, or anyone.

LEGAL –A BRIEF SUMMATION:

All streams of legal evidence and jurisdiction, must be weighed, including, but not limited to the following questions:

1. What does Congress say about jurisdiction of substances marketed with intent to prevent disease? Congress clearly designates the jurisdiction to the FDA.
2. What has the FDA determined regarding fluoride ingestion with intent to prevent dental caries? Fluoride is a drug.
3. Has the Washington State Board of Pharmacy (Pharmacy Quality Assurance Commission, "PQAC" or "Pharmacy, or Board of Pharmacy) determined fluoride to be a legend drug? Yes. (Idaho Board of Pharmacy also determined fluoride to be a drug.) The PQAC is consistent with the FDA CDER, but neither the Board nor Department are consistent with the FDA or PQAC.
4. Does the Board or Department have the authority to determine the benefit, efficacy, of any substance with intent or claim to treat human disease i.e. drug approval? I have not found any Washington State law or provision where the Board or Department has authority to approve

drugs, regardless of dilution in tap water. Nor have I found the definitions, policies, experts, procedures, rules, or guidance recommendations the Board and Department must set up for the complex drug approval process. Nor have I seen laws exempting FDA CDER NDA from drug approval.

5. Does the Board and or Department have authority over assuring the safety of water? Yes.
6. Who has jurisdiction over the addition of drugs to tap water according to the EPA's (Environmental Protection Administration) water law office? FDA.
7. What jurisdiction does the CDC (Centers for Disease Control) have over approval of fluoridation's efficacy, safety, dosage or label? None.
8. What is a safe dose of fluoride exposure for everyone? The same as lead.
9. What have other Countries determined regarding fluoridation? Most developed countries have rejected fluoridation.

10. What does Washington RCW provide for guidance?

    Fluoride is undisputed as a highly toxic poison, exempt when regulated under drug laws.

    See more details and references below.

EFFICACY OF FLUORIDATION: examples

11. What is the intent of fluoridation, the addition of fluoride to public water, well known to the public and claimed by the Board of Health? Intent is to mitigate dental caries.
12. How effective is swallowing, ingesting, fluoride? Between none and half a cavity per person.
13. Is fluoridation cost effective when including real world costs estimated benefits, costs to fluoridate, and costs of known harm? No. Fluoridation is not cost effective.
14. Is fluoride a nutrient? Fluoride is not an essential nutrient and no disease is caused by a lack of fluoride ingestion.
15. What happens to caries when fluoridation stops?

    Research is mixed, probably no change.

DOSAGE OF FLUORIDE: examples

16. How much fluoride (mg/kg/day) is required to prevent dental caries? FDA says the evidence is incomplete.
17. How much fluoride (mg/kg/day) are people ingesting from all sources? Dosage is highly variable. The fetus, infants and children are most at risk of excess exposure.
18. What are the sources of fluoride for each individual and an individual's past exposure to fluoride? Highly variable.
19. Is the assumption that everyone needs more fluoride (supplementation) reasonable? No.
20. How much fluoride is an individual exposed to from toothpaste? Children often swallow half their toothpaste.
21. How much fluoride is the individual exposed to from the osteoclastic activity, turnover of bone? Bone contains between 1,000 and 8,000 ppm fluoride. Almost 100% of bone is remodeled in the first year of life and about 10% a year in adults.
22. How much fluoride is the individual exposed to from medical products? General anesthesia and medications can have fluoride and dosage is variable.

23. How much fluoride is the individual exposed to from foods such as mechanically deboned meat, pesticides such as cryolite and post-harvest fumigants, air such as freon and soil? Estimates vary.

SAFETY OF FLUORIDE INGESTION: examples

24. What is the purity, assay results, of the fluoride product used for fluoridation? Product is not pharmaceutical grade.
25. Is fluoride safe, lacking aesthetic or functional harm, for the teeth? Dental fluorosis, a biomarker of excess fluoride exposure, is arguable the most common disease of childhood.
26. Is fluoride safe, lacking neurotoxicity, for the developing brain? No.
27. Is fluoride safe for the fetus due to the transfer of fluoride from the mother? No.
28. Is fluoride safe for the endocrine system and thyroid? No
29. Is fluoride safe for the bones, the largest storage of fluoride in the body? Fluoride is 400% higher in those with bone cancer than normal patients for the same age.

30. What are synergistic effects, such as lead, mercury, or from other toxins? Still to be determined.

LABEL: Every approved substance with intent to prevent disease has a label for:

31. intent of use,
32. approved dosage,
33. approved label with warnings/cautions.

    Fluoridation has no label

TARGET POPULATION:

34. What percent of the population is to be protected from harm, 90%, 95% or 100%? How many thousands of people is the Board willing to put at risk? About 3.3 million in Washington State on fluoridated water. If the Board accepts 10%, that is 330,000 people ignored by the Board.
35. What margin of error, intraspecies variability, uncertainty factor is prudent? EPA uses 1:1 (no) margin of error or intraspecies variability and only accepts severe skeletal fluorosis or severe dental fluorosis as a risk.

Judging the “weight” or “power” from each of the more than 3 dozen streams of evidence is not intuitive to either researchers or lawyers. Most attempt to narrow the streams of judgment to one or two variables rather than be inclusive of all evidence.

The fluoridation lobby mistakenly claims safety by dividing the streams of risk, and each stream into drops of misty fog to obscure harm.

A “global” view, or totality of the evidence, a summation of weight is required to fully appreciate the extent of the harm and lack of safety.

“Proof” of efficacy requires randomized controlled trials (RCTs), prospective, double blinded, etc. An RCT would give subjects either the test substance or a placebo to consenting subjects and measure possible benefit and should look for harm and have blinded researchers. Intent to do good is valid research. FDA has determined evidence of efficacy is complete. Indeed, no RCTs exist on fluoridation.

“Proof” of safety is far more complex. In contrast, we cannot do randomized controlled trials giving people a poison and finding out when they are harmed or die. Harm must be determined

based on lower quality studies, such as correlation or ecological studies. Without RCTs, safety has been over-looked. No money is made on looking for harm and not selling the product. Safety is an orphan concept in a for profit culture.

Our point: Judgment requires adding the weight from each stream of evidence. A monumental task which the 2010 Board trusted to unauthorized agencies.

UNCERTAINTY FACTOR: Judgment requires the Board to select an uncertainty factor or margin of error and/or intraspecies variability? Not all humans respond the same due to genetics, health conditions, life stage, etc. Not everyone is average, drinking the average amount of water, average age, average health, etc. We all do not wear the same size shoe. The concept of "average" is important to grasp a concept, but an uncertainty factor, intraspecies variability, must be added to protect subpopulations.

This petition will not protect all the public; however, it could reduce, but not eliminate, harm to the most vulnerable.

LAWS

Washington Legislature, **RCW 43.20.05** designates authority for health and safety rules onto the Board of Health.

"RCW 43.20.050 Powers and duties of state board of health—Rule making—Delegation of authority—Enforcement of rules.

(1) The state board of health shall provide a forum for the development of public health policy in Washington state. ."

Since our petitions, 14 years ago and to our knowledge, the Board has not held a forum on fluoride exposure and fluoridation where both sides present laws and science. The Legislature did not give exemptions for difficulty, busy schedule, controversial topics, or cherry-picking participants, etc.

"RCW 43.20.050 continues:

(2) In order to protect public health, the state board of health shall:

*(a) Adopt rules for group A public water systems, as defined in RCW **70A.125.010**, necessary to assure safe and reliable public drinking water and to protect the public health."*

The Board has failed to assure safe public drinking water.

The Department's survey of public opinion on fluoridation demonstrates many, if not most of the public, do not trust the Boards opinion that fluoridation is safe. The Department should have spent the time and limited resources and surveyed "science" rather than public opinions.

Our first petition 14 years ago requested the Board advise or recommend water purveyors to apply to the FDA CDER for an NDA (New Drug Application) because fluoride was determined by the Board of Pharmacy to be a legend drug. The Board denied our petition, in part, on the grounds the rule change would *"essentially, prohibit all tap water fluoridation in Washington."*

The Board appears to have in part misunderstood the FDA. True, the FDA does not regulate contaminants in public water. However, the FDA regulates the fluoride when a health claim is made for the product, regardless of diluting the drug in tap water.

In other words, a "snake oil salesman" cannot simply take their elixir and dilute it in tap water and evade FDA oversight. EPA regulates water, FDA regulates drugs.

An important early step to assure the safety of public water when fluoride is added, was communicating with the FDA and EPA. Thank you, we agree this was a correct step.

The FDA confirmed and supported the Washington Board of Pharmacy and our petition that adopting our 2010 petition would effectively ban public water fluoridation in Washington.

It appears the Board of Pharmacy and certainly we assumed the Board would come to the logical conclusion, “if it can’t be approved, it isn’t safe.” However, the Board appears to have doubled down and promoted fluoridation for everyone, without assuring safety.

What about fluoridation would prove difficult to gain FDA approval? Lack of efficacy? Lack of controlled dosage? Lack of safety at that dosage? Lack of label? Lack of the patient’s doctor’s oversight? Lack of patient consent? Lack of chemical purity? Or, all of those? The Board cannot assure safety.

The Board does not determine **efficacy**. The U.S. Food and Drug Administration Center for Drug Evaluate and Research (FDA CDER) has jurisdiction over substances marketed with intent to prevent disease in humans.

In 1975 the FDA said the evidence of efficacy was incomplete.

The second step is to determine how much does it take to be effective, dosage.

The third step is to determine the risks and harm, i.e. safety, at that dosage. Without knowing any one of those steps, safety cannot be assured.

WAC 246-290-220 permits the Department to continue the use of non-certified chemicals (which would encompass fluoride chemicals), provided:

"(b)There exists no substantial evidence that the use of the chemical or material has caused consumers to register complaints about aesthetic issues, or health related concerns, that could be associated with leachable residues from the material;"

We are once again registering a complaint of dental fluorosis aesthetic and functional harm and other health concerns is made to the Board and Department of Health.

The Legislature appears to have concern for aesthetic issues which is part of dental fluorosis and health related concerns which is also part of dental fluorosis.

This petition for rule change is focused on the Board, but addressed to both the board and Department because both share responsibility to assure safety to the public, especially the fetus, infants and children. The Board and Department have for more than 14 years been fully aware, fluoridation is not safe.

FORUM REQUEST

A 2 or 3 minute public comment at Board meetings is not a "forum" where *"ideas, questions, and views on a particular issue can be exchanged."*[7]

For the health of the public, we have requested a forum as provided in RCW 43.20.050 where ideas, questions, views, science and laws can be exchanged on fluoridation.

"RCW 43.20.050 does not authorize the Board to dilute drugs in the water with the intent to treat humans rather than treat

[7] Oxford Languages Dictionary

water, nor does it permit the Board to reduce the safety of the water or determine efficacy of treating human disease.

RCW 43.20.050 does not appear ambiguous or uncertain. The Board is the authority in Washington State and SHALL assure the water is safe.

Assuring the water safe from unknowns is one problem; however, actually intentionally causing the unsafe water is iatrogenic harm.

When a doctor makes a mistake, the patient can be harmed. When the Board makes a mistake, millions can be harmed.

POISON DEFINED: fluoride is a highly toxic substance, a hazard, and must not be taken lightly or casually dismissed.

There is no physiologic process which requires fluoride, no "minimum daily requirement."

Fluoride is not a nutrient. No disease is caused by the absence of fluoride ingestion.

Fluoride is one of the most powerful elements known.

***"RCW 69.38.010 "Poison" defined**. As used in this chapter "poison" means:*

(1) Arsenic and its preparations;

(2) Cyanide and its preparations, including hydrocyanic acid;

(3) Strychnine; and

(4) Any other substance designated by the state board of pharmacy which, when introduced into the human body in quantities of sixty grains or less, causes violent sickness or death."

60 grains =3,888 mg.

The probable violent sickness or death of fluoride is estimated at 5 mg/Kg body weight. Although it might take 50 mg to cause violent sickness or death in an adult, an estimated 20 mg NaF could cause violent sickness or death in an infant. The probable fetus lethal dosage is unknown.

I summarized to **the Board of Pharmacy** that their job in the most simple terms was to determine whether 20 mg was less than 3,888 mg, (obviously) and if so, fluoride is defined by RCW 69.38.010 as a **"POISON."** and exempt when regulated under pesticide or drug laws, RCW 69.38.010.

Without dispute, fluoride is an extremely toxic substance, poison, more lethal than lead or gasoline.[8]

RCW 69.40.030 ". . . *and every person who willfully poisons any spring, well, or reservoir of water, is guilty of a class B felony and shall be punished by imprisonment in a state correctional facility for not less than five years or by a fine of not less than one thousand dollars."*

Do not mess around with poisons.

When evaluating fluoride, the Board must put on their "poison" hat and think serious caution with a highly dangerous toxic poison. Fluoride is not a play toy, nutrient, or food. Also keep in mind the only potential benefit of fluoride ingestion is an alleged reduction in dental caries which is a theory which lacks quality research and is disputed, unapproved by the FDA CDER in tablets or diluted in liquid.

The Board is attempting to mitigate dental caries, a very common disease, which can be seriously painful, life altering; however, not considered highly lethal or contagious.

[8] Estimate of lethal dose is by Wolford. For comparison, 1.5 to 2 mg/k for a 70 kg is considered lethal and 0.6mg/kg for arsenic. 450 mg/kg of lead is also considered lethal. 2,000 to 5,000 mg/kg of gasoline can be fatal.

RCW 69.38.010 Exempts poisons from poison laws when regulated under drug or pesticide laws.

The Board of Pharmacy exempted fluoride from poison laws and determined fluoride to be a legend drug.

Our point: Fluoride is highly toxic and unless regulated as a drug, under drug laws, fluoride remains under poison laws and for the safety of the public must be regulated as a poison, or drug.

DRUGS DEFINED

See also attachment #A

Drugs are defined as: *"articles intended for use in the diagnosis, cure, mitigation, treatment, or prevention of disease"* [FD&C Act, sec. 201(g)(1)].

The Board of Health responded in 2010, to my question of the intent of fluoride ingestion, responding:

This agency, therefore, is not in possession of any records related to the Board s purpose and intent for supporting the addition of fluoride to public drinking water."[9]

Seriously, the Board . . . had NOTHING to back up why they recommended adding fluoride to public water. However, FOI

[9] July 22, 2010 letter to Bill Osmunson regarding public information disclosure request.

evidence with thousands of pages clearly disagreed with the Board's claim of "no records" were available at the time on the intent of fluoridation. The public knew, and knows, why fluoride is added to public water. But not the Board?

The Board's claim of "no records" was simply an attempt to mislead, an egregious attempt to protect policy rather than the safety of the public, especially, the fetus, infants and children.

Once again, the Board denial in our 2010 petition, wrote:

"if the Board accepted the language proposed in the petition, (for FDA CDER approval) it effectively would ban public water fluoridation in Washington."

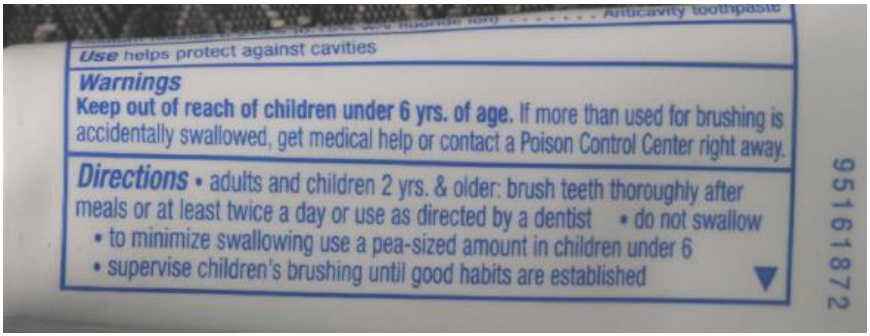
Our point exactly. The Board did not assure the safety of the public. If fluoridation cannot be approved, continuing the practice does not assure safety.

For the safety of the public, the Board must understand, either fluoride is a poison because it is highly dangerous, or it is exempt when approved under drug laws but not exempt when not approved by the FDA. Fluoride ingestion with intent to prevent dental caries is not approved for ingestion in pills, liquids, or diluted in public water.

FDA WARNING: DO NOT SWALLOW

The FDA has approved fluoridated toothpaste as an over-the-counter drug, not requiring a doctor's prescription.

The first step I took in evaluating fluoride was to read the toothpaste label. "Drug Facts." Fluoride is without dispute a drug.



The intent is clear, "helps protect against cavities."

The FDA continues:

"Keep out of reach of children under 6 years of age." "if accidentally swallowed, get medical help or contact a Poison Control Center right away."

*"Directions * adults and children 2 years. & older: brush teeth thoroughly after meals or at least twice a day or use as directed by a dentist * do not swallow * to minimize swallowing use a pea-sized amount in children under 6 * supervise children's brushing until good habits are established"*

In my pilot study, I then took a toothpaste tube and squeezed out what I thought were "pea size amounts" for the entire tube and calculated the number and the "dosage" of fluoride in each pea size amount, and took the pictures above. Then I looked up the data and realized my pea size amount as shown in the picture above on the tooth brush was twice the size as recommended by the FDA CDER, which should contain only 0.25 mg of fluoride. The picture above with the amount of toothpaste was a "large pea size" amount containing 0.5 mg of fluoride.

Consider for a moment, the Board recommends everyone be required without consent and regardless of safety, to swallow in each glass of water the same amount of fluoride (0.25 mg) as the FDA "Warnings" tell us "Do Not Swallow" and to "contact a Poison Control Center right away."

Seriously, who do you trust more? The FDA or Board?

Assuring safety is not possible if there were no other evidence.

The Department did a survey of voters to determine their opinion on fluoridation, in effect, the public was asked do they trust the messaging of the Board and Departments of Health regarding fluoridation. Many, and in some places most, do not trust the Board or Department on fluoridation. Now, who would have thought the public knows more than the Board of Health?

The FDA does not mince words, is precise, “Do Not Swallow.” The Board should have the same warning for fluoridated tap water.

The Board must think the implications through. If the Board cannot be trusted on fluoridation, can they be trusted for vaccinations, prevention of disease, sanitation, or any other public health recommendation?

Remember, the toothpaste label was approved by the FDA over a quarter of a Century ago, not fluoridation.

FLUORIDE IS A LEGEND DRUG: the Board has been fully aware for more than a decade that fluoride is a legend drug and the Board and Department have failed to assure the public water is safe.

In contrast, to the Washington State Board of Health the Washington State Board of Pharmacy (PQAC) determined*:*

"Fluoride is a legend drug regulated under chapter 69.41 RCW. RCW 69.41.010 defines a 'legend drug' as drugs 'which are required by state law or regulation of the state board of pharmacy to be dispensed on prescription only or are restricted to use by practitioners only."[10]

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[10] State of Washington Department of Health Board of Pharmacy June 4, 2009 letter to Bill Osmunson DDS; RCW 69.41.010(12) (#13 in 2024) defines legend drugs; WAC 246-883-020(2) states legend drugs are listed in 2002 *Drug Topics Red Book* (relevant *Red Book* pages including page 342 that lists "Fluoride" are attached to the above-referenced Board letter.

The current online red book 2023 edition for fluoritab lists
Adverse Reactions
Severe
exfoliative dermatitis / Delayed / Incidence not known
GI bleeding / Delayed / Incidence not known
hematemesis / Delayed / Incidence not known
Moderate
stomatitis / Delayed / Incidence not known
atopic dermatitis / Delayed / Incidence not known
anemia / Delayed / Incidence not known
dental fluorosis / Delayed / Incidence not known
synovitis / Delayed / Incidence not known
Mild

Note: The Board of Pharmacy referenced the “Red Book,” not the list of approved drugs in the FDA “Orange book.”

The WSBP (PQAC) references the 2002 Drug Topics Red Book which is industry, not published by the FDA CDER but rather the Physician’s Desk Reference. As a doctor, I use the PDR, a good book for doctors. Approval of substances intended to prevent disease in humans is the FDA responsibility, that’s the FDA Orange Book.

RCW 69.41.010 (13) *"Legend drugs" means any drugs which are required by state law or regulation of the pharmacy quality assurance commission to be dispensed on prescription only or are restricted to use by practitioners only.*

When reading the laws, “think fluoridation.” Think, “how does this law apply to fluoridation? Who is the practitioner dispensing the fluoridated legend drug?

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urticaria / Rapid / Incidence not known
weight loss / Delayed / Incidence not known
asthenia / Delayed / Incidence not known
abdominal pain / Early / Incidence not known
vomiting / Early / Incidence not known
hypersalivation / Early / Incidence not known
nausea / Early / Incidence not known

For Drug Interactions: The list is long and should be read. Some interactions include: Magnesium, Aspirin, Calcium, Vit D, etc.

WAC 246-945-*010* *Prescription and chart order—Minimum requirements.*
(3) A prescription for a noncontrolled legend drug must include, but is not limited to, the following:
(a) Prescriber's name;
(b) Name of patient, authorized entity, or animal name and species;
(c) Date of issuance;
(d) Drug name, strength, and quantity;
(e) Directions for use;
(f) Number of refills (if any);
(g) Instruction on whether or not a therapeutically equivalent

Who is keeping track of the chart order for each fluoridated patient? No one.

WAC 246-945-*005* *Commission inspections and investigations.*
§ 69.41.020. Prohibited acts -- Information not privileged communication

Legend drugs shall not be sold, delivered, dispensed or administered except in accordance with this chapter.

(1) No person shall obtain or attempt to obtain a legend drug, or procure or attempt to procure the administration of a legend drug:

(a) By fraud, deceit, misrepresentation, or subterfuge; or

(b) By the forgery or alteration of a prescription or of any written order; or

(c) By the concealment of a material fact; or

(d) By the use of a false name or the giving of a false address.

Let's think this through. Has the Board misrepresented the legend drug? Yes. Fluoridation, fluoride ingestion, requires a doctor's prescription for each patient.

Has the Board concealed a material fact? Indeed, the Board or fluoridation purveyors are indeed concealing material facts on hazard, jurisdiction, safety, label, FDA CDER approval, etc. Promoting an unapproved illegal drug as without risk, without need for prescription, by false name of the EPA rather than FDA CDER are violations of **WAC 246-945-*005***.

WAC 246-945-*005 continues:*

(2) Information communicated to a practitioner in an effort unlawfully to procure a legend drug, or unlawfully to procure the administration of any such drug, shall not be deemed a privileged communication.

(3) No person shall willfully make a false statement in any prescription, order, report, or record, required by this chapter.

(4) No person shall, for the purpose of obtaining a legend drug, falsely assume the title of, or represent himself or herself to be, a manufacturer, wholesaler, or any practitioner.

(5) No person shall make or utter any false or forged prescription or other written order for legend drugs.

(6) No person shall affix any false or forged label to a package or

receptacle containing legend drugs.

Not much in **WAC 246-945-*005*** that does not directly apply to fluoridation.

The Board must also consider. ***"WAC 246-945-030 Identification of legend drugs for purposes of chapter 69.41 RCW. (1) Those drugs determined by the FDA to require a prescription under federal law should be classified as legend drugs under state law because their toxicity, potential for harmful effect, methods of use, or collateral measures necessary to their use indicate they are only safe for use under the supervision of a practitioner.***

(2) The commission finds that under state law, legend drugs are those drugs designated as legend drugs under federal law, as of the date of adoption of this rule, and listed in at least one of the following publications:

(a) The 39th Edition, including supplements, of the Approved Drug Products with Therapeutic Equivalence Evaluations "Orange Book" (available at https://www.fda.gov/drugs/drug-approvals-and-databases/ approved-drug-products-therapeutic-equivalence-evaluations-orange- book).

(Emphasis supplied)

I asked the whether fluoride was a drug. The FDA responded:

"A search of the Drugs@FDA database . . . of approved drug products and the Electronic Orange Book. . . does not indicate that sodium fluoride, silicofluoride, or hydrofluorosilicic acid has been approved under a New Drug Application (NDA) or Abbreviated New Drug Application (ANDA) for ingestion for the prevention or mitigation of dental decay. . . . At the present time, the FDA is deferring any regulatory action on sodium fluoride products. . ."[1] Email from the FDA (7-22-09).

Our Point: “Deferring regulatory action” does not provide assurance fluoridation is safe. Fluoride is highly toxic, a poison, exempt from poison laws when dispensed as a legend drug. Fluoride is not an approved legend drug.

Are there ways to evade protecting the public? Silence is one. Relying on an unauthorized government agency is another.

Changing the law may protect policy but does not change science, empirical evidence or protect the public health, and does not assure safety.

For 14 years the Board of Health has not answered the obvious question, “who is the practitioner under who’s license the dispensing of the fluoridation drug is dispensed to everyone without their consent?”

Or, can anyone make a drug and sell it without FDA, RCW, or WAC regulatory oversight as a “snake oil salesman” simply by diluting it in tap water? No.

A person cannot, for example, mix vodka and cherry juice with some tap water and claim it to be a miracle drug to cure all

diseases and evade all drug regulatory authority. That is precisely why Congress passed the FD&C act, to stop hucksters selling fake products like fluoridation. (Remember, I promoted fluoridation for 25 years out of dental school. That "huckster" comment hits me squarely in the face.)

The Board mentions in the letter (Attachment #G) some points in our 2010 petition go beyond the rule change request. No, the Board misunderstood. Every point in that and this petition directly relates to the petition and to assure safe tap water.

Our point: **"Fluoride is toxic and to be safe must be regulated as a prescription (legend) drug and if fluoridation cannot be assured safe, fluoridation should not be endorsed by the Board."**

An FDA and Board of Pharmacy newsletter, stated:

Manufacturers of unapproved drugs are usually fully aware that their drugs are marketed illegally, yet they continue to circumvent the law and put consumers health at risk." Washington State Board of Pharmacy 7/2008 Newsletter

Those promoting, advising, the mass administration of a highly toxic substance, unapproved, illegal prescription drug are certainly complicit in the harm caused to the public.

RCW 43.20.050 does not authorize the Board to simply trust endorsements, the dental lobby, or any other agency, least of all an unauthorized agency. Ignoring an authorized agency does not assure safety. The Board's job is to assure safety.

RCW 57.08.012 Fluoridation of water is authorized.

"A water district by a majority vote of its board of commissioners may fluoridate the water supply system of the water district. The commissioners may cause the proposition of fluoridation of the water supply to be submitted to the electors of the water district at any general election or special election to be called for the purpose of voting on the proposition. The proposition must be approved by a majority of the electors voting on the proposition to become effective."

RCW 57.08.012 permits fluoridation but does not exempt the Board from ensuring the water is safe and correctly approved by the authorized regulatory agency.

Pause for a moment and critically evaluate **RCW 57.08.012**. Did the legislature expect each voter to spend the hundreds/thousands of hours to carefully review the many

streams of legal and scientific evidence in detail and make judgment on the legality, jurisdiction, efficacy, safety, current dosage, desired dosage, ethics with all streams of evidence of ingesting more fluoride for their neighbors? That expectation is not real world.

For example, just because RCW permits an individual to get a driver's license, does not mean they can ignore the laws of the road or the highway and can ignore safety standards.

In the denial of our 2010 first petition, the Board, in effect agreed their authority includes determining the "safety" of fluoridation by mistakenly relying on the CDC and EPA to assure the issue of safety. We agree the Board has jurisdiction over the laws and science relating to **RCW 57.08.01**. Science is dynamic. In the last 4 decades since **RCW 57.08.012** was passed, we have more evidence to consider.

Voting on an issue often relies on those with the largest marketing budget. And public relations authority will gain many voter's approval, rather than factual evidence. The dental lobby has convinced the Board to do the marketing for them.

The Board must take endorsements for fluoridation off the internet.

Our point: The Board must not rely on each voter, the EPA nor CDC nor NTP to determine the complex science on fluoridation efficacy, dosage, safety and label.

The Board appears in violation of WAC 246-290-220

"(5) The department may accept continued use of, and proposals involving, certain noncertified chemicals or materials on a case-by-case basis, if all of the following criteria are met:

(b)There exists no substantial evidence that the use of the chemical or material has caused consumers to register complaints about aesthetic issues, or health related concerns, that could be associated with leachable residues from the material;"

The law only rises the level of " no substantial evidence." I spent over 4 decades treating aesthetic and functional dental fluorosis, a known adverse effect of excess fluoride ingestion. NHANES reports a substantial 2 out of 3 children with dental fluorosis. That is a biomarker of excess fluoride exposure.

The evidence provided here is substantial evidence of both aesthetic issues and health related concerns, risks and harm.

CONTAMINATED ADULTERATED MISBRANDED PRODUCT

There are no shortages of laws regarding unapproved illegal drugs and manufacturing, requiring pharmaceutical quality ingredients. Although fluoride is not a narcotic, good manufacturing practices apply and purity of the product applies as set forth by the U.S. Pharmacopeia.

Examples: Chapter 69.50, RCW RCW 69.40.030, Chapter 18.64 RCW, RCW 18.64.005 (7) RCW 69.50.401, RCW 43.71C.060,

The chemicals added to public water for fluoridation are contaminated waste products of manufacturing, often foreign manufactured, misbranded, often without NSF[11] assay, not pharmaceutical grade, adulterated, contaminated, not manufactured under Good Drug Manufacturing Practices (GDMP), and neither approved before marketing or inspected by the FDA CDER during manufacturing and distribution.

The substance added to public water is NOT pharmaceutical grade which is assumed in the PDR and Pharmacopeia that the

[11] National Sanitation Foundation, a private company which seldom releases purity evidence to the public.

Board of Pharmacy relied on, but rather industrial grade products such as hydrofluorosilicic acid or industrial grade sodium fluoride, both are contaminated products, often containing:

Arsenic – 90 percent of the arsenic contributed by drinking water treatment chemicals is attributable to hydrofluorosilicic acid. Source: Wang C, Smith DB, Huntly GM. Treatment Chemicals contribute to Arsenic Levels. Opflow (AWWA), October 2000. EPA's MCLG is "0" "Ingestion of inorganic arsenic in drinking water has been linked to skin, lung, bladder, kidney, prostate, and liver cancers." Oregon Dept. Human Services. Drinking Water and Environmental Exposure, 2007

Lead – EPA's MCLG is "0" Ionescu Neuro Endocrinol Lett 2006, $15B to remove - awwa

Beryllium – Increase in cancer. Taylor-McCabe, Poteomics 2006

Vanadium – Mixed results

Cadmium – Increase in breast cancer McElroy J Natl Cancer Inst. June 2006

Mercury – Cancer Increase and Neurological Disorders Ionescu Neuro Endocrinol Lett 2006

Radium – Cancer Increase Lloyd Radiat Res. 2005

Radionuclides – Cancer Increase Sevan'kaev Raiats Biol Radioecol 2006

Silicon – Probably safe

Bauxite – Mixed opinions

It is important to note that not all batches have all of these contaminants, and contaminant concentrations are usually unknown. The fluoride chemical purity is assumed by the National Sanitation Foundation (NSF), a private company who refuses to

provide assay data to the public, and at times have said they do not test each batch.

When I asked NSF how the NSF permits fluoride to be added to the water at 1 ppm, when their standards do not permit more than 10% of the EPA's MCL's 4 ppm? 10% of 4 is 0.4 ppm. The NSF told me that fluoride is the product and not a contaminant in the product. The NSF response makes no sense. I commented, if the fluoride were called any other name, would NSF permit fluoride to be intentionally added to water? The choice of a name does not change the toxicity of a product. The NSF representative on the phone went silent.

And, further, China prohibits fluoride being added to their public water. Research from China on developmental neurotoxicity was some of the earliest and motivated researchers in the USA to question claims of fluoride's developmental neurotoxicity safety and start serious research.

China has excess fluoride and their toxic fluoride waste by-product of manufacturing is shipped to the USA, which the Board of Health recommends for all of us to drink, regardless of purity or dosage, an individual's health status or choice. The Board blindly

trusts China's quality of industrial product to be safe, which China does not permit in their water.

Tell the public China's industrial waste product is being disposed of in our tap water and see if the public thinks that assures them the water is safe.

THE SAFE DRINKING WATER ACT DOES NOT PERMIT FLUORIDATION.

The Board appears in violation of the **Safe Drinking Water Act** as detailed below and Attachment #F.

Our point: The SDW Act prohibits the addition of anything to tap water to treat humans. No assurance of safety from the SDWA.

THE FOOD DRUG AND COSMETIC ACT CHARGES THE FDA TO APPROVE DRUGS.

The Board also appears in violation of the **FD&C Act** as detailed below and in Attachment #A. (Eight points below)

1. RCW 18.64.011 (14) and **[FD&C Act, sec. 201(g)(1)].**

"Drugs" means:

(a) Articles recognized in the official United States pharmacopoeia or the official homeopathic pharmacopoeia of the United States;
(b) Substances intended for use in the diagnosis, cure, mitigation, treatment, or prevention of disease in human beings or other animals;
(c) Substances (other than food) intended to affect the structure or any function of the body of human beings or other animals; or. . . "

Fluoride is in the US Pharmacopoeia.

The intent of fluoride ingestion is not in dispute and well known to the public, to allegedly prevent dental cavities.

Neither the PHS (U.S. Public Health Service) CDC (U.S. Centers for Disease Control), nor EPA (U.S Environmental Protection Agency), have authority from Congress to approve any substance with intent to prevent, mitigate or cure disease in humans.

Our point: Congress, in the FD&C Act United States Code, Title 21, has charged the FDA with approval of substances marketed with intent to prevent disease.

The purpose of drug approval is to protect the public from harmful substances such as fluoride.

As presented above, **RCW 57.08.012** authorizes a water district board of commissioners or public to vote on fluoridation, but does not address the toxicity, efficacy or safety of fluoridation. Nor does **RCW 57.08.012** designate the agency which has jurisdictional oversight to determine the efficacy, dosage, safety and label. Nor does the **RCW 57.08.012** designate who the prescribing practitioner, who the legal intermediary must be for fluoridation.

RCW 57.08.012 does not remove the requirement for the Board to assure the public that fluoridation is safe.

Nor does **RCW 57.08.012** authorize the Board or Department to be the marketing, promotional or the advertising arm for fluoridation lobby.

Our point here is although RCW 57.08.012 permits fluoridation, determining oversight jurisdiction, science on efficacy, dosage, safety, and label was never removed from the Board's responsibility to assure safe water.

2. The FDA in 2000 responded to the Honorable Ken Calvert, House of Representatives, (See letter at Supplement #D attached) to his question #1:

"If health claims are made for fluoride-containing products. . . do such claims mandate that the fluoride-containing product be considered a drug, and thus subject the product to applicable regulatory controls?"

FDA's response:

"Fluoride, when used in the diagnosis, cure, mitigation, treatment, or prevention of disease in man or animals is a drug that is subject to Food and Drug Administration (FDA) regulation . . . ."

Question #2:

"Are there any New Drug Applications (NDA) on file, that have been approved, or that have been rejected, that involve a fluoride-containing product (including fluoride-containing vitamin products). . . . "

FDA's response:

"NO NDA's have been approved or rejected for fluoride drugs meant for ingestion.. . . "

Question #3:

"Does FDA consider dental fluorosis a sign of over exposure to fluoride?"

FDA Response:

“Dental fluorosis is indicative of greater than optimal ingestion of fluoride. In 1988, the U.S. Surgeon General reported that dental fluorosis, while not a desirable condition, should be considered a cosmetic effect rather than an adverse health effect. Surgeon General M. Joycelyn Elders reaffirmed this position in 1994.”

Question #4:

“Does FDA have any action-level or other regulatory restriction or policy statement on fluoride exposure aimed at minimizing chronic toxicity in adults or children?

FDA Response:

“The monograph for OTC anticaries drug products sets acceptable concentrations for fluoride dentifrices, gels and rinses (all for topical use only). This monograph also describes the acceptable dosing regimens and labeling including warnings and directions for use. FDA's principal safety concern regarding fluoride in OTC drugs is the incidence of fluorosis in children. Children under two years of age do not have control of their swallowing reflex and do not have the skills to expectorate toothpaste properly. Young children are most susceptible to mild fluorosis as a result of improper use and swallowing of a fluoride toothpaste. These concerns are addressed in the monograph by mandating maximum concentrations, labeling that specifies directions for use and age restrictions, and package size limits.”

3. Also see attached FDA letter, Supplement #C and note that accepted fluoride containing dentifrices contain the warning “do not swallow.”

4. Children may swallow half the fluoride toothpaste they use which contains 1,000 to 1,500 ppm fluoride.

I watched my 11-year-old daughter brush her teeth one night. She objected saying she knew how to brush her teeth and didn't need her daddy dentist to watch. I said I wanted to watch to be sure she did not swallow before rinsing. She leaned over the sink and I saw her little "Eve's apple" bob up and down and she then spit. The reflex of swallowing first at 11 was still strong. Children swallow toothpaste, estimated in research as often half the toothpaste they use.

5. The fluoridation lobby will object to the suggestion for FDA CDER NDA, in part, on the grounds that fluoridated bottled water is approved at 0.7 mg/L by the FDA.

Not so fast. We can learn about safety from bottled water. There are two main sections to the FDA: CFSAN (Center for Food Safety and Applied Nutrition) (Food), and CDER (Center for Drug Evaluation and Research) (Drug).

[See Supplement #B attached (stamped Exhibit 4) which is a 2006 letter from CFSAN the "Food" side of the FDA, not the Drug side.]

Supplement manufacturers would like to make health claims and the FDA CDER had stopped them. The supplement lobby went to Congress and was able to get a law to state in part:

"a manufacturer may submit to the . . . FDA a notification of health claim based on an authoritative statement from an appropriate scientific body of the United States Government or the National Academy of Sciences or any of its subdivisions."

The law firm Covington and Burling NOTIFIED the FDA that a health claim would be made for fluoridated bottled water and a claim of reduced risk of dental caries. It is important that the Board of Health understand that bottled water with fluoride did not gain FDA CDER approval. Rather the "food section of the FDA" was "NOTIFIED."

No science was provided to the FDA on efficacy, dosage, total exposure, label or safety. And no empirical evidence, facts, were provided on risk factors, margin of error or safety. Zero science, just "notification." The only evidence was endorsements by the CDC (2001), Surgeon General (2000) who heads the Public Health Service and the Public Health Service (1991).

Neither the Surgeon General, nor the Public Health Service, nor the Centers for Disease Control, nor the FDA's Center for

Food Safety and Applied Nutrition (CFSAN) nor does the Washington State Board of Health have drug approval authority.

Our Point: Writing new laws does not change the empirical factual scientific evidence and does not assure the safety of the poison/legend drug.

The FDA Warning letter (See Attachment #B) has a concentration range of 0.6 to 1.0 mg/L which in 2022 FDA food section lowered to 0.7 mg/L. The letter states,

*"The language is: "Drinking fluoridated water **may** reduce the risk of [dental caries or tooth decay]."* [emphasis provided]

The FDA language says, "MAY." Until the FDA CDER provides more confidence, "may" is a reasonable word. Of course, fluoride ingestion may not reduce dental caries. The FDA had, in 1975, determined the evidence of efficacy of fluoride ingestion was incomplete.

In contrast, the Board of Health is certain and confident that fluoridation reduces an amazing 25% of tooth decay, the Board states: ***Water fluoridation reduces tooth decay by about 25 percent over a person's lifetime."***

The FDA warning letter (See Attachment #B) continues:

"In addition, the health claim is not intended for use on bottled water products specifically marketed for use by infants."

This Petition to the Board of Health is in keeping with the Food section of the FDA, to protect infants.

The second FDA WARNING LETTER in #B, in part states,

"your product label has serious violations. . . Your product is misbranded. . . bears an unauthorized health claim in its labeling." "Health claims may not be made for food products, including bottled water, for which the label represents or purports that the food is for infants or toddlers less than two years of age. . . ."[12]

The FDA continues: *"In addition, we have the following comments:*

The serving size of your Nursery Purified Water product is based on 8 fluid ounces. While the FDA has not established a reference amount customarily consumed (RACC) for water by infants and toddlers, we recommend that you use the infant and toddler RACC for juices, which is 4 fl oz."

[12] Supplement #B attached has a second **"WARNING LETTER"** stamped as Exhibit 5 dated 2009, from the FDA PHS to CEO's at DS Waters of America, LP regarding "NURSERY Purified Water with added fluoride."

Even though the FDA was “notified,” and had no authority to refuse, the FDA cautiously added in effect the beginning of a label and a reduction in dosage, 4 fl oz. Did industry comply. . . no.

Our Point: At a minimum the Board should start a label of caution and warning which our petition intends.

6. In June, 1975, Drug Therapy reported the FDA had rejected 35 new drug applications for fluoride/vitamin combinations because*: **“There is NO substantial evidence of drug effectiveness as prescribed, recommended, or suggested in labeling.”***

The FDA CDER is still correct. Almost 80 years of fluoridation, only one randomized controlled trial on fluoride ingestion has been published. And that was on pregnant mothers, reporting no statistical benefit for their infants. None published using fluoridated water.

I applied and received FDA approval for a dental device which is less stringent than for a drug. The FDA was fair, strict, strong, scientific and raised my confidence in their efforts to protect the public. They have my respect. The Board would begin to assure safety by following the FDA CDER’s advice and label.

Our point: The efficacy of fluoride ingestion is still incomplete.

7. At first, “lack of efficacy” stuck in my throat in disbelief. For me, the paradigm shift was extremely difficult. I was confident I could see benefit in my patients. But the science convinced me, I had been wrong. For example, my rich patients had better oral health. Socioeconomics is a confounder. I had given fluoridation credit for the rich having better health.

8. A Board member mentioned they are not supposed to have to review the science.

For drugs, well said, I agree. Drug approval is not part of the Board’s job, but ensuring the drug has been properly approved is part of the Board’s job, and the Board must assure safety.

THE BOARD’S DENIAL OF OUR 2010 PETITION

The Board of Health letter June 9, 20010, denying our petition to protect the public health, see attachment #G WA-board-of-health-memo-6-9-10, stated: (Letter quotes in brown)

“Motion: The Board denies the petition for rule making from Dr. William (sic) Osmunson dated May 11, 2010 because the US Food and Drug Administration has a memorandum of

understanding with the U.S. Environmental Protection Agency clarifying that the latter agency has authority for regulating tap water."

1. EPA & FDA MOU (Memorandum of understanding)

The Board was misdirected or misunderstood, believing the EPA had jurisdiction over drug approval ensuring fluoridation was: effective, correct dosage, with a protective label**.** See Attachment #F, February 14, 2013, EPA Letter

Steven M. Neugeborn, Associate General Counsel, Water Law Office, U.S. Environmental Protection Agency, regarding the status of an MOU between EPA and FDA states [highlight supplied] in part:

"Your first question is whether, from the viewpoint of EPA, the purpose of a 1979 Memorandum of Understanding (MOU) between EPA and the Federal Drug Administration (FDA) was 'to take away from FDA, and give to EPA, responsibility for regulating public drinking water additives intended for preventative health care purposes and unrelated to contamination of public drinking water?' Your second question is whether, if that was the purpose of the 1979 MOU, the MOU was terminated through a subsequent Federal Register notice.

"The answer to your first question is no, so there is no need to address your second question. The purpose of the MOU was not to shift any responsibilities between the Agencies. Rather, it was to help facilitate effective coordination of our respective legal authorities. . . . EPA does not have responsibility for substances added to water solely for preventative health care purposes, such as fluoride, other than to limit the addition of such

substances to protect public health or to prevent such substances from interfering with the effectiveness of any required treatment techniques. . . . The Department of Health and Human Services (HHS), acting through the FDA, remains responsible for regulating the addition of drugs to water supplies for health care purposes.

"The 1979 MOU was intended to address contamination of drinking water supplies as a result of direct or indirect additives to drinking water, not to address the addition of substances solely for preventative health purposes. . . ."

The basis for the Board's motion to deny our petition in 2010 is, in part, jurisdictionally incorrect. The FDA, not the CDC or EPA has jurisdiction over substances used with intent to prevent disease.

2. The Board's Denial of our petition to gain FDA CDER approval also includes:

"The Board has authority . . . to adopt rules for Group A public water supplies 'necessary to assure safe and reliable public drinking water and to protect public health.. . ."

"RCW 57.08.012 gives each water district the authority to decide whether to ask the electors of the water district to vote on adding fluoride to its tap water. The Board does not appear to have authority to adopt rules related to a water district deciding whether to fluoridate. The Board's authority is to regulate allowable concentration levels and method of approval of water additives.

The Board appears to accept jurisdiction to adopt rules for Group A public water supplies, and we agree.

However, *"the state board of health shall:*

*(a) Adopt rules for group A public water systems, as defined in RCW **70A.125.010**, necessary to assure safe and reliable public drinking water and to protect the public health."*

We will demonstrate below, fluoridation is not safe.

3. The Board's letter continues:

"The Board has adopted under WAC 246-290-460 an allowable concentration range for artificial fluoridation of public tap water. This range is 0.8-1.3 ppm and is based on the Centers for Disease Control and Prevention (CDC) "optimal" recommended levels to help prevent tooth decay."

The Board of Health has accepted concentration but not dosage. Concentration is not dosage because some drink little or no water, others 10 times the mean and not everyone is the same size or health, intraspecies variation. Determination of safety cannot be based on concentration.

The Board is mistaken when relying on the CDC to determine the "optimal" range or dosage of any drug with intent to prevent disease. CDC has no authority to recommend any unapproved drugs.

The letter goes on in detail on why fluoridation is set at a target of "0.7-1.2 ppm to help prevent cavities" and the Board's standard at 0.8-1.3 ppm in WAC 246-290-460. Clearly, the "intent

of use" defines fluoride as a drug which is unapproved and unapproved drugs are illegal and regulated by the FDA CDER, NOT the CDC or EPA..

Even if fluoridation were effective at 1 ppm, what evidence does the Board have that fluoridation is effective at 0.7 ppm? The Board claims a historical 25% reduction in dental caries at 1 ppm fluoride in water. What evidence does the Board have the same effect happens at 0.7 ppm?

The letter of denial continues, listing endorsements from the CDC, surgeon general, and dental lobby Ned Therien and William Bailey.

As you watch Fluoride On Trial: The Censored Science on Fluoride and Your Health | Childrens Health Defense

note the Dental director of the CDC when under oath was unable to cite research demonstrating efficacy of fluoridation. No evidence of efficacy? He was prudent and correct.

Neither efficacy, dosage, safety or label is the job of the CDC and has not been approved by the FDA. The director ended the questions before he was taken down a rabbit hole of problems

on the lack of evidence of fluoride ingestion's efficacy. The Director saved the CDC's "bacon" by not suggesting he had evidence to support fluoridation. He does not have good evidence.

Our point: The Board would be wise to no longer reference the CDC or EPA for efficacy of fluoride ingestion.

4. The Board's letter of denial continues with the 1979, EPA, FDA MOU as discussed above and supported by our attachment #F. The Board talked to Ned Therien EPA and John Kelsey DDS at the FDA. Both confirmed EPA regulates water. (silence on drugs)

The Board did not appear to push John Kelsey and specifically ask whether diluting a drug in tap water removes FDA CDER jurisdiction and places the jurisdiction with the EPA. Indeed, EPA regulates tap water, FDA CDER regulates drugs.

When the tap water is used to make a drug, the FDA CDER still has jurisdiction over the drug. See our attachment #F EPA letter.

5. The Board's letter of denial continues with support for the mass medication of everyone without consent based on **endorsements** from the dental lobby and those profiting from

fluoride sales. What science on efficacy, dosage, safety and label does the Board provide? The Board is silent.

6. The Board's letter (#G) of denial continues:

- *"EPA is lead federal agency for regulating maximum levels of contaminants and additives in tap water under the Safe Drinking Water Act."*

Yes, for maximum levels of the fluoride contaminant, but not to determine the efficacy of the drug or for drug manufacturing oversight. Simply diluting a drug in tap water does not change jurisdiction to the EPA.

The Safe Drinking Water Act states*:*

"No national primary drinking water regulation may require the addition of any substance for preventive health care purposes unrelated to contamination of drinking water."

I wrote to the EPA to ask for their understanding of that section of the SDWA, and the EPA responded:

"The Safe Drinking Water Act prohibits the deliberate addition of any substance to drinking water for health-related purposes other than disinfection of the water." HQ-FOI-01418-10

Our point here in simple terms: **"The Board must not promote what the SDWA prohibits."**

8. The Board's denial letter continues:

- *"FDA has relinquished any authority it might have for regulating fluoride levels in tap water under the memorandum of understanding with the EPA"*

See our attachment #F EPA water law office: . . . . *The Department of Health and Human Services (HHS), acting through the FDA, remains responsible for regulating the addition of drugs to water supplies for health care purposes."*

The Board assumes the FD&C Act permits the FDA CDER to delegate authority for drug approval and regulation to any agency, let alone an unauthorized agency.

9. The Board's Denial states:

- ***"The Board cannot direct a federal agency to take action."***

Our petitions have never petitioned the Board to direct a federal agency to take any action. A "New Drug Application" is not "direction" and this petition should, but as a compromise does not request the Board to make NDA or require the water purveyors who are the final drug manufacturers to gain NDA. This petition focuses on assuring safety.

10 The Board's Denial states:

- *"The State Board of Pharmacy has stated it cannot regulate tap water fluoridation under its authority."*

Our petitions have not asked the Board of Pharmacy to regulate tap water but to designate fluoride as a poison as provided by RCW. The Board of Pharmacy did not play games with us. The Board of Pharmacy was professional and went directly to the focal intent of our request. Fluoride is a legend drug.

11. The NRC 2006 report:

- *"An NRC committee evaluated the scientific evidence of the health effects of fluoride in drinking water and published a report in 2006 that concluded fluoride levels in drinking water below 2 ppm are safe for health."*

The Board does not provide a correct understanding of the NRC 2006 report. Dr. Robert J. Carton, PhD, with over 30 years writing regulations for the federal government and worked for 20 years at the EPA wrote the first regulations on asbestos. His review of the NRC 2006 report is the most concise and clear review.

"The committee apparently believed that it was their mission to identify only health effects known with total certainty. . .
Dental fluorosis: *the committee agreed it is a "dose-related mottling of enamel, which is permanent once a child's teeth are formed. It is described as a toxic effect. . . . taking moderate dental fluorosis into account, the MCLG would be lower than 0.7 mg/L "*

Bone Fractures: *Hip fractures above 1.5 mg/L. "What is not discussed is the magnitude of the safety factors necessary to insure protection from anticipated adverse health effects."*

Skeletal Fluorosis*: EPA used Stage III severe fluorosis as a baseline, the NRC 2006 committee included Stage II as an adverse health effect. "Thus we have a possibility of Stage I and Stage II occurring with a daily dose over a lifetime of 1.42 mg and 2.86 mg, respectively. These are both within the range of current fluoride exposures from all sources documented in the NRC report."*

Endocrine Effects: *Decreased thyroid function, impaired glucose tolerance (Type II diabetes), and earlier sexual maturity. The Executive Summary of the report merely states that these effects are achievable with fluoride concentrations in drinking water of 4 mg/L or less.. . .*

NRC report summary at the end of the chapter, "In humans effects on thyroid function were associated with fluoride exposures. . . 0.01-0.03 mg/kg/day when iodine was inadequate."

Mean intake of water is1 liter of water a day at 0.7 mg/kg is 0.7 mg. However, pregnant mothers do and should drink more, often 3 liters/day and some drink 10 liters/day. The additional fluoride hits the fetus exactly at a most vulnerable time for the developing brain.

Assuming half the total fluoride intake is from water and half from other sources, intake would be 1.4 mg/day for the mean and almost 3 mg/day for a pregnant mom. A 70 kg person would ingest between 0.2 – 0.4 mg/kg/day, well within the effects of fluoride intake for many if not most people. What about the 90th

percentile drinking 2L/day or those drinking 10L/day? And what about those ingesting more toothpaste or having a general anesthesia? All of those are far outside the statistical mean. And no margin of error or safety factor is included.

The dental lobby had and has not seriously researched safety for most health risks. An authoritarian claim of "safe and effective" had everyone trusting each other and no one researched to be sure.

Carton continues:

"Thus, there exists a lowest observed effect level of 0.06 mg/L of fluoride to develop an MCLG using the preventative approach of the Safe Drinking Water Act. . . An appropriate safety factor does not have to be mentioned to see clearly that fluoridation at 1 mg/L cannot be considered acceptable for an MCLG."

Carton ends his conclusion: *"Using the preventive public health intent of the law, the Maximum Contaminant Level Goal for fluoride in drinking water should be zero."*

Our point: to protect the public from harm, the Board of Health should recommend the cessation of fluoridation.

12. Back to the Board of Health's denial of our petition:

- ***"EPA announced completion of a review of MCLs . . . that concluded it did not have evidence to revise the MCL for fluoride."***

That is politically true, but not based on their scientific evidence.

Instead of protecting the public, EPA did the opposite of the NRC 2006 recommendation and protected fluoride by changing the definition of "safe" and eliminating many high-risk individuals. Even then, their data does not demonstrate safety.

The Board fell into the trap of political jargon, lacking empirical evidence.

13. The denial letter continues:

- *EPA will be conducting additional reviews regarding fluoride levels in drinking water.*
- *EPA recognizes NSF/ANSI Standard 60 as appropriate for the approval of drinking water additives*
- *The range of 0.8 ppm to 1.3 ppm fluoride in WAC . . . .*

The current TSCA court trial and the NTP report have brought out additional political pressure from the dental/industry lobby, blocking the protection of the public health. The video we asked you to watch covers some of the political influence.
NTP report on developmental neurotoxic effect is covered below.

14. The Board's denial letter concludes:

The Committee further recommends the next time the Department undertakes a major review of chapter 246-290 WAC, it consider proposing the word "optimal" in section 460(3) be changed to a phrase such as "generally regarded as safe." The Committee further recommends the Board continue to review legal points raised in the petition concerning state law and Attorney General opinions.

Those recommendations do not appear to have been followed.

The letter and Board's web page now clearly states the intent of administering fluoride is to reduce dental caries, claiming fluoridation reduces dental caries by 25%. The health claim confirms intent and intent confirms jurisdiction is with the FDA CDER.

The Board relies on the fluoridation lobby and does not provide empirical evidence to support their claim of health benefit. However, the preventive health claim confirms the Board knows fluoride, because of intended use, Board of Pharmacy, FDA CDER, and listed in the U.S. Pharmacopeia, is a drug, legend drug. See: Washington State Board of Pharmacy, RCW 18.64.011(14) and 21 U.S. Code § 321 and FDA

THE ETHICS OF VOTING TO MEDICATE OUR NEIGHBORS

What does the Legislature mean to “assure safety?”

The Oxford Dictionary defines assure:

“tell someone something positively or <u>confidently</u> to <u>dispel</u> any <u>doubts</u> they may have.”

The Legislature did not require absolute proof of harm or even mention efficacy (benefit).

The Legislature charged the Board to be confident the water is safe and, in effect, be able to assure the public without doubt the water is safe.

Board members need to be able to publicly say, “I have reviewed all streams of evidence and can assure the public fluoridation is safe for everyone. And if the Board members are not confident that they can dispel all doubts in their own minds that the water is safe for everyone, then the Board must take steps to assure safety of the water. That is your ethical job, your mandate. We the public are relying on your confidence, that you have reviewed laws and science. The job is not to be delegated, that is your job to assure the public the water is safe.

We have covered parts of ethics in each section. Voters rely on water commissioners, the Board, and Department to dig into the evidence with due diligence to assure safety.

THE BOARD OF HEALTH'S EXISTING RULES DO NOT ASSURE SAFETY

Health must be built on science.

The Board of Health has no caution, warning, label, dosage, or safety evidence. Safety for all is not assured.

The product is misbranded, adulterated, and contaminated. This petition takes the first step to protect some in the public with a simple label. This petition is a compromise.

The Board of Health would be correct to advise the manufacturers of fluoridated water, purveyors, such as Seattle, to at a minimum place a label on the product (billing, etc.)

If Seattle applied to the FDA, and the fluoridated drug approved, Seattle could then patent the product and make enough money to house the homeless and pay for their dentistry and

more. Circumventing the law is costly to the public's health and finances.

Our point: The Board must first assure safety in their own minds for their own family and then assure safety for everyone else. Remove your endorsement of fluoridation off your website.

*"According to the **U.S. Food and Drug Administration (FDA)**, unapproved prescription drugs pose significant risks to patients because they have not been reviewed by FDA for safety, effectiveness or quality. Without FDA review, there is no way to know if these drugs are safe and effective for their intended use, whether they are manufactured in a way that ensures consistent drug quality or whether their label is complete and accurate[1].*

If your doctor prescribed a non-FDA approved drug, it is important to discuss the risks and benefits of the drug with your doctor. You may also want to ask your doctor if there are any FDA-approved drugs that could be used to treat your condition. If you have suffered serious side effects from a non-FDA approved drug, you may have a claim against your physician and the drug manufacturer[2].
Please note that the FDA permits some unapproved prescription drugs to be marketed if they are relied on by health care professionals to treat serious medical conditions when there is no FDA-approved drug to treat the condition or there is insufficient supply of FDA-approved drugs[1]." Reference 1 is fda.gov, 2 is liljegrenlaw.com[13]

[13] This quote appears to be a Bing AI generated report to my question regarding the prescribing of unapproved drugs. A correct statement, but not generated by me.

SCIENCE:

DENTAL CARIES ARE NOT HIGHLY LETHAL OR CONTAGIOUS.

Dental caries is very common, can become very painful, disfiguring, disabling, but are not considered highly lethal nor contagious and treatment is usually considered elective.

Fluoride is not considered an essential nutrient and has no physiologic or minimum daily requirement.

Public Health Authorities have police powers to prevent highly contagious and lethal diseases from harming and spreading throughout the public. As we have seen with the COVID vaccinations, the public has serious reservations when asked to blindly trust my public health profession, even with approved drugs for highly lethal contagious diseases.

Our point: Dental caries is not considered highly contagious or lethal, I was taught dental treatment is almost always elective.

A. Recommended Dosage

Without FDA approval for efficacy, dosage is speculation and unknown.

"The recommended optimal fluoride intake for children to maximize caries prevention and minimize the occurrence of dental fluorosis is often stated as being 0.05-0.07 mg/kg/day." (Levy 1994; Heller et al. 1999, 2000).

Burt (1992) attempted to track down the origin of the estimate of 0.05-0.07 mg/kg/day as an optimum intake of fluoride but was unable to find it." National Research Council 2006 p 68. See a Review by Carton a former EPA scientist.

"Hodge (1950) studied children consuming fluoride in their drinking water. Fluoride levels of 0-14 ppm were investigated. Dental mottling was the parameter of interest. Fluoride levels of 2-10 ppm produced a linear dose- response curve (increasing mottling with increasing dose). Fluoride levels of 0.1-1.0 ppm produced no observable effect. An assumption of 20 kg bw and 1 L/day water consumption for children was used, since the children studied were 12-14 years old. It is further assumed that a 20-kg child consumes 0.01 mg of fluoride/kg bw/day in the diet (50 FR 20164). Thus, a total intake would be approximately 0.06 mg/kg/day. " http://www.epa.gov/IRISsubst/0053.htm#oralrfd

B. As a side note, the EPA has used 0.06 mg/kg/day as their reference dose for the fluoride contaminant in water until about 2010. The NRC 2006 report on fluoride in water (covered in more detail below) told the EPA their MCL was not protective. Instead of protecting the public, the EPA changed their definition of safe, "RfD" or safe dose to 0.08 mg/kg//day, the opposite recommended by the EPA.

Changing the definition, doing the opposite of the NRC 2006 recommendation, did not change the science or assure safety.

C. The fetus, infants, and those drinking more than the 90th percentile were ignored. The only possible risk considered publicly in 1950 was severe dental fluorosis. But they knew much more as evidenced by the release of classified documents from the time. Watch: the Fluoride On Trial: The Censored Science on Fluoride and Your Health | Childrens Health Defense and the NTP 2023 report on fluoride. Ignoring 10% of the population does not assure safety.

D. HHS ASTDR in 2003 suggested infants AI (Adequate intake) be 0.01 mg/day or 0.0014 mg/kg/day, the same as recommended in 1950. (See IOM's Table 2-1)

Mean concentration of mother's milk has been reported at 0.004 mg/L for samples where fluoride was detected, reasonably consistent for infants as suggested by HHS ASTDR.

How much fluoridated water is 0.0014mg/kg/day for a 3 kg (6.6 pound) new born exclusively on formula 3 kg X 0.0014 mg = 0.0042 mg. 0.7 mg/L fluoride in water divided by 0.0042 is 0.006 L of water or about 2.9 teaspoons of food made with fluoridated water per day for the infant.

Our point: An infant needs more than 2.9 teaspoons of food a day. Note: The Institute of Medicine's AI is "Adequate

Table 2-1. Adequate Intake Levels for Fluoride[a]

Age range	Adequate intake level (mg/day)	Adequate intake level (mg/kg/day)[b]
0–6 months	0.01	0.0014
6–12 months	0.5	0.056
1–3 years	0.7	0.054
4–8 years	1	0.045
9–13 years (males and females)	2	0.05
14–18 years (males)	3	0.046
14–18 years (females)	3	0.053
>18 years (males)	4	0.052
>18 years (females)	3	0.049

[a]Source: IOM 1997
[b]mg/kg/day doses were calculated by using reference body weights reported by IOM (1997)

Intake" and does not reflect a safe dosage and the AI was their best guess/estimate assuming fluoride was effective.

E. Mother's milk provides about 150 to 250 times **less** fluoride than formula made with water at "optimum" fluoride concentrations. In other words, infants bottle fed formula made from fluoridated water have the greatest risk of being overdosed with fluoride.

F. What about the fetus? Although the mother's body protects their milk and infant from significant fluoride, in contrast, fluoride passes through the placenta to the fetus and has been measured in fetal brain. Although the Board claims fluoridation safety has many studies, in reality, not much research is available on the effect of fluoride to every cell, tissue, organ and system of adults, let alone the fetus.

The fetus has another source of fluoride. Human bone retains fluoride and the concentration increases with age. Ranges I've seen are 1,000 ppm (similar to toothpaste at 1,500 ppm) to 8,000 ppm reported in cancer patients.

The bone resorbs (osteoclasts) and builds up (osteoblasts) throughout life. The half-life of fluoride in bone is about 20 years. In other words, if a person stopped all fluoride intake for 20 years, the fluoride concentration in the bone would be about half.

The fetus during the final trimester of life needs lots of calcium and in a deficient intake of calcium, the mother's bones resorb to provide the calcium. As the bone is broken, fluoride is released and increases the burden of fluoride on the fetus at the same time the fetal brain is developing.

The fetal brain goes through essential stages of development. If the stages are interrupted, the brain may never recover and fully develop.

For optimal development of the brain, the mother should start out with a low fluoride bone concentration.

Our petition takes this source of fluoride into consideration and we recommend the mother have low fluoride exposure starting at least 20 years prior to pregnancy.

More on this below

G. **Too many are ingesting too much fluoride,** as evidenced by 2 out of 3 children showing a biomarker of having ingested too much fluoride, dental fluorosis,[14] and the EPA's Dose Response Analysis for Non Cancer Effects and Fluoride Exposure Relative Source Contribution of 2010. EPA Figure 8-1 below is

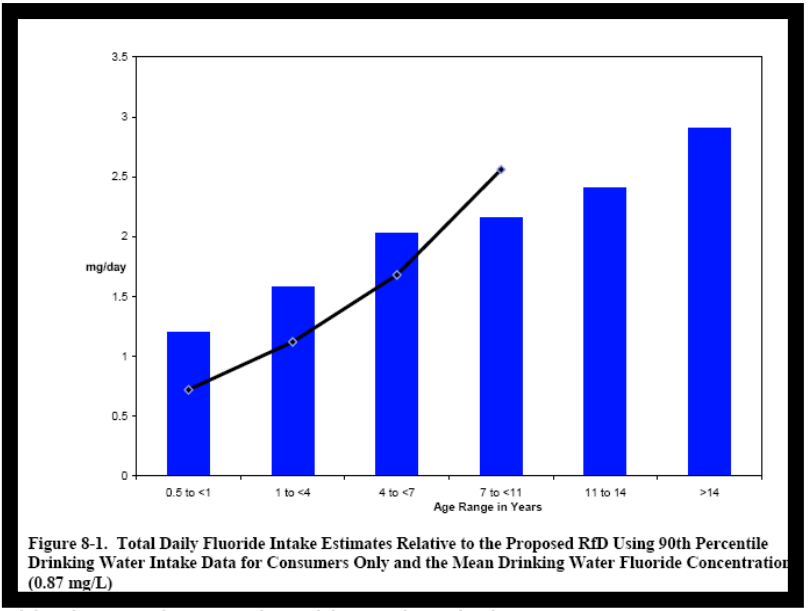


Figure 8-1. Total Daily Fluoride Intake Estimates Relative to the Proposed RfD Using 90th Percentile Drinking Water Intake Data for Consumers Only and the Mean Drinking Water Fluoride Concentration (0.87 mg/L)

critical to understand and keep in mind.

[14] Neurath C, Limeback H, Osmunson B, Connett M, Kanter V, Wells CR. Dental Fluorosis Trends in US Oral Health Surveys: 1986 to 2012. JDR Clin Trans Res. 2019 Oct;4(4):298-308. doi: 10.1177/2380084419830957. Epub 2019 Mar 6. PMID: 30931722.

The proposed mean intake/dosage is shown in mg/day represented by the blue lines for each age group. The black line is the proposed (which was adopted) RfD (maximum safe dose) for each age.

#1. Note: about a third of infants 0.5 to <1 year of age are ingesting too much fluoride. The EPA's estimate indicates about 20,000 infants at this age are ingesting too much fluoride in Washington State.

#2. Note**: Infants, birth to six months of age are omitted, ignored, unprotected.** All under six months on formula made with fluoridated water would exceed the RfD.

RCW does not exempt infants under six months of age from Board protection. New parents are busy and should not be expected to do rigorous research on the toxicology of fluoride.

#3. Note: 10% of the public drinking the most water are not included, about 330,000 directly on fluoridated water and the "halo" effect reaches many more. EPA only includes up to the 90th percentile of the public in their calculations. The EPA/Board

is totally ignoring 10% of the 3.3 million drinking the most water. RCW does not exempt the Board from protecting thee people.

#4. The fetus is ignored. That is all of us. . . at one time. The most vulnerable infants are ignored by the EPA, unprotected. No wonder research demonstrates breast feeding is superior, lack of fluoride maybe one contributing factor.

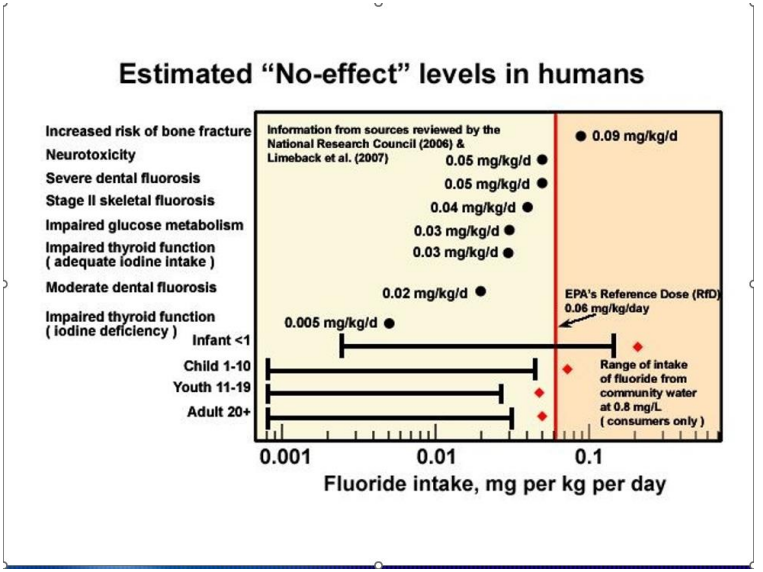
#5. Note: the "Proposed RfD" is a third higher. EPA was proposing a "safe" dosage from 0.06 mg/kg/day to 0.08 mg/kg/day and the new higher RfD, opposite the NRC 2006 recommendation, was adopted.

#6. And also remember, for Fluoride, the EPA's margin of error, uncertainty factor, intraspecies variation, is "0". The EPA is certain all humans fit in the "mean" or "average."

Our point: NRC (2006) said MCL is not safe. Instead of protecting the public, the EPA protected the contaminant and changed the definition to protect policy rather than the fetus, infants, and children. The EPA did the opposite of the NRC 2006 recommendation.

Parents give children 6 to 7 times more fluoride toothpaste than the recommended “pea size,” and 40% don’t know about the recommended amount of toothpaste.[15]

The NRC 2006 report estimated a “no-effect” level for humans about two decades ago with the following summarized evidence:



[15] Sudradjat, H., Meyer, F., Fandrich, P. *et al.* Doses of fluoride toothpaste for children up to 24 months. *BDJ Open* **10**, 7 (2024). https://doi.org/10.1038/s41405-024-00187-7

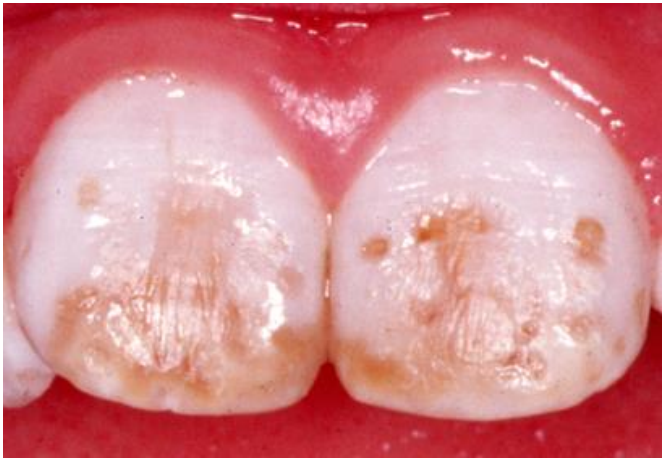
In 2006, we had fair evidence fluoridation was harming many with bone fractures, neurotoxicity, dental and skeletal fluorosis, impaired glucose metabolism, impaired thyroid function, moderate dental fluorosis and impaired thyroid function with iodine deficiency all within the range of fluoride exposure.

We brought these risks to the Board's attention in 2010 and the Board failed to protect the public. No wonder the EPA scientists said, through their union, fluoridation borders on a criminal act of governments.

EPA's THRESHOLD OF HARM

The EPA uses crippling skeletal fluorosis, like these people

Or pitting of teeth like this picture as the threshold of harm from fluoride ingestion.



Harm for the EPA does not start till severe structural damage has occurred.

Obviously, the EPA threshold of harm and the Board's, assuring safety, are not the same the same level of confidence.

The question the EPA fails to answer and the Board must answer,

"is there any harm detected before crippling skeletal fluorosis and severe dental fluorosis?"

The answer is a resounding "YES."

The fluoridation lobby dismisses the harm as "cosmetic blemish."

The EPA appears to refuse to consider any other risks from excess fluoride exposure even though they have paid researchers to provide the evidence.

Our point: The EPA must not be trusted to assure safety.

RCW instructs the Department to have aesthetic concerns as a threshold and in contrast the EPA has severe harm as a

threshold for concern. And RCW puts the threshold of harm at assuring without doubt fluoridation is safe.

Both aesthetic and health harm is reported from fluoride

The EPA in 2011 provided "Questions and Answers on Fluoride." None of the questions and answers deal with the effectiveness or effectiveness dosage of fluoride. Silence.

EPA does not weigh the benefit/risk of fluoridation. They simply protect the contaminant so those choosing may.

HOW MUCH FLUORIDE DOES A PERSON INGEST AND HOW MUCH WATER DO THEY DRINK?

Although the concentration of fluoride in water is well controlled, the amount of water ingested is highly variable and thus the dosage is highly variable.

In effect, the Board must NOT use the "statistical mean" or the EPA's RfD or the IOM's AI as a reasonable dosage of fluoride to protect everyone.

Foods can contain a significant amount of fluoride, especially some teas and foods such as mechanically deboned meat.[16]

The EPA and NRC (2006) reports the median intake of water is about 1 L/day. 90th percentile at about 2 L/day. Some drink over 10 liters/day. The NRC (2006) also reported **2-4 yr. olds ingest 0.125-0.3 mg fluoride per brushing, 2 times as much as from food and water combined and 75% more fluoride ingested for**

[16] Fluoride Content of Foods Made with Mechanically Separated Chicken | Journal of Agricultural and Food Chemistry (acs.org)
finalfluoridedatabase.pdf (tees.ac.uk)
Fluoride concentrations of infant foods - University of Iowa (uiowa.edu)

those who do not rinse. No wonder dental fluorosis, a biomarker of excess fluoride exposure has gone up to 70% of children.[17], [18]

This petition is to start protecting our most vulnerable.

Although water is most often the largest amount of individual fluoride exposure and toothpaste usually comes in second (or 1st), many other sources of fluoride affect individual exposure.

PROFUME: Ellen Connett has a brief history of a new fluoride product, Profume. Note: if a pesticide or drug has the letter "f" or letters "fu" in the name, it probably contains fluoride. The residue of fluoride on food when "Profume" is applied can be very high, although not all foods are treated. Her report includes:

[17] Neurath C, Limeback H, Osmunson B, Connett M, Kanter V, Wells CR. Dental Fluorosis Trends in US Oral Health Surveys: 1986 to 2012. JDR Clin Trans Res. 2019 Oct;4(4):298-308. doi: 10.1177/2380084419830957. Epub 2019 Mar 6. PMID: 30931722.

[18] Dong H, Yang X, Zhang S, Wang X, Guo C, Zhang X, Ma J, Niu P, Chen T. Associations of low level of fluoride exposure with dental fluorosis among U.S. children and adolescents, NHANES 2015-2016. Ecotoxicol Environ Saf. 2021 Sep 15;221:112439. doi: 10.1016/j.ecoenv.2021.112439. Epub 2021 Jun 22. PMID: 34166938.

“. . . EPA approved two “tolerances” (permitted levels in or on food): one for Fluoride levels and the other for Sulfuryl Fluoride levels. See the tolerances approved for food by US EPA as of July 15, 2005.

. . . FAN submitted comments and formal Objections and then in 2004 and 2005 EPA approved its use with high fluoride levels on all processed food, beans, grains, flour -and much more, including a fluoride residue of 900 ppm on dried eggs!

Incredibly, after many years of hard work, in January 2011, EPA concluded that it agreed with all but one of our objections and published their proposal to phase-out sulfuryl fluoride. According to protocol, EPA simultaneously solicited public comments on the phase-out. That was when the Dow Chemical Company, the proprietary owner of Sulfuryl Fluoride, did everything a powerful corporation can do to dissuade EPA from enacting the phase-out. They successfully lobbied Congress to add a few short sentences to the Farm Bill of 2014 that nullified the phase-out. . . .”

There are many sources of fluoride, water and dental products provide the most for many people. However, fluoride in foods such as mechanically deboned meat, tea, wine and medications, may provide significant dosages of fluoride to sub-populations.

GENERAL ANESTHESIA: especially for infants and children:

Characteristics of Anesthetic Agents Used for Induction and Maintenance of General Anesthesia
". . . desflurane (halogenated solely with fluorine halogenation increases potency and is essential to ensure nonflammability), halothane (halogenated with fluorine, chlorine, and bromine), isoflurane (halogenated with fluorine and chlorine), and sevoflurane (halogenated solely with fluorine). Halothane was the first fluorinated inhaled anesthetic that was wildly successful, rapidly displacing all other potent inhaled anesthetics. Efforts to develop other halogenated anesthetics with more of the characteristics of the ideal inhaled anesthetic agent than halothane led to the introduction of isoflurane, desflurane, and sevoflurane." Edgar

Our point: There are many sources of fluoride and each person is exposed to an unknown dosage.

LACK OF AN UNCERTAINTY FACTOR, MARGIN OF ERROR, AND/OR INTRASPECIES VARIATION

Some individuals are more at risk than others. For example:

Diet, such as a low iodine intake or calcium intake.
Kidney dysfunction, inability to excrete as fluoride
High water intake: athletes, diabetics, pregnancy
Socioeconomics
People of color
Age, fetus, infant, child, senior
Genetic Polymorphism, etc.

In contrast, the EPA/NIH and Board claim or imply fluoridation is so safe for everyone that a margin of error, uncertainty factor, intraspecies factor has been set at "1:1," in effect no margin of error or uncertainty factor or intraspecies variability. About 1.3% of the 3.3 million in Washington State are infants on formula and ¾ of them on formula made with water, or about 20,000 infants on formula made with fluoridated water.

"One size shoe" does not fit everyone, all munas are not at the "mean" or "average."

Condition of use is important for determining hazards and risk. Duration of fluoride is from conception (or before), frequency is several times a day and for the fetus constantly. The “halo” effect of fluoridated water shipped outside fluoridated communities must also be considered for those not on fluoridated water.

The exposure level and the hazard level is almost the same with no safety for at risk individuals.

To protect the public, an uncertainty factor of 10 and margin of error should be included, but has not. Not everyone in the public fits in the statistical mean. (NRC 2006) At a minimum, the EPA MCL should be 10% of their 4 ppm and on that item alone, fluoridation should not exceed 0.4 ppm.

The Board should not be surprised that the EPA scientists ethically spoke up with their concerns:

> *"In summary, **we hold that fluoridation is an unreasonable risk**. That is, the toxicity of fluoride is so great and the purported benefits associated with it are so small - if there are any at all – **that requiring every man, woman and child in America to ingest it borders on criminal behavior on the part of governments."***
>
> - ***Dr. J. William Hirzy, Senior Vice-President, Headquarters Union,***
> - ***US Environmental Protection Agency, March 26, 2001***

WAC 246-290-220 requires the Board of Health to have a more protective threshold of aesthetic issues, rather than the EPA's skeletal or dental disability. The Board must protect the public from aesthetic concerns which are long before severe harm occurs such as structural damage to teeth and crippling of the bones. EPA does not protect the public from harm or aesthetic concerns.

RCW 43.20.50 (1) instructs the board to "protect public health" with "safe and reliable public drinking water" but does not provide excuse for the board to recommend or promote the use of water, or to dispense an illegal drug, a prescription drug (Board of Pharmacy), or an "additive" with known aesthetic harm and without duly authorized designated oversight. Aesthetic harm is harm. If someone scratches your car, it may only be an aesthetic scratch, but it is still harm.

Our point: To assure safety, the statistical mean is not protective of many or most people. An uncertainty factor and margin of error of at least 10 should be used.

BENEFIT OF FLUORIDE INGESTION

Fluoridation is claimed to be one of public health's greatest achievements or blunders of the 20th Century, depending on whether profit or safety are considered.

A <u>recent study</u> reported a 2-3% reduction in dental caries over 20 years was just released in the UK involving millions of subjects.

Systemic Fluoride has theoretical benefit while the enamel is developing. NRC 2006 & HHS HTSDR 2003 p 9

". . . fluoride prevents dental caries predominately after eruption of the tooth into the mouth, and its actions primarily are topical for both adults and children…"[19] CDC

Keep in mind, about 60-70% of the population show signs (biomarker) of excess fluoride, dental fluorosis, which is caused from ingestion of fluoride prior to eruption of the tooth. CDC says

[19] CDC (1999). Achievements in Public Health, 1900-1999: Fluoridation of

Drinking Water to Prevent Dental Caries. MMWR, 48(41); 933-940, October 22.

benefit is primarily topical after tooth eruption. FDA has approved topical but not ingestion.

Dental saliva has about 0.019 ppm of fluoride and contact time is minimal, so it would not have much if any benefit. Studies report toothpaste below about 1,000 ppm does not show benefit. Swishing with fluoridated water is unlikely to provide significant therapeutic value.

LACK OF KNOWN MECHANISM OF ACTION

The tooth is highly resistant to the migration of fluoride. Fluoride does not flow from the pulp through the tooth to the outside of the enamel where the caries are developing. No rational mechanism for systemic fluoride benefit has been suggested. See more below.

The FDA's determination the evidence for fluoride's efficacy is incomplete has been supported with other studies.[20] [End note]

- [20] **"** Fluorosis prevalence increased significantly with higher water fluoride levels; however, caries prevalence did not decline significantly."

Hong L, Levy S, Warren J, Broffit B. (2006). Dental caries and fluorosis in relation to water fluoride levels. *ADEA/AADR/CADR* 2006.

- **"**No fluoride, socioeconomic status or beverage variables were significantly associated with lesion progression.

Warren JJ, Levy SM, et al (2006). Longitudinal study of non-cavitated carious lesion progression in the primary dentition. *JPHD* 66(2):83-7.

- **"**In the present study, fluoridated water did not seem to have a positive effect on dental health**. . .** *Community Dentistry Oral Epi* 34:63-70
- "The WHO data do not support fluoridation as being a reason for the decline in dental decay in 12 year olds that has been occurring in recent decades."

Neurath C. (2005). Tooth decay trends for 12 year olds in nonfluoridated and fluoridated countries. ***Fluoride*** 38:324-325

- **"**Our analysis shows no convincing effect of fluoride-intake on caries development." Komarek A, et al. (2005). ***Biostatistics*** 6:145-55.
- **"**Levels in fluoridated and non-fluoridated areas were similar. " Harding MA, et al. (2003). ***Community Dental Health*** 20(3):165-70.
- **"**There was no statistically significant difference between DMFT in municipalities of the same size, regardless of the presence or absence of fluoride

in the water supply..." Sales-Peres SH, Bastos JR. (2002). [An epidemiological profile of dental caries in 12-year-old

- Water fluoridation status of the children's area of residence did not have a significant effect on Early Childhood Caries (ECC)**."** Shiboski, et al. (2003).
- "[E]ven a longitudinal approach did not reveal a lower caries occurrence in the fluoridated than in the low-fluoride reference community." Seppa (2002).

- The magnitude of [fluoridation's] effect is not large in absolute terms, is often not statistically significant and may not be of clinical significance." Locker.

(1999). Benefits and Risks of Water Fluoridation. An Update of the 1996 Federal-Provincial Sub-committee Report. ***Ontario Ministry of Health***

- "[R]esults of recent large-scale studies in at least three countries show that, when similar communities are compared and the traditional DMFT index

of dental caries is used, there is no detectable difference in caries prevalence. **This has been demonstrated for school children in the major cities of New Zealand, Australia, the US and elsewhere."** Diesendorf, M. et al. (1997). New Evidence on Fluoridation. ***Australian and New Zealand Journal of Public Health***. 21: 187-190

- **Higher fluoride proportions appeared to be associated with lower dfs + DFS, with an estimated difference between fluoridated and non-fluoridated groups of 0.65 decayed or filled surfaces per child, but this** association was not statistically significant. The

effects of fluoridation on the other outcomes were small and not statistically significant." Domoto P, et al. (1996). ***JDR*** 75:1947-56

- **"**Children attending centers showed no significant differences (in baby bottle tooth decay) based on fluoride status. ***Public Health Reports*** 107: 167-73
- **The fluoride incorporated developmentally – that is, systemically into the normal tooth mineral – is insufficient to have a measurable effect on acid solubility." Featherstone JDB, M.Sc., Ph.D. , Cover Story; *J American Dental Association*, Vol. 131, July 2000, p. 890.**
- **Centers for Disease Control; *MMWR Weekly Report.* 1999;48:933-940. "laboratory and epidemiologic research suggests that fluoride prevents dental caries predominately after eruption of the tooth into the mouth, and its actions primarily are topical for both adults and children."**

3 FALSE CLAIMS ON THE Board's website

#1. The Board claims: "For water systems serving 20,000 people or more, every $1 invested in fluoridation saves $38 in dental treatment costs." No reference provided.

Cost of **HARM** is not included.

The Board's claim does not include the real-world costs of fluoridation, supplies, equipment, wages, and all manufacturing costs and avoids any costs to treat harm.

DENTAL FLUOROSIS:

I have treated dental fluorosis for more than 4 decades and made hundreds of thousands of dollars off of fluoride. I assumed the good outweighed the bad. I was wrong. If there were no other risk than dental fluorosis, the Board should at a minimum accept our petition for rule change.

COMPLAINT NOTICE: This petition is notice and registering a complaint of dental fluorosis harm.

WAC 246-290-220 "(5) The department may accept continued use of, and proposals involving, certain noncertified chemicals or materials on a case-by-case basis, if all of the following criteria are met:

(b)There exists no substantial evidence that the use of the chemical or material has caused consumers to register complaints about aesthetic issues, or health related concerns, that could be associated with leachable residues from the material;"

There is no dispute, fluoride causes dental fluorosis and fluoridation increases dental fluorosis. There is no dispute fluoridation increases "**aesthetic issues,"** long before severe skeletal fluorosis. NHANES survey reported about 2 out of 3 children with dental fluorosis.

FLUORIDATION IS NOT COST EFFECTIVE: The cost of treating dental fluorosis harm is almost never included in a cost benefit analysis.

As a treating clinician, having made many hundreds of thousands of dollars treating dental fluorosis both aesthetic and functional, I do not understand how those in ivory towers have failed to include the cost of harm from just dental fluorosis when considering the cost effectiveness of fluoridation.

Perhaps they assume fluoride only comes from fluoridation. And they assume no risk or harm except slight tooth blemishes. Another possible reason is dentists, blocked by the American Dental Association, is the only health care profession not obligated to document any diagnosis. Even if we had to document a diagnosis, we sometimes do not reasonably consider the etiology of the pathology.

Dental fluorosis is a biomarker of excess fluoride exposure.

A US Environmental Protection Agency (EPA) study[21] (1987)., funded by the EPA with fluoride concentrations between

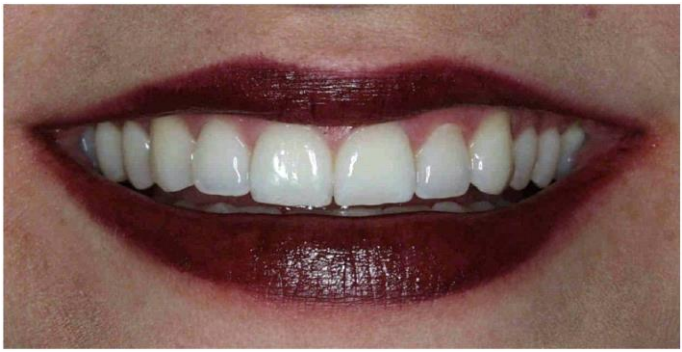
[21] Collins, E., V. Segreto, H. Martin, AND H. Dickson. ANALYSIS OF COSTS FOR THE TREATMENT OF DENTAL FLUOROSIS. U.S. Environmental Protection Agency, Washington, D.C.,

1.0-4.0 mg/L evaluated the cost of treating dental fluorosis, finding:

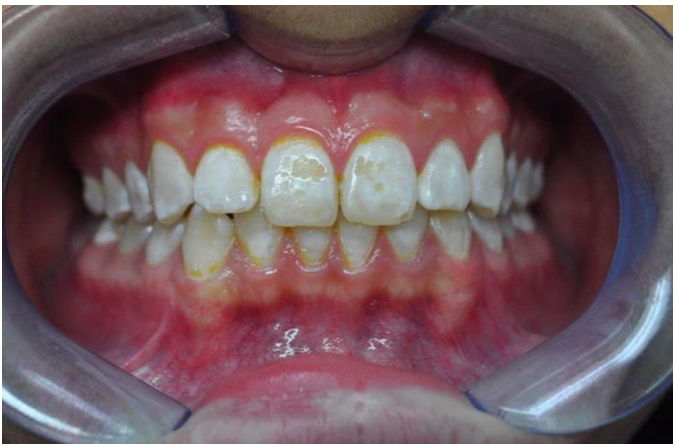
"A mean cost for all consultants shows that the estimated costs for restoring function exceeds the cosmetic costs in all categories except the minimum later costs. This represents a new finding and raises an issue that has been overlooked or ignored by previous investigators and the profession. i.e. that repair of the cosmetic discoloration was the only cost involved; or that repair of dysfunction was never considered to be a problem."

Functional harm, pits, fractures, chips, are one reason we do fillings and crowns, which may cost more than the cosmetic damage. However, as a dentist when I was young, I would see teeth with pitting or fractures and not blame my fluoride, I would blame the patient for not proper diet and cleaning, chewing ice, biting rocks, anything but fluoride.

Here is an example of teeth without fluorosis.



Here is an example of severe dental fluorosis.



His mom was certain he only had fluoridated bottled water and no fluoride toothpaste when he was young.

Dentists placing black mercury fillings are not always on the same page as our patients when it comes to aesthetics. In one study[22] of 12 year-old adolescents, 52% reported dental fluorosis at 0.7 ppm fluoride in water. Of those, 95% wished to

[22] Moimaz SA, Saliba O, Marques LB, Garbin CA, Saliba NA. Dental fluorosis and its influence on children's life. Braz Oral Res. 2015;29:S1806-83242015000100214. doi: 10.1590/1807-3107BOR-2015.vol29.0014. Epub 2015 Jan 13. PMID: 25590503. [PubMed]

remove the spots. In contrast only 14.5% had professionally diagnosed dental fluorosis.

Suppose someone took a key and scratched a line on your car. The car would drive fine, but the scratch is damage and you should be compensated.

The Department should not endorse and recommend fluoridation which is known, without dispute, to cause aesthetic damage to teeth. The Board must assure safety and dental fluorosis is damage.

If dental fluorosis, a known risk of excess fluoride, were the only risk, and if the Board wanted to assure safety of fluoridation, the Board would recommend tap water not have fluoride added.

ESTIMATED Cost to fluoridate water $3-$10 PPPY (Per Person Per Year) Ko and Thiessen

Averted caries (money saved)	$6.08 PPPY
Dental fluorosis Treatment[23]	$3.24-$153 PPPY

[23]Previously, I provided the basis for these estimates to the Board. If you would like the references and math, let me know.

Fluoridation is not cost effective if only damge from dental fluorosis is included.

Consider the study by Maupome, HMO's over 90,000 cohorts,

"Community water fluoridation was associated with reduced total and restorative costs among members with one or more visits, but the magnitude and direction of the effect varied with locale and age and the effects were generally small. In two locales, the cost of restorations was higher in nonfluoridated areas in young people (<age 18) and older adults (>age 58). In younger adults, the opposite effect was observed. The impact of fluoridation may be attenuated by higher use of preventive procedures, in particular supplemental fluorides, in the nonfluoridated areas."

Maupome squeaked out as much positive as possible and reported the cost savings was negated if only part of the costs of fluoridated materials and equipment repairs were included. No costs for treatment of functional or aesthetic harm, brain damage, thyroid damage or any other risk was included. Looking at his data and children in the non-fluoridated had lower dental costs.

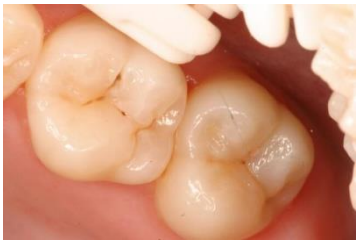
"Harm is the cost, not the treatment."

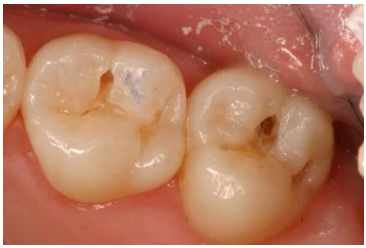
Ko 2014 *The U.S. Government states that $1 spent on CWF saves $38 in dental treatment costs. . . . Recent economic evaluations of CWF contain defective estimations of both costs and benefits. Incorrect handling of dental treatment costs and flawed estimates of effectiveness lead to overestimated benefits. The real-world costs to water treatment plants and communities are not reflected. . . .* ***Conclusions :*** *Minimal correction reduced the savings to $3 per person per year (PPPY) for a best-case*

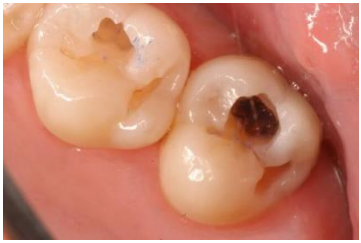
scenario, but this savings is eliminated by the estimated cost of treating dental fluorosis."

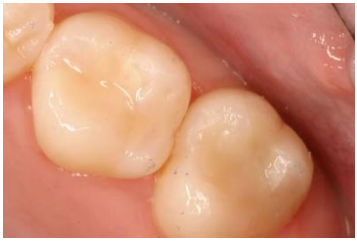
For example, the Board accepts labor costs between $7 and $9/hour while real world labor is closer to $100/hour. And no risk or harm or cost of treating harm is factored in for the Board'a claim of cost effective.

Below is a patient of mine with early functional dental fluorosis. The teeth look great, nice shiny hard enamel, just a touch of early caries. If the patient had not had fluoride, the enamel might not have been so hard and would have probably broken away sooner and pathology diagnosed sooner, and thus with less depth of caries. We call this the "fluoride bomb."

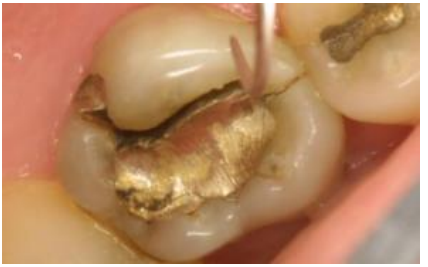


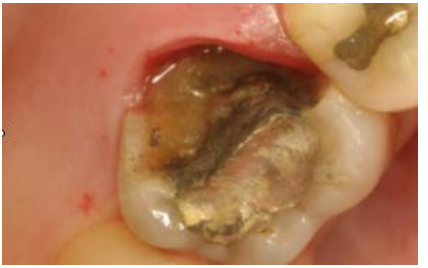






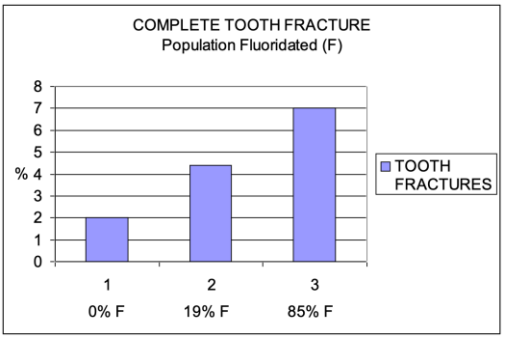
The fluoride hardens the teeth and like bones they become more brittle, like this:





Both systemic and topical fluoride excess may increase harm which has not been included in most cost benefit analysis of fluoridation.

I found a couple authors reporting “complete cusp fractures” and more than 300% increase in fractures in the 85% fluoridated community vs the community lacking fluoridation. See graph below.



Increased fluoride exposure can also increase dental caries.[24] If there is a "sweet spot" of fluoride dosage exposure to prevent caries, the spot is not hard to detect.

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[24] Awadia AK, et al. (2002). Caries experience and caries predictors - a study of Tanzanian children consuming drinking water with different fluoride concentrations. *Clinical Oral Investigations* (2002) 6:98-103. (See abstract)
Binbin W, et al. (2005). Dental caries in fluorine exposure areas in China. *Environmental Geochemistry and Health* 27:285-8. (See abstract)
Budipramana ES, et al. (2002). Dental fluorosis and caries prevalence in the fluorosis endemic area of Asembagus, Indonesia. *International Journal of Paediatric Dentistry* 12(6):415-22. (See abstract)
Ekanayake L, Van Der Hoek W. (2002). Dental caries and developmental defects of enamel in relation to fluoride levels in drinking water in an arid area of sri lanka. *Caries Research* 36(6):398-404. (See abstract)
Grobleri SR, et al. (2001). Dental fluorosis and caries experience in relation to three different drinking water fluoride levels in South Africa. *International Journal of Paediatric Dentistry* 11(5):372-9. (See abstract)
Grobler SR, van Wyk CW, Kotze D. (1986). Relationship between enamel fluoride levels, degree of fluorosis and caries experience in communities with a nearly optimal and a high fluoride level in the drinking water. *Caries Research* 20:284-8.
Mann J,et al. (1990). Fluorosis and dental caries in 6-8-year-old children in a 5 ppm fluoride area. *Community Dentistry and Oral Epidemiology* 18(2):77-9. (See abstract)
Mann J, et al. (1987). Fluorosis and caries prevalence in a community drinking above-optimal fluoridated water. *Community Dentistry and Oral Epidemiology* 15(5):293-5. (See abstract)
Olsson B. (1979). Dental findings in high-fluoride areas in Ethiopia. *Community Dentistry and Oral Epidemiology* 7(1):51-6. (See abstract)
Ramseyer WF, et al. (1957). Effect of Sodium Fluoride Administration on Body Changes in Old Rats. *Journal of Gerontology* 12: 14-19. (See excerpt)
Retief DH, et al. (1979). Relationships among fluoride concentration in enamel, degree of fluorosis and caries incidence in a community residing in a high fluoride area. *Journal of Oral Pathology* 8: 224-36. (See abstract)
Roholm K. (1937). Fluoride intoxication: a clinical-hygienic study with a review of the literature and some experimental investigations. H.K. Lewis Ltd, London. (See excerpts)
Smith MC, Smith HV. (1940). Observations on the durability of mottled teeth. *American Journal of Public Health* 30: 1050-1052.

` The most recent publication on dental fluorosis 2024, is an "Expert Panel Meeting on Health Effects of Fluoride in Drinking Water" The Panel was chosen by Canadian Health, the strongest promoter of fluoridation in Canada. A single study from1942 by Dean was the key endpoint used by the committee to determine harm, a study more than 80 years old with significant limitations. Seriously, I've been listening to 8 days of court presentations by experts. EPA experts reject the dozens of studies reporting harm as inadequate, yet accept a single study from 8 decades ago as point of departure. The fluoridation lobby make no sense.

The panel Summarized:

"Selection of a point of departure is a critical step in the development of a health-based value. The point of departure for neurocognitive effects (i.e., IQ reduction) is not yet well defined because of uncertainties, including the shape of the exposure-response curve at low concentrations of fluoride in drinking water.

Teotia SPS, Teotia M. (1994). Dental caries: a disorder of high fluoride and low dietary calcium interactions (30 years of personal research). *Fluoride* 27: 59-66. (See abstract | See study)

Wondwossen F, et al. (2004). The relationship between dental caries and dental fluorosis in areas with moderate- and high-fluoride drinking water in Ethiopia. *Community Dentistry and Oral Epidemiology* 32: 337-44. (See abstract)

Ziegelbecker R, Ziegelbecker RC. (1993). WHO data on dental caries and natural fluoride levels. *Fluoride* 26: 263-266. (See excerpt)

See also:

Steelink C. (1992). Fluoridation Controversy. (Letter). *Chemical Engineering News* July 27: 2-3.

Therefore, moderate dental fluorosis was selected as the key endpoint of concern with a point of departure of 1.56 mg F/L in drinking water.

"The tolerable daily intake is normally calculated by dividing daily intake on a µg/kg/day basis by an uncertainty factor. Since the point of departure in this case is already a measurement in drinking water, this step (and calculation of the health-based value) can be simplified by applying an uncertainty factor directly to the point of departure to account for the database deficiencies about the potential occurrence of neurotoxicity from exposure to fluoride at low doses.

"Therefore, the drinking water concentration (DWC) is calculated by dividing the point of departure (POD) by the uncertainty factor (UF).

DWC = POD/ UF

A health-based value (HBV) for fluoride in drinking water would be calculated by multiplying this DWC by an allocation factor (AF) to account for exposure to fluoride from other sources.
HBV = DWC × AF

Focusing on just **dental fluorosis** at this point and their use of 1.56 mgF/L: A safe drinking water concentration would be 1.56 mgF/L divided by the uncertainty factor (to be determined by Health Canada) or intraspecies variability. Most would agree, not all humans are the same age, health, drink the same amount of water, have the same health, in other words not all humans wear the same size shoe. The NRC 2006 and EPA reported the average person drinks about 1 liter of water a day and some drink

over 10 liters of water a day. To assure safety, an intraspecies consumption of just water, ignoring all other differences in humans, an uncertainty factor of 10 would need to be used. The committee used the formula:

DWC = POD/ UF (DWC = Desired Water Concentration) (POD = Point of Departure) (UF = Uncertainty factor)

DWC= (POD) 1.56 mgF/L X (UF) 10 = 0.156 mgF/L in public water. The Board recommends 0.7 mgF/L.

0.7 is greater than 0.156.

To assure safety, the Board would need to select 0.156 mgF/L (same as ppm) fluoride concentration in water instead of current 0.7 ppm. 0.156 ppm would be an estimated safe water fluoride concentration to prevent moderate dental fluorosis.

However, the panel also noted an “allocation factor” (how much total fluoride comes from fluoridation) of 0.5, which is a good rule of thumb, but varies more typically from 1/3 to 2/3rds and can be over 90% for some.

Assuming allocation is 0.5, total exposure reduction would go down by half if fluoride in water were 0 mg/L. Even eliminating fluoridation, some will ingest too much from other sources.

There is so much more to understand when considering the cost of fluoridation. We must add developmental neurotoxicity, more below.

If we assume just 3 lower IQ points lost and and assume about $500/person/IQ lower income, my estimates based on research and adjusted for 2021dollars IQ loss would be about$1,500/year/ person. Including dental fluorosis harm wiped out benefit. Including IQ loss gets us even further in a loss. But I have not included the other risks below.

Fluoridation is very costly.

A cost estimate resulting in savings requires the dental lobby to only use some costs to fluoridate, ignore harm, and exaggerate cost savings.

#2. The Board claims: *Water fluoridation reduces tooth decay by about 25 percent over a person's lifetime."*

No current research is provided because none is available. A public health intervention should be measured in the public at large and the Board fails to provide the evidence for their claim. The Board's claim of benefit is consistent with the CDC Oral Health Division which is virtually in lock step with the American Dental Association and CDC is part of the fluoridation lobby. The fluoridation lobby is profiting from the disposal of fluoride in public water rather than having to pay thousands of dollars a ton to dispose of the toxic waste.

When fluoridation started a 65% reduction in dental caries was claimed, based on lower quality studies, and then shown not to be true. Later, a 25% reduction was claimed and now shown not to be true. Higher quality research, more careful review of the research does not support significant benefit.

If such a robust reduction in caries were in fact true (25%), we would see significant decrease in treatment and dental costs in fluoridated communities along with lower insurance payment for

dental treatment. But costs are not lower in fluoridated communities and dentist/patient ratio is not lower in fluoridated communities.

FDA CDER REQUIRES RANDOMIZED CONTROLLED TRIALS (RCT) FOR EFFICACY.

The Board appears to disagree with the FDA CDER which has not approved ingestion of fluoride reporting: *. . . there is no substantial evidence of drug effectiveness. . . ."* Drug Therapy 1975.

And in 2010 the FDA indicated application for NDA would effectively ban fluoridation. The Board cannot assure safety if the only drug authorized regulatory agency would not approve fluoridation.

When the FDA CDER evaluates the quality of research on drug "**efficacy**," the FDA CDER requires RCTs. Note[25]

[25] "Randomized controlled trials (RCT) are prospective studies that measure the effectiveness of a new intervention or treatment. Although no study is likely on its own to prove causality, randomization reduces bias and provides a rigorous tool to examine cause-effect relationships between an intervention and outcome. This

In contrast, evaluation of **"safety"** is more complex because we cannot intentionally give a person enough of the substance to find out when they get sick or die. The FDA CDER requires monitoring for side effects, risks in the RCT studies. Absent RCT studies, as is the case with fluoride exposure, safety must be determined with lower quality ecological studies, comparing peoples or populations are the option. But those studies do not look for safety and the Board cannot assure safety without safety studies.

The fluoridation lobby will claim that ecological studies of harm are not reliable. If we disallow ecological studies, we would also throw out the studies we have on benefit.

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is because the act of randomization balances participant characteristics (both observed and unobserved) between the groups allowing attribution of any differences in outcome to the study intervention. This is not possible with any other study design. In designing an RCT, researchers must carefully select the population, the interventions to be compared and the outcomes of interest. Once these are defined, the number of participants needed to reliably determine if such a relationship exists is calculated (power calculation). Participants are then recruited and randomly assigned to either the intervention or the comparator group.[1] It is important to ensure that at the time of recruitment there is no knowledge of which group the participant will be allocated to; this is known as concealment. This is often ensured by using automated randomization systems (e.g. computer generated). RCTs are often blinded so that participants and doctors, nurses or researchers do not know what treatment each participant is receiving, further minimizing bias."

When comparing populations, the Board must keep in mind, to estimate total fluoride exposure we need to take the 0.7 mg/L from water and at least double that to include background exposure. In other words, the Board needs to actually look at 1.5 mg/L in studies on safety to even consider the mean exposure.

Comparing high and low fluoride populations does not compare the absence of fluoride with 1.5 mg/L but a lower concentration with a higher concentration.

And 1.5 mg/L does not account for those drinking more than the mean amount of water, frequently pregnant moms.

If ingesting fluoride had benefit, the Board and/or industry (dentists) could simply get FDA CDER approval and make a profit from selling the fluoride license/patent.

In fact, the Board contacted the FDA and was told requiring FDA approval would effectively ban fluoridation. And I tried to get FDA approval. Not because I thought fluoride safe or effective, but because an application might force the FDA to more closely evaluate fluoride's lack of benefit and risks, and take regulatory

action. The FDA denied my application because I'm not a water district.

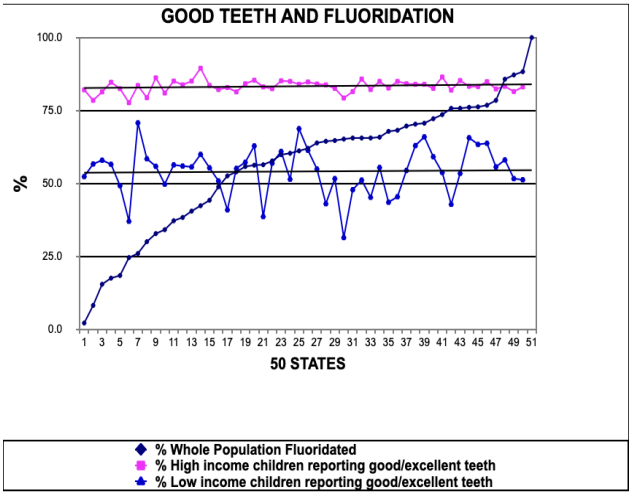
The FDA CDER have the highest standards, are highly qualified pharmacologists, toxicologists, experts and have the most respect for drug approval of all federal and state agencies.

The Board should consider that their intent to protect vulnerable populations from some dental caries is not supported by quality science and plenty of science reports additional harm to those subpopulations (low socioeconomics, increased lead exposure in fluoridated communities, etc.)

The following correlation graph was generated when I ranked the USA states on the percentage of their whole population fluoridated and reported good to excellent teeth.[26] A 25%

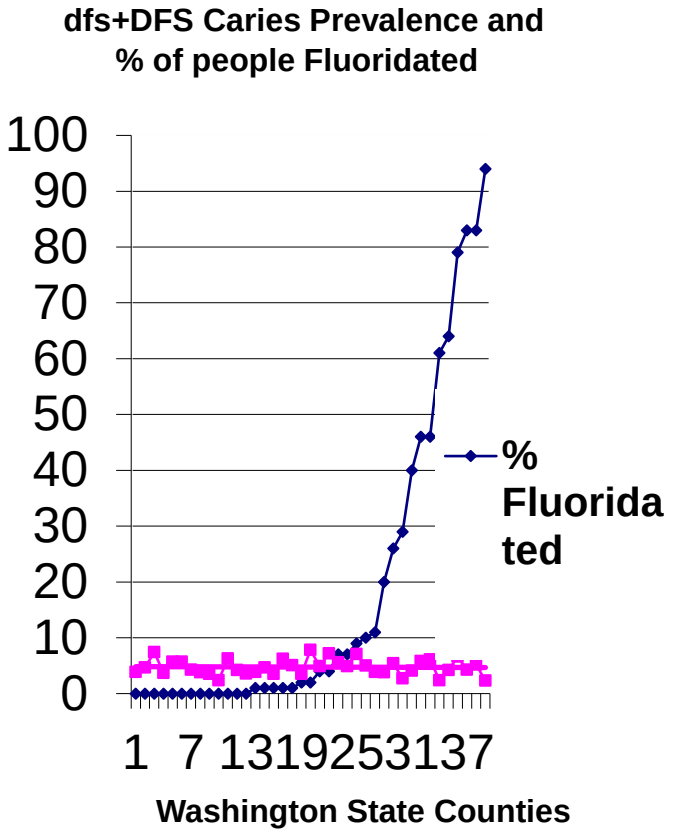
[26] http://mchb.hrsa.gov/oralhealth/portrait/1cct.htm National Survey of Children's Health.
U.S. Department of Health and Human Services, Health Resources and Services Administration, Maternal and Child Health Bureau.
The National Survey of Children's Health 2003. Rockville, Maryland: U.S. Department of Health and Human Services, 2005
http://www.cdc.gov/oralhealth/waterfluoridation/fact_sheets/states_stats2002.htm
http://pubs.usgs.gov/circ/2004/circ1268/htdocs/table05.html

reduction, or any reduction, is not evident when similar SES groups are ranked.

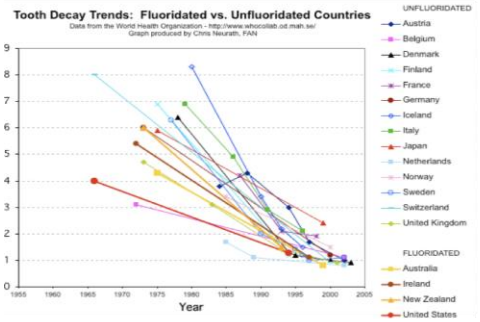


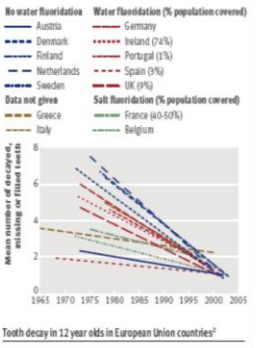
Socioeconomics is highly significant for caries prevalence, but fluoridation has no “common cause” or correlation. For 20 years as a dentist, I promoted fluoridation and thought I could see proof of benefit from fluoridation in my patients. However, after reading the research it was clear I had been comparing socioeconomics rather than fluoridation.

I also ranked Washington State Counties on the percentage of their population fluoridated and dental caries. No reduction in dental caries is supported by the population at large in Washington State, caries is about the same regardless of fluoridation.



Two published studies[27] ranking WHO data on caries over about 3 decades does not report lower caries in fluoridated countries or those who use fluoride salt, graphs below.

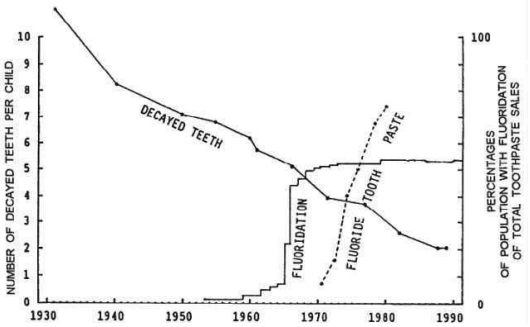




All developed countries have reduced dental caries to low levels, regardless of fluoridation or fluoride salts. Giving fluoride credit for a reduction of caries in non-fluoridated countries prior to fluoridation is not reasonable.

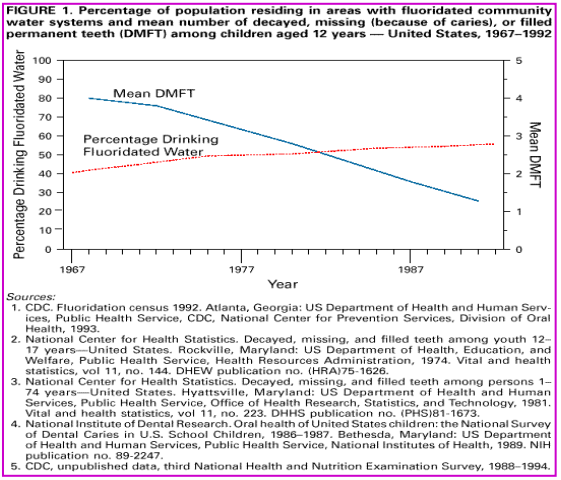
[27] Neurath http://www.fluoridealert.org/health/teeth/caries/who-dmft.html and Chen et al, BMJ 5 October 2007

To the right is a graph of caries over a longer period of time.[28] What caused the decline in dental caries,



more than half before the beginning of fluoridation? No one knows. No research on fluoridation has taken into account the huge unknown(s). We cannot give fluoridation credit for caries reduction prior to fluoridation. And any research must be suspect if it does not correct for those unknowns after fluoridation started, and no research corrects for those unknowns because they are unknown.

However, on the CDC website, a 1999 graph (right) is



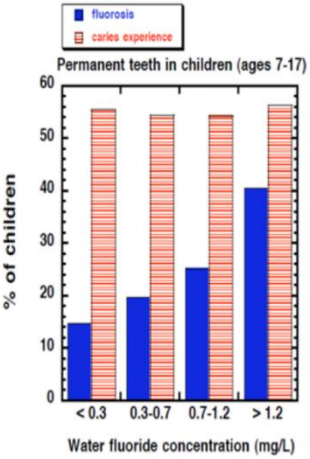
[28] In 1998, Colquhoun graphed the trend of dental caries in the USA, see graph below (ISFR 1998)

presented which at first glance looks impressive. Indeed, caries declined and fluoridation rates increased, but the graph is misleading by only looking at a few years. And it is not plausible that an increase of perhaps 10% of the public “randomly” fluoridated resulted in a decline from 4 DMFT (adult decayed, missing, filled teeth) to just over 1 for everyone. Simply not plausible. Even if the fluoride were dispensed to only the high-risk children individually, that would not have produced about a 70% decrease in DMFT. Fluoridation is not targeted, and started in some cities, not just for high-risk individuals.

The Journal of the American Dental Association published the following data which was graphed by Thiessen.

The red lines represent caries experience. Any difference in caries experience (red lines), at any concentration, is hard to detect and certainly not 25% as alleged by the Board. All red lines are at a similar height, although perhaps 2% lower at about 0.7 mg/L.

Iida, H., and Kumar, J.V. 2009. The association between enamel fluorosis and dental caries in U.S. schoolchildren. JADA 140:855-862.



The blue lines represent reported dental fluorosis. As expected, an increase in fluoride concentration in water increases the damage from excess fluoride, dental fluorosis, more than double. Dental fluorosis occurs while the tooth is developing under the skin, mostly before age 6. The developing brain and other organs are developing during the same time, and would not be spared from the excess fluoride. The teeth are not the only

tissues harmed, but they are the easiest to diagnose. (The NTP 2023 report and the Fluoride On Trial: The Censored Science on Fluoride and Your Health | Childrens Health Defense must be reviewed.)

Mechanism of Fluoride's Action (continued from above): Topical fluoride at high concentrations (over 1,000 ppm) has been shown to be effective (toothpaste) and is FDA CDER approved and listed in the Orange Book of approved drugs, but not fluoride ingestion.

On the other hand, to be effective, ingested fluoride must go from the pulp chamber through the calcium rich dentin and enamel to the surface of the tooth where the dental caries are forming.

Topical fluoride (like toothpaste) can get to the dental caries, ingested fluoride cannot. The tooth is highly resistant to the migration of fluoride. In the graph below, there is an increase in fluoride concentration near the pulp and at the surface of the tooth from topical fluoride, but in the middle the concentration is

low. Saliva has a low concentration of fluoride and cannot have much benefit.

Think of fluoride like suntan lotion. Put it on the outside and “do not swallow.”

The graph (right) shows the fluoride concentrations in the tooth.

A few of the limitations on fluoridation research often Include:

- A. Not one Study corrects for Unknown Confounding Factors. Think of the graph above reporting significant decline prior to fluoridation. That huge massive crushing dental caries prior to fluoridation is unknown and not controlled for in any study because no one knows what it is. Did it stop when fluoridation started? No, other countries prove it did not. Therefore, the most logical cause of caries reduction is the unknown(s), not fluoridation.
- B. Not one Prospective Randomized Controlled Trial (one on supplements reported no statistical benefit) And without RCT’s, no meta-analysis of RCT’s can be done.

- C. Socioeconomic status usually not controlled
- D. Inadequate size
- E. Difficulty in diagnosing decay
- F. Delay in tooth eruption not controlled
- G. Diet: Vitamin D, calcium, strontium, sugar, fresh and frozen year-round vegetables and fruit consumption not controlled.
- H. Total exposure of Fluoride not determined
- I. Oral hygiene not determined
- J. Not evaluating Life-time benefit
- K. Estimating or assuming subject actually drinks the water.
- L. Dental treatment expenses not considered
- M. Mother's fluoride exposure, Breast feeding and infant formula excluded
- N. Fraud, gross errors, and bias not corrected.
- O. Genetics not considered
- P. Studies reporting benefit were done at 1.0 ppm, we are now fluoridating at 0.7 ppm. Does the lower dose provide benefit? We don't know.

CDC: **“Ingestion of fluoride is not likely to reduce tooth decay.”**[29]

“The results show that the reviewed original studies on economic evaluation of caries prevention do not provide support for the economic value of caries prevention.”[30]

Former Director of the National Toxicology Program (NTP) and Office of Health Assessment and Translation (OHAT) at (NIEHS) (NIH) Linda Birnbaum, Ph.D., D.A.B.T., A.T.S. is a microbiologist and board-certified toxicologist. (See endnote 1.) Her sworn testimony is critical for evaluation by the Board. VIDEO: Former NTP Director’s Statement on Fluoride Neurotoxicity — Fluoride Action Network (fluoridealert.org)

Even if fluoridation at 1.0 ppm were effective, that does not prove 0.7 ppm fluoride in water is equally effective. . . if at all.

[29] Achievements in Public Health, 1900-1999: Fluoridation of Drinking Water to Prevent Dental Caries. MMWR, 48(41); 933-940, October 22, 199

[30] Källestål C et al. Acta Odontol Scand. 2003 Dec;61(6):341-6. Economic evaluation of dental caries prevention: a systematic review.

In 1975 my fluoride professor suggested the possible delay in tooth eruption with fluoride ingestion was adequate proof of fluoridation's benefit. Or could be simply a delay in diagnosis.

If the tooth is protected under the skin from food and harm for just a few months, researchers evaluating caries by a child's age, will be comparing different amount of time the teeth have been exposed to the environment. Of course, the concern that a delay in tooth eruption could cause a delay or premature development of other systems and organs must be considered. But we dentists only look at structures of the mouth.

Not all studies agree there is a delay in tooth eruption with fluoridation; however, the evidence should be considered, see

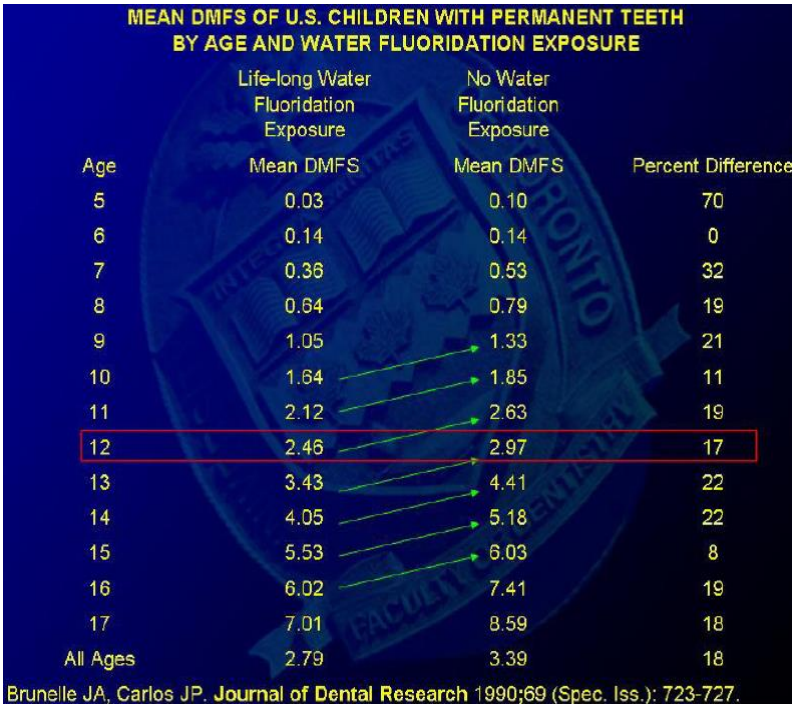
data below, the first from 1957, the second from 1990.

CAClinch © 2010

Newark, Delaware

	Decayed-Missing-Filled Teeth		Percent Caries-Free Children	
Age	After Fluoridation	Before Fluoridation	After Fluoridation	Before Fluoridation
6	0.2	1.1	88.8	54.8
7	1.1	2.3	44.9	22.7
8	1.7	2.9	31.5	8.6
9	2.8	3.7	11.3	4.8
10	3.4	4.9	6.4	7.5

Reference: Journal American Dental Assoc. Vol. 54, June 1957
Note: 1-year DELAY in DMF per child. At age 10 FEWER caries-free children AFTER fluoridation than BEFORE.



REPUTABLE AGENCIES OPPOSED TO FLUORIDATION:

The fluoridation lobby has claimed there are no “reputable” health agencies which oppose fluoridation, yet their definition of “reputable” limits their search to those agencies which promote fluoridation.

Austria REJECTED: “toxic fluorides” NOT added

Belgium REJECTED: encourages self-determination – those who want fluoride should get it themselves.

Finland STOPPED: “...do not favor or recommend fluoridation of drinking water. There are better ways of providing the fluoride our teeth need.” A recent study found ...”no indication of an increasing trend of caries....“

Germany STOPPED: A recent study found no evidence of an increasing trend of caries

Denmark REJECTED: “...toxic fluorides have never been added to the public water supplies in Denmark.“

Norway REJECTED: “...drinking water should not be fluoridated“

Sweden BANNED: “not allowed”. No safety data available!

Netherlands REJECTED: Inevitably, whenever there is a court decision against fluoridation, the dental lobby pushes to have the judgment overturned on a technicality or they try to get the laws changed to legalize it. Their tactics didn't work in the vast majority of Europe.

Hungary STOPPED: for technical reasons in the '60s. However, despite technological advances, Hungary remains unfluoridated.

Japan REJECTED: "...may cause health problems...."

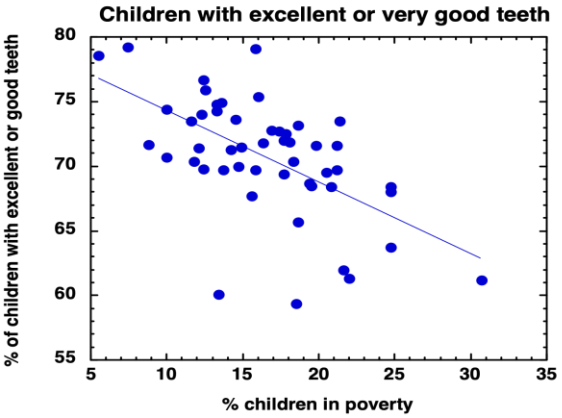
Israel SUSPENDED mandatory fluoridation until the issue is reexamined from all aspects.: June 21, 2006 "The labor, welfare and health Knesset committee"

China BANNED: "not allowed" Some of the earliest studies raising concern on developmental toxicity were done in China. China should be given credit for starting to wake the USA up to fluoride's developmental neurotoxic risks.

When the 50 states are ranked based on their whole population fluoridated, we do see a slight decline in the states with

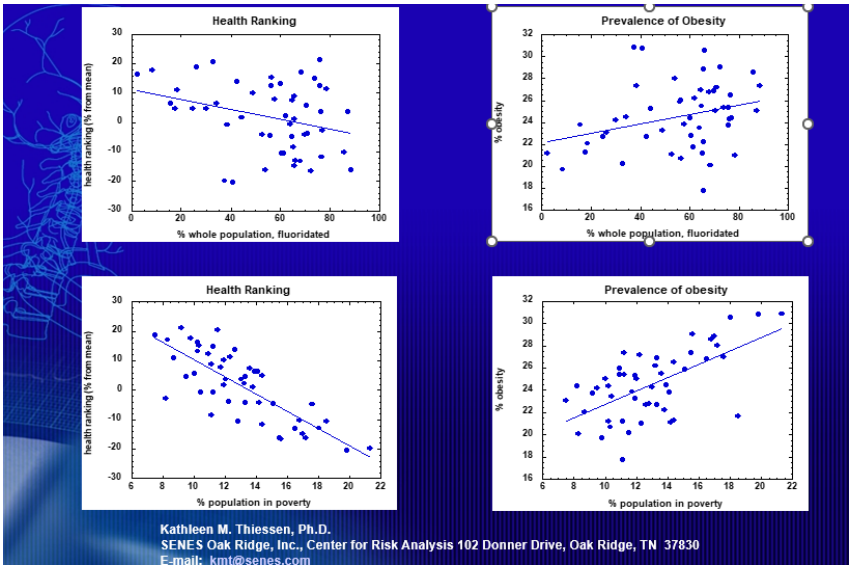
more of their population fluoridated.[31] Based on this data we see about a 7% caries reduction for third graders.

Dr. Thiessen ranked the states on socioeconomics. The wealthier appear to have better dental health.



Additional graphs by Thiessen below. Health ranking appears to decline with fluoridation and significant decline for those states with a higher percentage of poor. Obesity increases and obesity is affected by the thyroid and fluoride harms the thyroid, more seriously for the poor. Fluoridation harms the poor the most.

[31] Kathleen Thiessen PhD kmt@senes.com *SENES Oak Ridge Inc.* Center for Risk Analysis



ADA awarded Kentucky with "50 Year Award" for (100%) fluoridation in 2003 at the same time 42% were edentulous, #1 in USA (2002 Mortality Weekly Report) Connecticut, Detroit, and Boston all reported a crisis of dental caries and all have had fluoridation for decades.[32]

32

http://www.fortwayne.com/mld/newssentinel/7521679.htm?template=contentModules/printstory.jsp
http://www.enquirer.com/editions/2002/10/06/loc_special_report.html
http://www.fluoridealert.org/f-boston.htm

When questioned about the scientific evidence for benefits and safety of fluoridation, the Washington Department of Health responded: “DOH will rely on known national entities like the CDC and EPA to assess the science. . . .” (Letter from DOH)

See Fluoride On Trial: The Censored Science on Fluoride and Your Health | Childrens Health Defense for the CDC’s response. See attached letter from EPA for EPA’s response.

Even when the CDC reported the CDC does not determine the safety of fluoridation and the CDC along with the ADA warned infants should NOT have fluoridated water for formula and drinking, the Washington Department of Health responded in disagreement, reporting: *“Parents and health providers should weigh the balance.”* Seriously? Does the Department of health expect parents to review the literature when the Department doesn’t have the experts or money to review the evidence?

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http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=13678102&query_hl=1
http://www.nhregister.com/site/news.cfm?newsid=14472801&BRD=1281&PAG=461&dept_id=517515&rfi=8&xb=kasan

And the Board expects parents and health providers to do what the Board and Department fail to do. I doubt the legislature expected the public to weigh the complex scientific data.

Our point: The Board should not assume a 25% reduction in dental caries exists.

In 2003, the EPA asked the NRC to review EPA's Maximum Contaminant Level Goal (MCLG) for fluoride. The NRC unanimous agreement was that EPA's MCL for fluoride was too high. For 18 years the EPA has not changed the MCL or MCLG for fluoride. The NRC 2006 report based their decisions on concerns for:

- **Tooth Damage**
- **Rheumatoid and Osteoarthritic-like Pain**
- **Bone Cancer**
- **Bone Fractures**
- **Thyroid Reduction**
- **Diabetes**
- **Obesity**
- **Kidney damage**

- **Reproductive problems**
- **Lower IQ and increased Mental Retardation**
- **Allergies (overactive immune system)**
- **Gastrointestinal disorders**

#3. The Washington Board of Health also claims:

Community water fluoridation is safe. After 65 years in service and hundreds of studies it continues show its safety."

"*Over the past 75 years, health authorities have declared that community water fluoridation-a practice that reaches over 400 million worldwide-is safe. Yet, studies conducted in North America examining the safety of fluoride exposure in pregnancy were nonexistent. . . .*

The tendency to ignore new evidence that does not conform to widespread beliefs impedes the response to early warnings about fluoride as a potential developmental neurotoxin. Evolving evidence should inspire scientists and health authorities to re-evaluate claims about the safety of fluoride, especially for the fetus and infant for whom there is no benefit."[33]

[33] Till C, Green R. Controversy: The evolving science of fluoride: when new evidence doesn't conform with existing beliefs. Pediatr Res. 2021

Scientists have avoided the controversies of fluoride exposure. Publishing controversial research is a career killer. As one of my mentors would say, tongue in cheek: *"Never let a rational thought interfere with a lucrative procedure."*

If fluoridation were the only source of fluoride, fluoridation would not be safe.

If teeth were the only tissues of the body, fluoridation would not be safe. Fluoride ingestion may or may not have benefit, but fluoride without dispute harms teeth both aesthetically and functionally. The dental lobby only considers benefit to teeth and discounts harm as only aesthetic.

Endorsements of benefit, are not science, empirical evidence, facts or evidence of safety.

The Board is assuming endorsements by unauthorized agencies, industry, claiming or "declaring" benefit and safety are factual evidence. "The absence of safety evidence is not proof of safety."

Nov;90(5):1093-1095. doi: 10.1038/s41390-020-0973-8. Epub 2020 May 22. PMID: 32443137; PMCID: PMC9922476.

THE FETUS: (See attachment H.)

I have found no safety studies determining the safety of fluoride exposure for the developing fetus. The Board cannot assure safety for the fetus without safety studies.

Here are the two most vulnerable cells starting the dividing and growing process of life, the mother is probably not even aware. Fluoride passes from the mother through the placenta to those cells.

As the fetus grows, there is no developed blood brain barrier to protect the fetus's developing brain from toxins. In time, the fetus drinks the amniotic fluid, the developing kidneys excrete some of the fluoride and we assume half stays in the fetus, mostly the developing bones. The fetus drinks the fluoride fluid laced urine, concentrating the fluoride mostly in the bones, but also potentially affecting every cell, system, organ of their body, anatomy and physiology.

Excess fluoride is "recycled.". Yet the Board, without research, blindly assumes the fetus is not affected and safe.

Challenged on safety, dentists often claim everything outside of the mouth is not their purview.

DEVELOPMENTAL NEUROTOXICITY (Fluoride's toxic effect to the developing brain):

"Fluoride is most definitely a developmental neurotoxicant."[34]

A large volume(s) could be written on just fluoride's effect on the brain, especially for the fetus, infant and child, who are receiving the highest dose of fluoride.

Our point: The consistency and number of studies reporting lower IQ for children in a linear relationship as dose of fluoride increases is reasonable. The more fluoride, the more brain damage.

The brain is the most precious gift of life. The brain goes through stages of development and if harmed at a stage, may never recover.

Knowingly harming the brain is inexcusable and no Board recommendation and policy should steel the essence of the highest quality of life a person can have.

[34] Dr. Grandjean February 1, 2024 in sworn testimony in the TSCA EPA trial.

Over a decade ago, the Board of Health refused our petitions to protect the health of the developing brain, fetus, infant, and children. Instead, the Department and Board trusted the EPA and CDC who have no jurisdiction over the efficacy, dosage, safety or label of fluoride. (See attached #F, letter from the EPA)

I, and others, turned to the U.S. National Toxicology Program (NTP), the highest scientific authority in the USA to review the toxicity, safety, of fluoride exposure. Due to cost, time, and the need to evaluate thousands of other toxins, the NTP agreed to review just one aspect of fluoride's toxicity, developmental neurotoxicity i.e. as measured with lower IQ. This link is to the 700+ page draft which includes reviewers' comments and NTPs responses. The NTP Board of Counselors voted unanimous approval. NTP's review of just one harm does not imply brain damage is the only harm from fluoride exposure.

Again, links to the Draft NTP Monograph on the State of the Science Concerning Fluoride Exposure and Neurodevelopmental and Cognitive Health Effects: A Systematic Review. and Table of Contents.

The dental lobby will dismiss applicability of the NTP Monograph to fluoridation, in part, because the NTP was politically prevented from evaluating fluoridation. However, the science clearly shows fluoride exposure to be a developmental neurotoxicant at dosages common to many. In court, we learned some of the reviewers have strong ties to vested fluoride interests.

In October and December of 2022, evidence in a TSCA (Toxic Substance Control Act) legal action against the EPA for failure to protect the public, reported political pressures from HHS's Rachael Levine, prevented release of the NTP monograph. It took a Court order for release of the science.

I must digress. Withholding of medical research is research misconduct. The World Health Organization reported it is an ethical imperative to support full disclosure of all clinical trial research. Lack of full disclosure puts the public at risk of ineffective and harmful medical products*. "In short, disclosing clinical trial results leads to better-informed science and saves lives."*

WHO states further, *"Withholding clinical trial results defeats the purpose of medical research."*[35]

A great deal of tax payer money, thousands of hours of researchers' time and cohort time went into research provided to the NTP and their 6 years of research and review. Any attempt to cover up, hide, withhold the research is unethical, an insult to the researchers and subjects. Levine and collaborators should be disciplined for withholding the NTP monograph.

The Department of Justice had attempted to block testimony from the NTP, but the court ruled he could testify. Withholding evidence tarnishes the credibility of the person and agency.

Back to the TSCA trial. The Judge said the report would be considered final and would be given "a fair amount of weight." Like the Court, the Board of Health should also give the report a fair amount of weight.

[35] Vasee Moorthy, a technical officer with the WHO, in email to CMAJ. And Dr. Ben Goldacre author of the books Bad Science and Bad Pharma

May 4, 2023, the Board of Scientific Counselors approved the NTP report. HHS has still not officially published the monograph, and the Director reported the monograph may never be published. Political pressure is blocking good science.

The NTP monograph included 72 human fluoride IQ studies of which 64 found a relationship between fluoride and lower IQ. 19 of the studies were considered high quality and 18 reported IQ loss, the vast majority.

Of the vast majority of human studies accepted by the NTP evaluating developmental neurotoxicity, 95% report harm. The consistency is remarkable and is a growing data base.

Fluoride has met the standard of EPA hazard causation.

Due to political pressure, the report was divided into two sections. The first is called the State of the Science and the second is the Meta-analysis. The State of the Science appears to be more influenced by the dental lobby. The meta-analysis appears to have more empirical, factual, evidence.

A few NTP quotes:

"Our meta-analysis confirms results of previous meta-analyses and extends them by including newer, more precise studies with individual-level exposure measures. The data support

a consistent inverse association between fluoride exposure and children's IQ."

When an unnamed government fluoridation proponent claimed:

"The data do not support the assertion of an effect below 1.5 mg/L…all conclusory statements in this document should be explicit that any findings from the included studies only apply to water fluoride concentrations above 1.5 mg/L."

The NTP responded:

*"**We do not agree with this comment**…our assessment considers fluoride exposures from all sources, not just water…because fluoride is also found in certain foods, dental products, some pharmaceuticals, and other sources… Even in the optimally fluoridated cities…individual exposure levels…suggest widely varying total exposures from water combined with fluoride from other sources."*

"Discussion

The results of this meta-analysis support a statistically significant association between higher fluoride exposure and lower children's IQ. The direction of the association was robust to stratification by risk of bias, sex, age group, timing of exposure, study location, outcome assessment type, and exposure assessment type. There is also evidence of a dose-response relationship. Although the estimated decreases in IQ may seem small, research on other neurotoxicants has shown that subtle shifts in IQ at the population level can have a profound impact on the number of people who fall within the high and low ranges of the population's IQ distribution [50-54] For example, a 5-point decrease in a population's IQ would nearly double the number of people classified as intellectually disabled [55]."

The NTP's meta-analysis raises confidence that fluoride is indeed harming the developing brain. And as with the early reports of lead's harm, further more precise, focused study on lead confirmed rather than disputed the earlier studies.

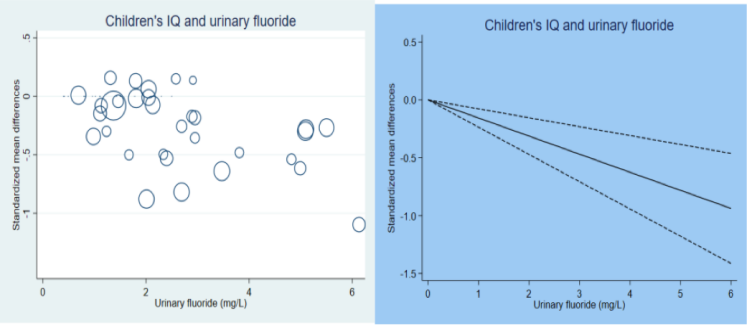
Note: One standard deviation is 15 IQ points.

The NTP charts below, for example, show a mother with 1 mg/L of fluoride in her urine would have a child with about 0.1 standard deviation loss of IQ. At 2mg/L about 0.3 SD loss and 3 mg/L fluoride urine concentration, common for women in the third trimester of pregnancy, about half a SD IQ loss.

Half a standard IQ loss would be about 7-8 IQ points lost. Under oath the EPA's expert conceded that fluoride is a neurotoxicant.

The NTP graphs below should be reviewed. For reasonable estimates, urine fluoride concentration approximates total fluoride exposure because about half the fluoride stays in the body, mostly, but not all, in the bones.

Urinary Fluoride Exposure



eFigure 18. Pooled Dose-Response Association Between Fluoride in Urine and Standardized Mean Differences in Children's IQ

Left panel: Circles indicate standardized weighted mean differences in individual studies; size of bubbles is proportional to precision (inverse of variance) of the standardized mean differences. Right panel: Urinary fluoride levels were modeled with a linear random-effects model (solid line). Dashed lines represent the 95 % confidence intervals for the linear model. Please see **eTable 2** for characteristics of the studies included in the *dose-response meta-analysis* (studies with urinary fluoride exposure and at least two exposure levels).

Urine fluoride concentration of 3 mg/L representing about half a standard deviation would expect to have a child with about 7 IQ less. A mom drinking 3 liters per day at 0.7 mg/L would ingest about 2.1 mg of fluoride just from water, more than the NTP hazard level. Additional fluoride from other sources could easily push the mom over 3 mg fluoride per day.

Figure 2 of the NTP meta-analysis, page 19 presented below:

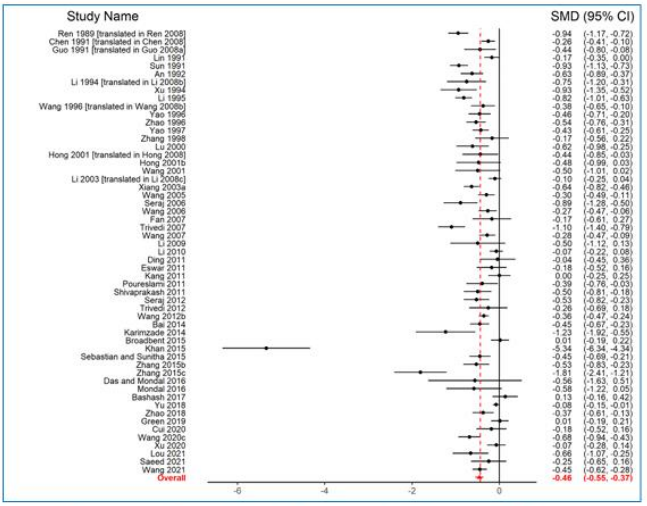


Figure 2. Association Between Fluoride Exposure and IQ Scores in Children

Forest plot for random-effects meta-analysis of the association between fluoride exposure and child's IQ scores. Effect size is expressed as the standardized weighted mean difference for heteroscedastic population variances (SMD). The random-effects pooled SMD is shown as a solid triangle. Horizontal lines represent 95% CIs for the study specific SMDs.

Research seems to mostly be around -0.46 mean overall standard deviation which represents about 7 IQ point loss. (1 SMD is 15 IQ points)

Performance IQ is reported at 8.8 IQ loss, full scale 4.4 IQ loss[36] with an increase of 0.5 mg/L fluoride in water.

36 Till C, Green R, Flora D, Hornung R, Martinez-Mier EA, Blazer M, Farmus L, Ayotte P, Muckle G, Lanphear B. Fluoride exposure from infant formula and child IQ in a Canadian birth cohort. Environ Int. 2020 Jan;134:105315. doi: 10.1016/j.envint.2019.105315. Epub 2019 Nov 16. PMID: 31743803; PMCID: PMC6913880. [PubMed]

Two studies in Australia, evaluating the same area did not find IQ loss. One did not control for fluoride supplements in the non-fluoridated cohorts. Low exposure levels are more difficult to see.

One study[37] not reporting IQ loss is promoted by the fluoridation lobby and is impossible, an outlier. The samples need to be sent to a different laboratory for testing.

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[37] A study by Dr. Jesus Ibarluzea, at low fluoride concentrations not only does fluoride NOT lower IQ, but it can transform an average-IQ boy living in a non-fluoridated area, that is correct, a NON-fluoridate area with some fluoride into a genius with low levels of fluoride exposure, for example raising IQs for boys by 28 points. . . but not girls. When asked if he would be looking into why such a large increase, he said he had no interest in finding the problem. This study is an outlier from other studies.

In fact, the 15 IQ and 28 IQ point increase for boys as reported, is based on using 1 mg/g. In fluoride neurotoxicity epi studies, a common exposure increase is 1 mg/L of urine. The difference is about 30%. This correction increases the implausibility of a 15 or 28 IQ point increase for boys to an impossible 20 to 37 IQ increase for boys.

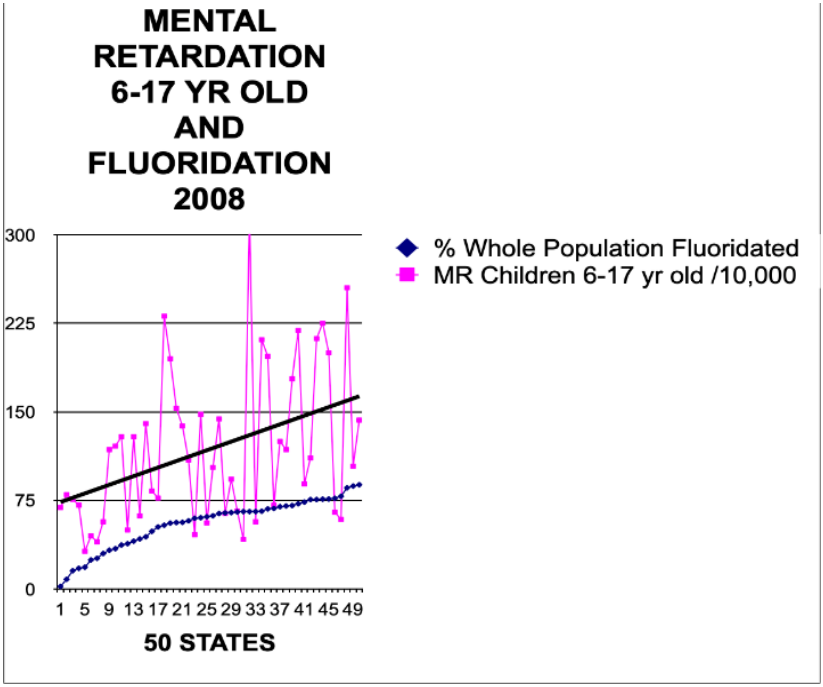
During his deposition as a witness in the TSCA trial, Dr. Ibarluzea was asked whether he ever asked anyone to delete information about his fluoride study, to which he responded, “Never, never, never, ever.” According to a FOIA document, however, Dr. Ibarluzea sent the CDC’s Division of Oral Health an email about his study, which ended with the words “Please delete this message.” The contents of the message remain unknown because CDC redacted the entire email with the exception of the “Please delete this message” instruction.

Dr. Ibarluzea then withdrew from any further participation in TSCA legal case. Another study promoted by the dental lobby, a meta-analysis, relied heavily on Dr. Ibarluzea’s study to report no harm from fluoride exposure.

Dr. Grandjean who has published over 500 studies on toxic substances, is a risk assessment expert, testified in court under oath, said he had never seen or could imagine such an outlier as accurate. He said the authors should immediately send samples back to the lab, or a different lab, for verification.

Future studies evaluating will likely report with further clarity more serious harm for individuals at various socioeconomic levels, various races, ages, and gender (males), more sensitive to fluoride various types of IQ loss and greater harm.

After the 2006 NRC report suggesting possible brain damage from fluoride, I wanted to personally see if I could confirm the NRC 2006 report. I ranked the 50 states and plotted their



reported mental retardation (intellectual disability) and percent of the whole population fluoridated, a correlation study. The trend, more than doubling of "mentally retarded," about 7-8 IQ loss, (half

a standard deviation) raised concerns and is supported with more recent published studies including the NTP meta-analysis.

Nearly doubling the number of “mentally retarded ” would represent close to 7 IQ point loss. The EPA uses just one IQ loss as their threshold of harm. Equally of concern is the serious reduction in gifted and of course the rest of us in the middle are harmed.

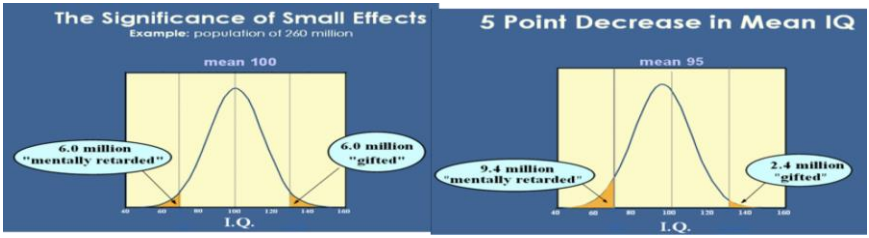
When other confounders are considered for ranking the 50 states, socioeconomics is slightly lower in the more fluoridated states. Socioeconomics and IQ are related, to a degree.

Remember the “Bell Curve.” The graphs below illustrate 5 IQ loss with over 50% increase in the number of low IQ, and a third the gifted is a concern.

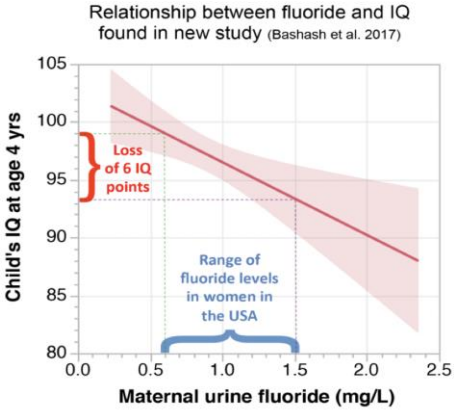
To assure the public fluoridion is safe, the Board must provide quality research to support safety.

Think of our special education classes. Think of employers, parents and those children who know they are not as “smart” as others. Low IQ tend to be incarcerated more, higher

divorce rates, homeless, etc. And a loss of more than half the gifted is serious.



Bashash in 2017, reported about 4 IQ loss at 0.7 ppm fluoride in water.



The Board's claim and recommendation that fluoridation is safe is factually, empirically unsupported, and is not based on current scientific evidence, law or logic. For almost two decades the Board has been given quality research, but not in as high a scholarly presentation as the NTP monograph. The Board's claim of efficacy and safety is wrong and harming the public.

Hearing a Board member say*, but we are not supposed to have to review science"* makes the term "Board of Health" at best a rubber stamp of industry. Either health is based on science or trust. Trust is not empirical and factual evidence. HHS Rachael Lavine's blocking of release of the evidence did not change the science or protect the public health and neither does the Board of Health promote health if they avoid and evade science.

Fluoridation at 0.7 mg/L is not reported safe. **"A Benchmark Dose Analysis for Maternal Pregnancy Urine-fluoride and IQ in children . . . 0.2 mg/L"** Grandjean 2022.

Dr. Granjean is a professor at both Harvard and the University of Southern Denmark and has published hundreds of studies on the toxicity of chemicals. You will hear from equal but

not more accomplished research scientists in the field of toxicology.

How does the fluoridation lobby respond to the evidence?

In court the defense (fluoridation lobby) agreed fluoride is a developmental neurotoxicant. The question they refuse to answer is at what dosage is the end point. . .they are uncertain but don't claim fluoridation is safe. In other words, over 70 human studies are just not quite enough to be sure, absolutely confident, fluoride harms the developing brain at any specific developmental stage, age, location, gender, race, dosage, etc. Yes, high dosages, but they won't answer what is safe.

The trick to defending toxic substances is to divide the evidence enough to remove confidence. For example, avoid studies with higher concentrations than the "mean" intake as inconclusive to confirm absolute confidence of harm. Demand the evidence show proof of harm. The lower concentrations of fluoride studies can be divided from those evaluating prenatal IQ loss from infant IQ loss. Discount studies from countries like China

(Ok to use their toxic waste in our water, but their research reporting harm is not to our standards.) Avoid total fluoride exposure, don't include those who drink the most water, avoid any other possible risks or confounders. In other words, divide the research enough times and there are not enough studies in each sub section to reach their level of confidence to establish a threshold.

The fluoridation lobby is requiring PROOF OF HARM rather than assuring the public of safety.

And the Epidemiologists and toxicologists will clearly state they are not risk analysis experts but they don't agree with the risk analysis experts, because the risk analysis experts claim fluoride at fluoridation concentrations is a developmental neurotoxicant.

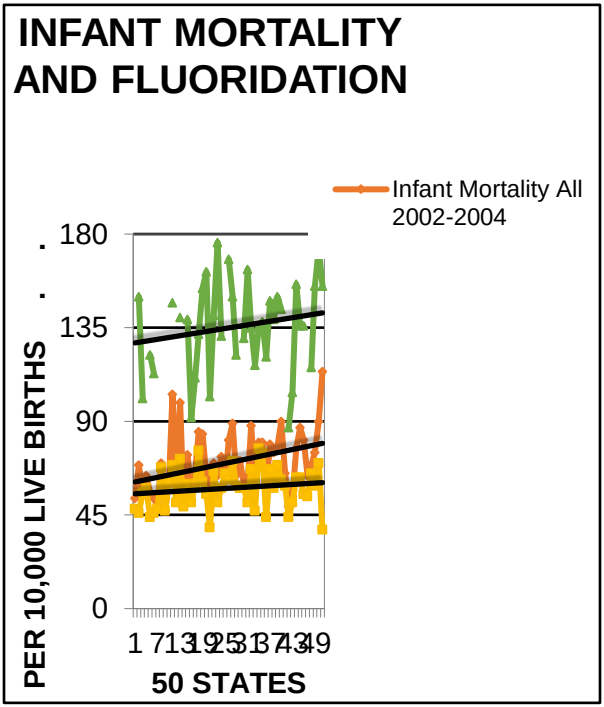
Not once do the fluoridation lobby experts answer the question "can you assure the public that fluoridation is safe?"

Not once do the fluoridation lobby experts answer, "would you recommend your pregnant daughter or grandchildren drink fluoridated water? The answers are pretty clear.

INFANT MORTALITY

It should be noted that IQ is simply one method of measuring brain damage and developmental toxicity from fluoride. I once again ranked the states on the percentage of their whole population fluoridated and plotted infant mortality per 10,000 live births, and found about 15% increase in infant mortality. See graph below.

Infant mortality is complex. The most common causes of infant mortality in the United States are birth defects, preterm birth and low birth weight, sudden infant death syndrome (SIDS), pregnancy complications,



accidents and toxins such as lead and the evidence fluoride contributes to infant mortality is growing.

Do not assume these other birth defects are not increased with fluoridation, we simply have not looked.

Data on infant mortality is readily available and the USA has a poor record compared to other countries trying to keep babies alive during their first year of life. Confounding factors need to be considered. This is a pilot study and not proof. However, the Board cannot assure the public fluoridation is safe simply because we do not have absolute proof of harm for each risk.

A pilot study using U.S. Government records reported an increase in infant mortality (perhaps 20% increase) and premature births in fluoridated communities with soft water, such as Seattle water. See Figure 3 below.

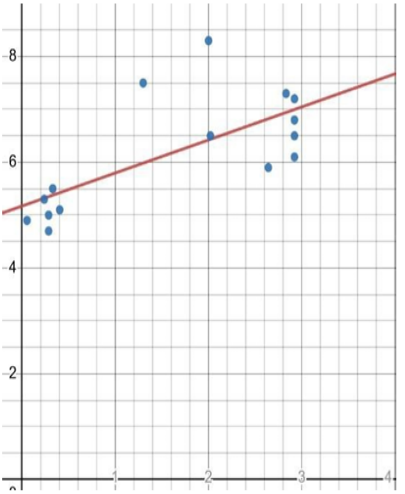


Figure 3: Infant mortality per 1,000 live births in hard water and soft water U.S. States on the vertical axis is plotted as a function of the ratio of the percent of the state population provided fluoridated water (0.7 ppm recommended) to water hardness as the calcium carbonate concentration (mg/L). Points were fitted with linear regression given by

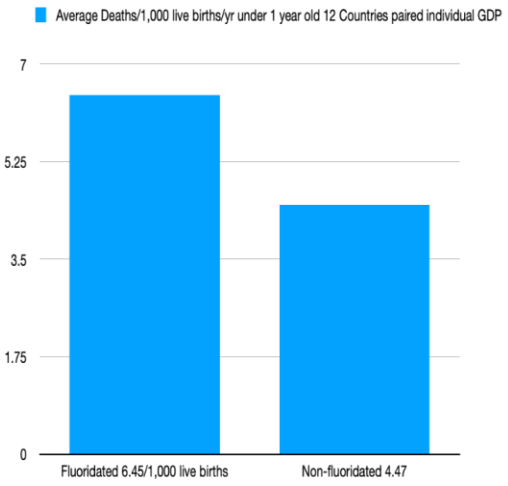
$Y = 0.627X + 5.167$ ($r = 0.694$).

In other words, add fluoride to soft Seattle water and infants have greater chance of harm and death.

Research reporting an increase in infant mortality in fluoridated communities is growing. The concern for miscarriage, and preterm birth must be considered. Although more study is always wanted, the Board must weigh the evidence with judgment.

Even if there were a decrease in dental caries from fluoridation, potential increase in infant mortality far out-weighs potential alleged benefit to teeth, which we can fix.

I recently compared six highly fluoridated countries paired economically (individual GDP) with six countries without fluoridated water or salt. Comparing these countries results in almost 30% increase in infant mortality.[38] Six countries is a small sample and fluoride is certainly not the only contributing factor for infant mortality.



The trend is serious and in keeping with the developmental neurotoxicity of fluoride.

[38] Six highly fluoridate countries were paired with six countries with no fluoridated water or salt and similar individual GDP's or area. Infant mortality rates based on CIA.gov data, GDP per Capita - Worldometer (worldometers.info), and fluoride concentrations in water

Preterm birth is defined as birth prior to 37 weeks of pregnancy. Damage to cerebral white matter is the most commonly recognized pathology of prematurity, say neuroscientists at the Dana Alliance for Brain Initiatives. "Babies born preterm face a range of potential neurological disruptions … The earlier the birth, the greater the risk that these disruptions will produce devastating and potentially life-long cognitive, behavioral, and socialization deficits."[39]

Hart reported, in 2009,

"Domestic water fluoridation was associated with an increased risk of PTB (9545 (6.34%) PTB among women exposed to domestic water fluoridation versus 25278 (5.52%) PTB among those unexposed, p < 0.0001)). This relationship was most pronounced among women in the lowest SES groups (>10% poverty) and those of non-white racial origin. Domestic water fluoridation was independently associated with an increased risk of PTB in logistic regression, after controlling for age, race/ethnicity, neighborhood poverty level, hypertension, and diabetes."

1.[39] Patoine B. The vulnerable premature brain: Rapid neural development in third trimester heightens brain risks. Dana Foundation. May 2010. Available at https://www.dana.org/media/detail.aspx?id=27882.

The fluoridation lobby demands proof of harm. One public health dentist told me he would promote fluoridation until it was proven people were falling over in the street dead from fluoridation.

These possible deaths of our babies, our future, our most vulnerable who the Board is NOT protecting must not be ignored. Harming their brains and possibly their deaths, certainly harming teeth and bones, without proof of efficacy is unforgivable. The Board members, and all of us who did and still do promote the ingestion of additional fluoride without patient consent are or have been complicit. And I too promoted fluoridation and was complicit in the harm.

The Board makes no sense to medicate everyone with a highly toxic poison, to be regulated as a drug but not, with 2 out of 3 children showing a biomarker of excess fluoride exposure, with doubtful benefit for a non-contagious, almost never lethal disease, without a doctor's supervision, of a known legend drug, and the Board expects the patient to provide absolute proof of harm and precise dosage.

The Legislature did not charge the voters to assure safety.

DENTAL FLUOROSIS was briefly covered above when discussing the Board's website. See page 109. Much more could be added.

FLUORIDE AND CANCER

It has been said, "Genes load the cancer gun, environment pulls the trigger."

One of the problems with cancer research is latency. It can take 20 to 30 years after exposure to the primary etiology.

Dean Burk PhD, head of cytochemistry, National Cancer Institute 1974, Co-discoverer of Biotin compared 10 large unfluoridated cities as controls 6.3 million people with 10 large cities which became fluoridated between 1952-1956, 11 million people.

Cancer Deaths/100,000

year	1940	1950	1970
CDRo (+F)	154.2	186.3	222.6
CDRo (- F)	153.5	183.6	188.8

Representing a 31.3/100,000 increase in deaths/yr after 15-20 years of fluoridation

When I was in Dental School, we were shown a critical review of Burk's work which suggested two significant numbers were transposed and no adverse effect had been shown.

However, we were not told that Burk had responded with evidence that the critics had transposed the numbers and he was indeed correct.

Burk's study stopped when the unfluoridated cities became fluoridated.

Although NRC (2006) committee reviewing fluoride for the EPA was charged with "non-cancer" effects of fluoride, fluoride increasing cancer is biologically plausible and a connection between fluoride and osteosarcoma, focuses on three facts:

1. Most fluoride is stored in bones, particularly during growth spirts.
2. Fluoride is a mutagen
3. Fluoride stimulates osteoblasts which “increases the risk for some of the dividing cells to become malignant.” (NRC 2006) See a timeline link.

Some history on fluoride and cancer as reported by Ellen Connett in 2014. See endnote[40]

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[40] In the 1980s the US Congress mandated the National Toxicology Program to conduct animal studies to determine if fluoride causes cancer. Battelle Columbus Laboratories were contracted to perform the studies that began in 1985 and ran for 2 years. In 1988 Battelle submitted their final report that included the finding of a dose-dependent increase of a rare liver cancer (hepatocholangiocarcinoma) in male & female mice and a small but statistically significant dose-related increase in osteosarcomas in male rats but not in the female rats. For the rare liver cancer, the first scientist to describe this cancer said that Battelle made a correct diagnosis. However, this rare liver cancer was reclassified by a government review panel as a non-cancer and one of the osteosarcomas was downgraded leading to the

classification of “equivocal evidence of cancer”. There were also increases in oral and thyroid cancers, but they were not considered statistically significant.

The politics that raged around this study.
William Marcus, the senior scientist in the Office of Drinking Water at the Environmental Protection Agency, expressed concerns about the “systematic downgrading” of cancers in the 1990 published study and requested that the EPA assemble an independent board of pathologists and others to review the data produced in the study. In the 2013 documentary *Fluoridegate: An American Tragedy*, Marcus has this to say about the study:
“… rats got cancer of the bone and they got a very unusual cancer of the liver. And that was extremely surprising. First of all to produce cancer of the bone in rodents is never seen because the time that you have between birth and death of a rodent is only 3 ½ to four years and it usually takes longer than that to produce a cancer in bone. The cancer of the liver is extremely rare … and the fact that it happened meant that it was significant. This doesn’t happen. I wrote this memo in which I claimed that I thought fluoride was a carcinogen and that we had as much evidence with the animal studies to show that it was a carcinogen as we had

with any of the other compounds [that EPA studied] and therefore should be treated as such."

Also, three out of four in-vitro tests proved fluoride to be mutagenic, which Marcus said supported "the conclusion that fluoride is a probable human carcinogen." The internal memorandum that Marcus wrote was leaked to the press. It caused embarrassment to senior EPA officials and Marcus was fired.

"An Enemy of The State"
The National Whistleblowers Association represented Marcus in his two trials against the EPA and they won both. The EPA was forced to pay Marcus' legal fees, 2 ½ years of back pay, and an undisclosed sum for damages to his reputation.

In the *Fluoridegate* documentary Stephen Kohn of the National Whistleblowers Association stated:
"… I do not know why the agency (EPA) did what it did to Dr Marcus. But I do represent whistleblowers and I can tell you they went after Dr Marcus with a vengeance, a vengeance. He was a board certified toxicologist with years of seniority, the most respected toxicologist in the agency with an international reputation. When he wrote that memo they went after him like he

was an enemy of the state. They just hammered, and hammered, and hammered, and they went way over the line by destroying evidence and obstructing justice. And even after we won the first case where he was ordered reinstated they went after him again. And even though there were 2 court rulings finding retaliation they never touched or disciplined those agency officials involved. This case marks a black mark on the EPA and raises fundamental issues about scientific freedom and about fluoride and why this agency went against one of its most respected scientists on that issue."

Robert Reich as Secretary of Labor in the Clinton administration upheld the decision of the Administrative Law Judge in 1994 who said that "the true reason for the discharge was retaliation." Reich wrote that he found particularly disturbing that the trumped-up charges against Marcus were accepted by his supervisors "in the absence of any convincing documentation."

The principal finding of NTP's study, performed by Battelle Columbus Laboratories, was a dose-dependent increase in osteosarcoma (bone cancer) among the fluoride-treated male rats.

However, despite the fact that

1) the cancer occurred in the target organ (bone) for fluoride accumulation,
2) the increase in bone cancer was statistically-significant,
3) the doses of fluoride were low for an animal cancer study, and
4) NTP acknowledged it is "biologically plausible" that fluoride could induce bone cancer,

the NTP ruled that the study only provided "equivocal evidence" that fluoride was the cause of the cancer.

The NTP did not assure the public fluoridation did not cause cancer. NTP did not have absolute proof of harm.

According to a 1990 report by Bette Hileman in *Chemical & Engineering News*: "A number of government officials who asked not to be identified also have told C&EN that they have concerns about the conclusions of the 1990 NTP study. They, too, believe that fluoride should have been placed in the "some evidence" category, in part because osteosarcoma is a very rare form of cancer in rodents."

In 2000, Dr. J William Hirzy testified before the U.S. Senate's Subcommittee on Wildlife, Fisheries and Drinking Water on behalf of the EPA's professional union, NTEU Chapter 280, requesting an independent review of NTP's cancer bioassay study.

In 2002, the World Health Organization (Fluorides: Environmental Health Criteria 227) advised scientists to take NTP's finding seriously. According to the WHO: "Such a (dose-dependent) trend associated with the occurrence of a rare tumour in the tissue in which fluoride is known to accumulate cannot be casually dismissed."

In 2005, the Environmental Working Group "asked the National Toxicology Program (NTP) of the National Institutes of Health (NIH) to list fluoride in tap water in its authoritative Report on Carcinogens, based on its ability to cause a rare form of childhood bone cancer, osteosarcoma, in boys."

In addition to increased bone cancer, the NTP study also found increases in rare liver cancers, oral cavity cancers and thyroid cancers among the fluoride-treated rats. The NTP ruled, however, that the cancers were not related to the fluoride treatment – despite reaching "statistical significance" in some of NTP's analyses.

"We observed that for males diagnosed before the age of 20 years, fluoride level in drinking water during growth was associated with an increased risk of osteosarcoma, demonstrating a peak in the odds ratios from 6 to 8 years of age. All of our models were remarkably robust in showing this effect, which coincides with the mid-childhood growth spurt. For females, no clear association between fluoride in drinking water during growth and osteosarcoma emerged." (Bassin EB, et al. 2006. Age-specific fluoride exposure in drinking water and osteosarcoma (United States). Cancer Causes & Control 17(4):421-8. May.)

Chester Dougles published a small study, 20 controls, too small for reliable conclusions, the controls were over twice the age, representing about 400% higher bone fluoride concentrations for age paired. Douglas not only used controls averaging more than double the age, but compared the osteosarcoma cases with other bone tumors as controls. Clearly, the data was collected to protect fluoride exposure. Just because the concentration of fluoride in bones of osteosarcoma patients and bone tumor patients are similar, does not mean the fluoride concentration in bone is safe. Using bone tumors as controls cooked the evidence.

As Editor of the Colgate report, Douglas received significant funding from Colgate.

Our point: Several researchers confirm, fluoride is a carcinogen. The question is the dosage for each patient.

FLUORIDE'S IMPACT ON THYROID HORMONES: THYROID, PARATHYROID, PANCREAS, PINEAL, ADRENAL, GONADS, ANTERIOR AND POSTERIOR PITUITARY, AND PLACENTA.

See Attachment #E Thyroid

Fluoride is considered an endocrine disruptor. As little as 2 to 5 mg/day can reduce most patient's thyroid activity. (Galletti & Joyet 1958)

For easy estimation, half of fluoride exposure is from fluoridated water. At 0.7 mg/L, **about six glasses of fluoridated** water along with the "average" fluoride from other sources can be expected to reduce thyroid hormones. But wait, many are ingesting more fluoride from other sources and drinking more than six glasses of water.

We in public health tell those with thyroid harm from fluoride that their obesity, diabetes, and malaise is their fault,

when in fact we are contributing to their health problems, idiopathic harm.

"We found that higher levels of fluoride in drinking water provide a useful contribution for predicting prevalence of hypothyroidism. We found that practices located in the West Midlands (a wholly fluoridated area) are nearly twice as likely to report high hypothyroidism prevalence in comparison to Greater Manchester (non-fluoridated area)." Peckham S, et al. (2015). *Journal of Community Health & Epidemiology* (see study)

The NRC 2006 review of fluoride's effect on the thyroid gland should be reviewed. See pages 224-236. *"Fluoride in Drinking Water: A Scientific Review of EPA's Standards."*

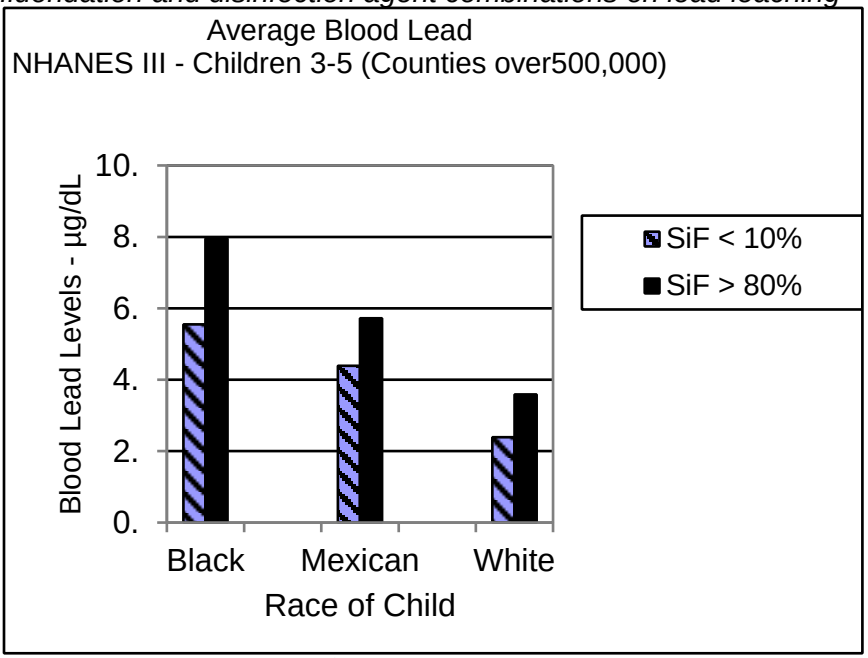
For a more referenced and scientific discussion of Fluoride's effects on the endocrine system, aggravated by iodine deficiency, effects on goiters, impact on thyroid hormones and excess iodine intake, see here and pubmed.gov.

FLUORID AND LEAD

*Blood **Lead** levels in Fluoridated areas 2X higher for Whites and 6X higher for Blacks*[41]

[41] Confirmation of and explanations for elevated blood lead and other disorders in children exposed to water disinfection and fluoridation chemicals. Coplan

Prevalence of children with elevated blood lead (PbB>10mug/dL) is about double that in non-fluoridated communities. When FSA was added "lead concentrations spiked to over 900 ppb. Effects of fluoridation and disinfection agent combinations on lead leaching



from leaded-brass parts.[42]

MJ, Patch SC, Masters RD, Bachman MS. Neurotoxicology. 2007 Sep;28(5):1032-42. Epub 2007 Mar 1.
See also: Masters RD, Coplan M. 1999 International Journal of Environmental Science 56: 435-449.
And: Masters RD, Coplan MJ, Hone BT, Dykes JF. 2000 Neurotoxicology 21(6): 1091-1100.

[42] Maas RP, Patch SC, Christian AM, Coplan MJ. Neurotoxicology. 2007 Sep;28(5):1023-31. Epub 2007 Jun 30
See also: Blood lead concentrations in children and method of water fluoridation in the United States, 1988-1994. Macek MD, Matte TD, Sinks T, Malvitz DM. Environ Health Perspect. 2006 Jan;114(1):130-4.

FLUORIDE'S IMPACT ON BONES

Skeletal fluorosis is an undisputed effect of excess fluoride. The EPA uses severe skeletal fluorosis as a threshold of concern for excess fluoride exposure. But pathology from fluoride starts much sooner than crippling skeletal fluorosis.

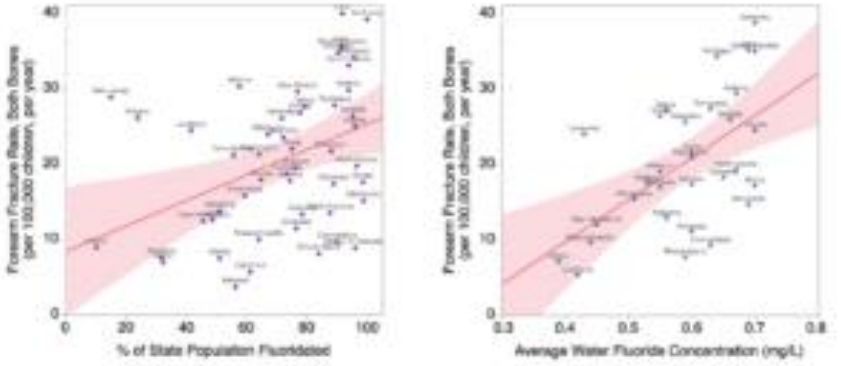
Fluoride seemed like a good idea for bones and teeth to make them harder, until studies such as Helte et al raised concerns of bone fracture and osteoarthritis, arthritic like symptoms, stiffness and pain in joints. BAO 2003 (Luo 2012; Su 2012; Bao 2003; Savas 2001; Tartatovskaya 1995; Chen 1988; Xu 1987)

A recent study in the Journal of the American Academy of Orthopaedic Surgeons by Lindsay et al. Results:

"Positive correlations were found between the percentage of state water fluoridation and fracture rates for both bone forearm fracture (BBFFx) and femur fracture. Fluoride levels had positive correlations with fracture rates for all fracture types. Increased fracture rates were found between states in the highest quartiles of percentage of state water fluoridation and fluoride water levels for supracondylar humerus fracture and BBFFx."

The study reported at 0.7 mg/L fluoride in water, rates of child forearm fractures were 2.5 times greater than in states with

the lowest average concentration, which was about 0.4 mg/L as illustrated here:



(quality of graph is also hard to read in the Journal, but the data is also printed)

AUTHORITIES

The fluoride lobby will often claim hundreds of organizations endorse fluoridation. I doubt any have reviewed the science, they simply trust others.

Here are a few with researvations:

I. The Washington State Board of Pharmacy and RCW:

The Board of Pharmacy was disbanded in part because they agreed with the law and science that fluoride ingested with intent to prevent disease is a prescription drug.

Neither the Board, voters, nor water purveyors have authority to prescribe drugs. At least the Board of Health can provide accurate information for water purveyors and the public.

Pharmacists have more training and expertise with toxins, dosage, adverse reactions and inter reactions of toxins than other licensed professions and weighing their judgment is essential.

"RCW 18.64.011

(14) "Drugs" means:
(a) Articles recognized in the official United States pharmacopoeia or the official homeopathic pharmacopoeia of the United States; [sodium fluoride is listed]

(b) Substances intended for use in the diagnosis, cure, mitigation, treatment, or prevention of disease in human beings or other animals;" [intended use is to prevent a disease]

II. **U.S. Congress** which has authorized the Food and Drug Administration Center for Drug Evaluation and Research (FDA CDER) to evaluate substances used with intent to prevent disease and Congress prohibit the EPA from adding anything for the treatment of humans.

Again, the authority of the US Congress, designating the FDA CDER with authority over drugs.

III. **FDA CDER** has determined fluoride ingestion lacks evidence of efficacy. And the FDA has given warnings to bottled water manufacturers (not FDA CDER approved) the fluoridated water must not be marketed to those under two years of age. The FDA indicated requiring FDA approval would effectively ban fluoridation. The Board of Health is harming the public by disagreeing with authorized regulatory agencies.

IV. The Environmental Protection Agency scientists finding over two decades ago that fluoridation borders on a criminal Act because of toxicity and lack of current benefit. And the EPA Dose Response Analysis and Relative Source Contribution of 2010 reporting that most or all infants and toddlers are ingesting too much fluoride.

V. The National Research Council 2006 report for the EPA that EPA's Maximum Contaminant Level for fluoride was not protective. That's right, fluoride is a contaminant the Board recommends adding to water.

VI. The National Toxicology Program: Draft Report of 2023 report of 55 human studies, 52 reported IQ loss, a 95% consistency. And their meta-analysis reports IQ loss. Not everyone has the same sensitivity to drugs/toxins or the same health or the same ability to handle drugs/toxins. Some individuals had much more IQ loss and some were probably unaffected. The mean is not protective or representative of each

individual. The Board must protect everyone, not just the healthiest and wealthiest.

VII. Lack of quality research: Only one RCT (randomized controlled trial, the highest quality of research) of fluoride ingestion has been published and it report no statistical benefit from ingesting the fluoride. That's right. NO, NONE, ZERO quality studies reporting dental benefit of fluoride ingestion. No wonder the FDA said the evidence of efficacy is incomplete.

VIII. The lack of mechanism of action: Fluoride cannot go from the blood to the tooth pulp chamber through the calcium rich dentin and enamel to the outside of the tooth where the dental caries are forming and active. Fluoride's contact with teeth during swallowing of water is short term, and little gets to the lower teeth. The theoretical slight increase of fluoride in saliva with water at 0.7 ppm is too dilute to have an effect. Research has not reported a benefit at 700 ppm let alone 0.7 ppm.

IX. 97% of Europe does not fluoridate their water. And their dental caries are a similar rate as fluoridated communities and states not fluoridated.

X. The Court: In Doe v Rumsfeld, ruled that even under emergency conditions of war, the Government cannot force an individual to be medicated with a substance which has not been specifically approved for the purpose and manner it is intended. Fluoride ingestion is unapproved and therefore illegal, unless an authorized prescribing health care provider prescribes the fluoride for their patient of record off label. Ther is no approved label for fluoridation or fluoride tablets.

The Board appears to trust industry who profit from the sales of fluoride**.** We dentists make a ton of money off of fluoride. . . topical which has good evidence of efficacy. Raising alarms of fluoride toxicity will reduce our income, but speaking up against fluoridation harms a dentist's reputation among peers.

The Board appears to trust the CDC dental division who are in lockstep with industry and politics, not scientific facts. The CDC does not determine either the efficacy, dosage nor safety of any drugs. Congress charged the FDA CDER with that job.

The Board appears to trust the US Public Health Service, but not the NTP within the USPHS. The USPHS has no Congressional authority to approve the safety, dosage or efficacy of any drugs and fails to review the scientific evidence.

The Board appears to trust public health reviews of fluoridation from like-minded believers rather than digging deep into the science.

This request for rule change is to protect the public from harm caused by too much fluoride ingestion, in part, promoted and encouraged by the Board of Health.

You will get pushback from the dental lobby and industry profiting from the sale of fluoride. And you will get push back from those who have not evaluated both sides of the science. We can

and should agree that many are ingesting too much fluoride and the early days, months, years of life appear to have the greatest risk of harm.

The exact individual health, dosage, mechanism, age, race, diet, and synergistic chemical effects from other toxins are less certain and in time will be more thoroughly studied. Fluoridation should be stopped; however, the paradigm shift maybe too much for the Board.

We must and will, someday after many millions are harmed, simply turn off the fluoride pumps. At a minimum the Board can start to consider science and start on a label to protect the unborn, infants and young.

Much more evidence could be added. This is a brief summary of reasons the Board cannot assure the public fluoridation is safe.

This petition will start to protect the public from over exposure to fluoride and although not assure the public is safe, will be a good first step. This is not a definitive review of literature, rather a more than adequate review to determine fluoridation cannot be assured to be safe.

Note, I promoted fluoridation for a quarter of a century and am complicit in the harm which has been caused to the developing brains of the public. If you feel I have thrown a stone at you, I am passionate because the stone hit me first.

Sincerely,

Bill Osmunson DDS MPH

Washington Action for Safe Water

Washington State Board and Department of Health
PO Box 47990
Olympia, WA 98504-7990
wsboh@doh.wa.gov

February 18, 2024

Washington Action for Safe Water

Bill Osmunson DDS MPH

Dear Washington State Board of Health (Board) and Department of Health (Department),

RE: PART II: SOME REASONS FLUORIDAITON IS AN ENTRENCHED DENTAL PUBLIC HEALTH BELIEF. FURTHER TO OUR PETITION FOR RULE MAKING: WATER FLUORIDATION,

As defined and regulated by the FDA, fluoride is a drug. It is the only drug anywhere in the world allowed to be administered through public drinking water. There is absolutely no control over who gets it, how much, for how long and no warnings of its potential harmful side effects. It violates every protocol of prescribing a drug by a physician.

"The dose makes the poison." If you put this drug into the drinking water, you can't control the dose. If you can't control the dose, you can't control the poison. It defies common sense to put ANY drug into tap water.

OUTLINE

I. Failure to consider long term disease trends, morbidity

II. Failure to critically examine and confirm how the theory started

III. Failure to require quality research and Failure to research HARM/SAFETY

IV. Failure to be inclusive of those who disagree with us

V. Failure to combine all streams of evidence.

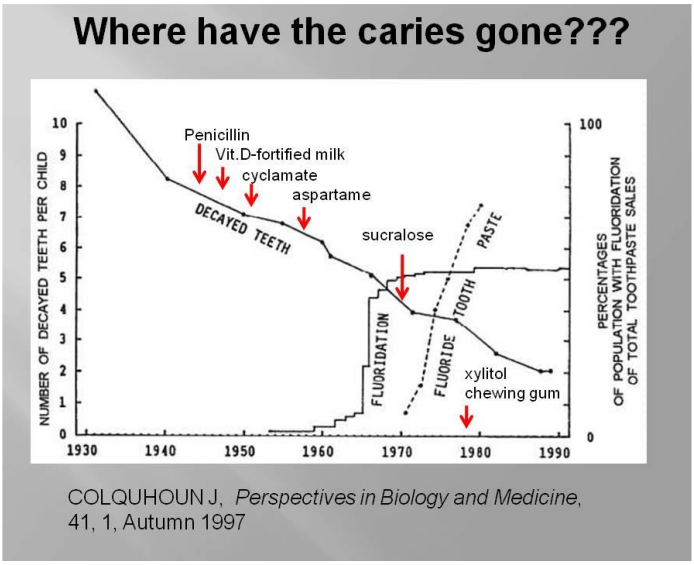
VI. How to Hide the Evidence of Harm

VII. Failure to critically question those we trust

I. Failure to consider long term disease trends, morbidity

Diseases have cycles. We know the yearly increase and decrease cycle in diseases such as influenza. However, there are long term cycles over many years. In research, controlling for the cycles can be problematic.

Once again, a graph of dental caries over 60 years. The fluoridation lobby has not answered the critical question, what caused dental cavities to decline **prior** to



fluoridation? No one knows. This graph suggests some possibilities for caries reduction. Others have speculated possibly better nutrition with fresh fruit and vegetables shipped year-round, i.e. transportation.

Certainty, the caries decline prior to fluoridation was not caused by fluoridation. Research can't knowingly and adequately control for the powerful effect of those unknowns. Critical thinking must question the confidence of later research when we don't know why caries declined before fluoridation and are unable to control for those unknowns. Just because two events happen, is not proof they are related.

II. Failure to critically examine and confirm how the theory started

An excellent easy book to read is by Christopher Bryson, “The Fluoride Deception” How a Nuclear Waste Byproduct Made Its Way Into the Nation’s Water Supply. I don’t like the title, because it sounds like a “deep state conspiracy.” However, the book is well documented, fluoridation is not “deep state conspiracy” and the book is an easy read.

To make the atomic bomb, back in the 1940’s, fluoride was and is used to refine uranium. Research was done to determine how hazardous the fluoride would be. The option was given, be safe, do the research slowly and with precautions, or build the bomb fast and some people will be harmed. World War II was in progress and many were dying. The choice was made to build the bomb fast at the risk of workers in an effort to save soldiers. The research was part of the Manhatton Project and not till years later became public. Meanwhile, the public was assured fluoride was safe.

Public Health employees were hired to promote fluoridation and early research, although flawed, became fact. Theory became fact without adequate research.

My last class in my master’s program the professor was telling us how we were to promote policy regardless of our personal opinion. I raised my hand and asked, “what if my boss tells me to promote tobacco smoking.” He paused and said, “promote tobacco smoking but not to the best of your ability.” I changed professions because I could not ethically support condoning or remaining silent when people are being harmed. Silence is not always silent.

Fluoride was alleged to prevent dental caries because people living in naturally high fluoride areas appeared to have fewer cavities, or was it the minerals? The phosphate fertilizer companies were spewing fluoride scrubbings into the environment causing serious damage. The obvious solution to the pollution was dilution.

Read Bryson’s book. I’ll send you mine if you ask. Well referenced and an easy read.

III. Failure to require quality research and Failure to research HARM/SAFETY

DO NOT CONFUSE CLAIMS OF EFFICACY WITH ASSURING SAFETY

The Board is to assure safety, not efficacy. To assure safe water requires safety studies. The fluoridation lobby will constantly move the discussion to efficacy rather than provide safety studies. Many studies "claim" safety but do not evaluate safety. The Board claims they have thousands of studies on safety, references are missing.

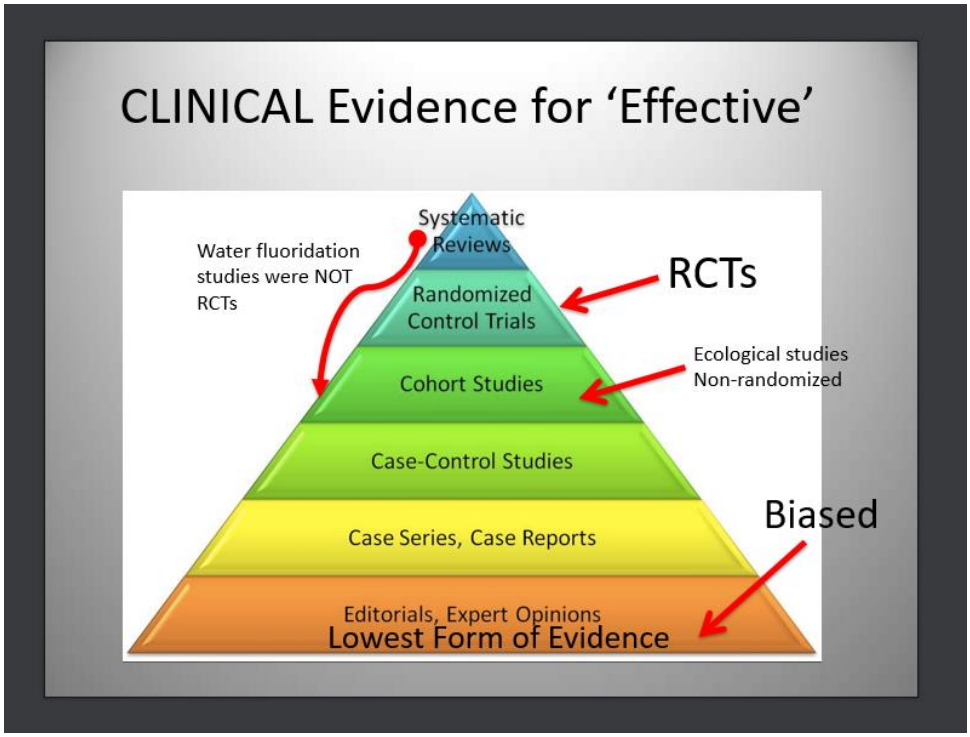
However, the Board should also understand the studies on efficacy have not risen to the quality level of FDA CDER approval. Randomized Controlled Trials (RCT) are considered the "gold standard" for research. RCTs are prospective studies, essential to determine efficacy. Researchers and subjects are both blinded, they don't know if they have the drug or a placebo. Subjects are randomly selected and the study is prospective in design. Even those are not "proof" positive of benefit and long-term risks are seldom considered.

No RCTs have been done with community water fluoridation. They could be done, but more complex because we are dealing with tap water. However, fluoride tablets could be used and one was published, but it did not show significance.

Without RCTs we are left with lower quality studies which have less confidence and safety is not readily observable.

Fluoridation efficacy studies are not RCTs and have more uncertainty. Safety studies are lacking, incomplete. For example, the NRC 2006 report highlighted inadequacies in many areas of "safety" research.

A pyramid of increasing quality of studies for efficacy is provided below.



Safety is difficult to study. Research on safety cannot have RCTs. It would be unethical to intentionally cause harm. The study of fluoridation's safety, at best, uses cohort studies, ecological studies and seldom monitors side effects other than dental fluorosis. Seldom is money put into determining risk and harm because there is no profit looking for harm. And if harm is found, liability becomes a concern.

Our recent experience with COVID should give the Board pause. Many were dying, hospitals full, an experimental vaccine came out and many of us agreed the public must get the vaccine, and most of us did. Risks were minimized. Harm may have been under reported and studies incomplete.

The same minimizing of risks, marginalizing of harm, lack of safety studies and robust support from authorities, has taken place with fluoridation over the past 80 years, except dental caries are not highly lethal nor contagious. The vaccine has RCTs, FDA CDER NDA approval, labels, dosage, doctor's oversight, and patient

consent. Fluoridation has none of those. Even choosing where we live does not avoid the water because we don't know if the drinks or processed foods contain fluoridated water. (I am not anti-vaccination. The illustration is used because I know the Board is well aware of the public's concern.)

Some research supporting fluoridation's efficacy, include the 2000 York Review, the Community Preventive Task Force of 2013, the 2017 Australian Government Review, the 2022 Brazilian Systematic Review. However, these and others were stacked with believers who confirmed their belief, and did not seriously evaluate safety. For example, if we survey Ford dealers, guess which truck comes out as the best. "Safe and Effective" has been repeated so many times, we assumed it true.

The Cochrane Review of fluoridation in 2015 was slightly different and a better quality of review. However, Cochrane Reviews require RCT studies. None exist, so Cochrane failed to require RCTs. Cochrane limited the studies reviewed to lower quality prospective studies.

When you listen to the fluoride lobby, they will almost always limit their comments to Cochrane's statements that *"fluoridation is effective at reducing levels of tooth decay among children."* And will fail to mention the Cochrane study reservations. Summarized:

1. "These results are based predominantly on old studies and may not be applicable today."
2. "we did not find any on the benefits of fluoridated water for adults."
3. "We found insufficient information about the effects of stopping water fluoridation."
4. "We found insufficient information to determine whether fluoridation reduces differences in tooth decay levels between children from poorer and more affluent backgrounds."

5. “We had concerns about the methods used, or the reporting of the results, in the vast majority (97%) of the studies.”
6. “For example, many did not take full account of all the factors that could affect children’s risk of tooth decay or dental fluorosis.”
7. “There was also substantial variation between the results of the studies, many of which took place before the introduction of fluoride toothpaste.”
8. “This makes it difficult to be confident of the size of the effects of water fluoridation on tooth decay or the numbers of people likely to have dental fluorosis at different levels of fluoride in the water.”
9. **Authors' conclusions:**

10. There is very little contemporary evidence, meeting the review's inclusion criteria, that has evaluated the effectiveness of water fluoridation for the prevention of caries.

11. The available data come predominantly from studies conducted prior to 1975, and indicate that water fluoridation is effective at reducing caries levels in both deciduous and permanent dentition in children. Our confidence in the size of the effect estimates is limited by the observational nature of the study designs, the high risk of bias within the studies and, importantly, the applicability of the evidence to current lifestyles. The decision to implement a water fluoridation programme relies upon an understanding of the population's oral health behaviour (e.g. use of fluoride toothpaste), the availability and uptake of other caries prevention strategies, their diet and consumption of tap water and the movement/migration of the population. There is insufficient evidence to determine whether water fluoridation results in a change in disparities in caries levels across SES. We did not identify any evidence, meeting the review's inclusion criteria, to determine the effectiveness of water fluoridation for preventing caries in adults.

12. There is insufficient information to determine the effect on caries levels of stopping water fluoridation programmes.

13. There is a significant association between dental fluorosis (of aesthetic concern or all levels of dental fluorosis) and fluoride level. The evidence is limited due to high risk of bias within the studies and substantial between-study variation.

The Cochrane review lacks confidence in efficacy. However, based on the limited evidence available, Cochrane reviewers reported “a significant association between dental fluorosis and fluoride level.” Safety from dental fluorosis is not assured, in fact undisputed. The Board’s job is to assure safety, not efficacy. The fluoridation lobby will claim dental fluorosis is just a slight blemish and of no concern. Patients disagree, almost half would like the spots removed. When both functional and cosmetic dental fluorosis harm is combined, dentists make

a significant amount of money selling the fluoride and treating the fluorosis caused by excess fluoride.

The absence of evidence of research safety is not proof the water is safe. In 2006, the National Research Council raised doubt fluoride was safe for:

1. Tooth Damage
2. Rheumatoid and Osteoarthritic-like Pain
3. Bone cancer
4. Bone Fractures
5. Thyroid Reduction, Obesity & Diabetes
6. Kidney damage
7. Reproductive problems
8. Lower IQ and Increased Mental Retardation
9. Allergies (overactive immune system)
10. GI disorders.

Further studies on each of those risks has supported concern of harm and has not assured us fluoridation is safe.

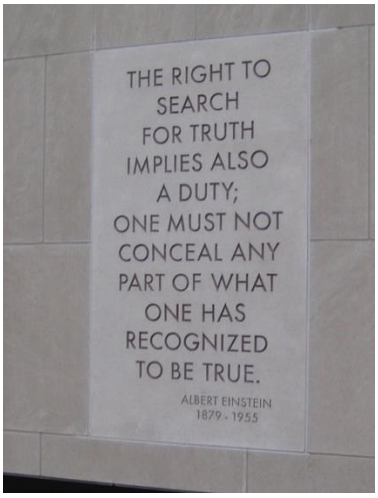
IV. Failure to be inclusive of those who disagree

The Chair of the National Research Council 2006 report on fluoride for the EPA which reported EPA's MCL was not protective, confirmed that this review was unique in that it was the first time a review committee had been formed which did not limit the members to those who supported fluoridation.

If only those who support a theory are asked to review the theory, the biased conclusion is possibly determined prior to the evaluation of the studies. Efficacy is claimed, harm is minimized and ignored.

V. Failure to combine evidence from all streams.

Our petition reviewed some streams of evidence. Keep in mind, it wasn't until the NRC 2006 committee raising concerns of fluoride's risks that some research funding started to evaluate primarily one risk, developmental neurotoxicity. The belief by authorities that fluoridation was safe, caused the few researchers evaluating safety to lose their laboratories, their funding, and some of them their jobs. Avoiding publishing their results of harm, protected future funding for their further research.



On the National Academies of Science Building, a plaque has been placed, pictured here. Failure to publish non-supporting evidence or cherry-picking evaluators or research is part of concealment.

Foreign countries started to evaluate fluoride's developmental neurotoxic effects before English speaking countries and those studies were first translated by the Fluoride Action Network. The Fluoride Action Network claimed to have a larger data base on fluoride than the Library of Congress (on developmental neurotoxicity of fluoride) due to translation of research into English.

Research started mainly on developmental neurotoxicity, brain damage while the brain is developing. Understanding the relationship between a toxin and the developing brain takes years for the child to develop and many more to understand toxic effects for adults. Measuring fluoride exposure and possible miscarriage, premature birth, infant mortality, and a host of other risks later in life, also takes time and funding. And studies must be repeated to achieve confidence.

Assuring safety is required, not proving harm beyond doubt.

VI. The Fluoridation lobby hides the harm: How to Hide Harm

1. Divide the streams of evidence and don't consider all the evidence.
2. Divide each stream of evidence enough times and raise doubt each specific aspect has absolute confidence of harm. Assuring safety is not the criteria. Proof of harm is required. And proof of harm takes many years, many studies, and a ton of money.
3. Divide each study, for example, divide subjects on natural fluoride from artificial fluoridation, divide the methods of measuring fluoride, age, gender, race, geography, health status, socioeconomics and the number of cohorts drops below significance.
4. If confusion and doubt on the harm is not achieved with those tricks, assume everyone fits in the mean. For example, assume everyone is in the mean and everyone drinks the same amount of water,
5. Assume other minerals in the water, such as calcium, have no effect.
6. Assume the comparison is only fluoridated water with zero fluoride exposure.
7. The EPA hired experts testified in court and agreed above 1.5 ppm fluoride concentration in water, the evidence is reasonably consistent fluoride is a developmental neurotoxin. However, below 1.5 ppm fluoride in water the EPA experts suggested the research is "inconsistent," less certain. Most of their doubt was based on one study which has been discredited.
8. Assume concentration is dosage. Pretend the only source of fluoride comes from water and if fluoridation is 0.7 ppm, then 1.5 ppm would be safe, assuming everyone drinks the same amount of water and no other source of fluoride.
9. Many experts suggest, for easy figuring, half the fluoride comes from water and half from other sources, although 1/3rd or 2/3rds is more realistic. In other

words, 0.7 ppm in water plus the same dosage of fluoride from other sources and would be close to an equivalent of 1.5 ppm fluoride. At that exposure level, court experts, both plaintiffs and defense, agreed with the NTP that fluoride was reasonably considered a developmental neurotoxin.

10. Although both a review by Canada Health experts and EPA's hired risk assessor in court refused to suggest an intraspecies uncertainty factor, even a 1:1 puts many in harm because not everyone is average. Most toxins have a 10:1 or 100:1 safety factor, or at least a 3:1 which would put many at risk of harm. If a 10:1 is used, water fluoridation should not exceed 0.15 ppm and a 3:1 would not exceed a 0.5 ppm concentration of fluoride in water. Fluoridated water at 0.7 cannot be assured safe. Even a 0.5 ppm concentration would still be much higher than mother's milk which has about 0.004 ppm fluoride concentration.
11. The third trimester of pregnancy is critical for fetal brain development and the average mom to be ingests 3.1L of water.
12. More research is always desired, but not necessary for us to be confident the Board cannot assure fluoridation is safe. For example, modeling or physiologically-based pharmacokinetic modeling that predicts how a chemical will be absorbed and metabolized by the body, hasn't yet been done for developmental neurotoxicity. . . or even after almost 80 years for fluoride ingestion, i.e. fluoridation.

The fluoridation lobby fails to take their criticisms of incomplete lack of proof of harm, safety research, and apply those criteria to their claim of efficacy.

VII. Failure to critically question those we trust

I trusted my professors on many issues and that was wise and essential, because that was the best they knew. However, one of my mentors reminded me that half of what they taught was wrong and they didn't know which half. In other words, we must be humble and not camp on any theory. We don't know it all and never will. The more of an expert in an area we become, the less dogmatic we become.

I was a school board trustee in a rural red neck community. The Chair wisely handled the public comments, "I've never learned anything from those who agree with me."

One of the reviewers of the NTP report on developmental neurotoxins, was also an expert witness in court defending the EPA's 4 MCL. The expert has, reportedly, been testifying for 34 years as an "Epidemiology Consultant," mostly lawsuits for defense of pesticides such as for Paraquat, manufacturers such as Syngenta and Chevron. Most countries have banned Paraquat which reportedly increases Parkinsons and is an acute poison. Another issue he has apparently been defense expert for is the cell phone companies and the research the electromagnetic fields from power lines and cell phones contribute to cancer. He does not say they are safe, simply raises enough doubt to stop regulation.

The Board's job is not to determine a confidence level of "proof of harm" but to assure without doubt the water is safe.

Our petition provides education for those evaluating fluoridation and the public. It is a start, although it will not stop fluoridation.

Washington Action for Safe Water

FLUORIDATION PRODUCTS (FLUORIDATED WATERS (TAP OR BOTTLED) AND FLUORIDATION CHEMICAL ADDITIVES) ARE DRUGS

by

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ABSTRACT: This paper presents a legal analysis that demonstrates that fluoridation products (fluoridated waters (tap or bottled) and fluoridation chemical additives) are drugs under the jurisdiction and responsibility of the federal Food and Drug Administrative (FDA) when the intended use is prevention of tooth decay disease.

(A public paper released October 29, 2016)

TABLE OF CONTENTS

ATTACHMENTS

ABBREVIATIONS

ANDA	Abbreviated New Drug Application
ANSI	American National Standards Institute
CFR	Code of Federal Regulations
DSHEA	Dietary Supplement Health and Education Act
EPA	Environmental Protection Agency
FDA	Food and Drug Administration
FDCA	[federal] Food, Drug, and Cosmetic Act
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FR	Federal Register
HHS	Health and Human Services
MCL	Maximum Contaminant Level
MOU	Memorandum of Understanding
NDA	New Drug Application
NRC	National Research Council
NSF	National Sanitation Foundation (now International)
OTC	Over-The-Counter
SDWA	Safe Drinking Water Act
TSCA	Toxic Substances Control Act
USC	United States Code

1. Review of federal drug laws and regulations

a. The 1906 and 1938 Acts of Congress

Drug regulation in the United States began with the Colonies and States adopting isolated laws as early as 1736. (Abigail Alliance for Better Access to Developmental Drugs v. von Eschenbach, 495 F.3d 695, 703-04 (D.C. Cir. 2007).) As early as 1848, the United States began limited drug regulation. (*Id*. at 704.) Congress adopted more comprehensive drug statutes in the Food and Drugs Act of 1906, which prohibited the manufacture of any drug that was "adulterated or misbranded." (*Id*. at 705.) This Act defined "drug" as:

> all medicines and preparations recognized in the United States Pharmacopoeia or National Formulary for internal or external use, and any substance or mixture of substances intended to be used for the cure, mitigation, or prevention of disease of either man or other animals;

and defined "food" as including "articles used for food [and] drink." (Food and Drugs Act of 1906 (emphasis supplied), 34 Stat. 768 (1906).)

Initially, this Act did not regulate false claims of the curative power of a drug but this was changed by Congress in 1912. (Samuels v. United States, 232 F. 536, 545 (8th Cir. 1916).) The 1906 Act, as amended, did not require government approval before a drug was introduced into the market. (United States v. Hiland, 909 F.2d 1114, 1125 (8th Cir. 1990).) This changed with the adoption by Congress of the federal Food, Drug, and Cosmetic Act ("FDCA") of 1938 which required a FDA approved new drug application ("NDA") to demonstrate a drug was safe before entering the market. (Samuels at 545.) No new approvals were required for drugs marketed under the 1906 Act only if their conditions of use remained unchanged. (*Id*.)

b. In 1952, after Congress defined prescription drugs, the FDA announced it would not enforce the FDCA for fluoridated public water

The Durham-Humphrey Amendment of 1951 (65 Stat. 648) for the first time explicitly defined two classes of medications (prescription and over-the-counter ("OTC")). (Christopher v. SmithKline Beecham Corp., 635 F.3d 383, 385 (9th Cir. 2011).) In 1952, in response to this amendment, the FDA adopted a regulation stating:

> (a) The program for fluoridation of public water supplies recommended by the Federal Security Agency, through the Public Health Service, contemplates the controlled addition of fluorine at a level optimum for the prevention of dental caries.
> (b) Public water supplies do not ordinarily come under the provisions of the Federal Food, Drug, and Cosmetic Act. . . .
> (c) The Federal Security Agency will regard water supplies containing fluorine, within the limitations recommended by the Public Health Service, as not actionable under the Federal Food, Drug, and Cosmetic Act.

(Former 21 CFR 3.27 (1952); 17 FR 6732; *infra* at B 23.) This regulation was recodified to former 21 CFR 250.203 in 1975. (40 FR 13996; *infra* at B 24.) It was published, as amended, in 1995. (*Infra* at B 25-26.)

c. In 1996 the FDA reversed its position to not enforce the FDCA regarding fluoridated water after the EPA/FDA MOU was terminated and after Congress adopted the DSHEA that defined minerals as drugs if used to prevent specific diseases

In 1996, the FDA determined that its 1952 regulation was obsolete or no longer necessary and the regulation was revoked. (61 FR 29476; *infra* at B 27.) The revocation of former 21 CFR 250.203 occurred after the federal Environmental Protection Agency ("EPA") announced the "Termination of the Federal Drinking Water Additive Program" effective April 7, 1990. (53 FR 25586-89; CP 142-45; *infra* at B 28-31.) The first and major Term of Agreement

of a 1979 Memorandum of Understanding ("MOU") between FDA and EPA was having EPA develop and operate the federal regulatory drinking water additives program:

> III. Terms of Agreement
>
> A. EPA's responsibilities are as follows:
>
> 1. To establish appropriate regulations, and to take appropriate measures, under the SDWA and/or TSCA, and FIFRA, to control direct additives to drinking water (which encompass any substances purposely added to the water)

(44 FR 42775-78; *infra* at B 33 and at B 38.) Arguably, EPA's Federal Register announcement of termination of its regulatory Federal Drinking Water Additives Program was effective notice to FDA that EPA was terminating the 1979 MOU and EPA was no longer obligated by this MOU to establish and operate a federal regulatory program to control direct additives to drinking water. (44 FR 42776, *infra* at B 33 and B 39 ("This [MOU] shall continue in effect unless . . . terminated by either party upon thirty (30) days advance written notice to the other.”))

The revocation of former 21 CFR 250.203 also occurred after the adoption by Congress of the Dietary Supplement Health and Education Act of 1994 (Pub. L. 103-417; "DSHEA"). This 1994 Act of Congress clarified Congressional intent that mineral additives [including fluoride] are drugs if the intended use is to prevent disease:

> A dietary supplement is deemed to be "food," [21 USC] 321(ff), which is defined in part as "articles used for food or drink for man or other animals," *Id*. § 321(f)(1), **except** when it meets the definition of a "drug," which is defined in part as "articles intended for use in the diagnosis, cure, mitigation, treatment, or prevention of disease in man or other animals."

(Alliance for Natural Health U.S. v. Sebelius, 714 F.Supp.2d 48, 50 (D.D.C. 2010) (emphasis supplied.)) Under the DSHEA, dietary supplements include minerals. (21 USC 321(ff)(1)(B);

infra at B 42.) **In adopting the DSHEA in 1994, Congress clarified its intent that fluoride minerals when used to prevent disease are drugs under federal law.** (21 USC 321(ff)(postscript), *infra* at B 43.) In 2000, the FDA Commissioner concurs.[1] (*Infra* at B 44.)

d. The 1962 Amendments to the 1938 Act

The Congress amended the FDCA in 1962 to change the standard for approval of a NDA or abbreviated NDA ("ANDA") from "safe" to "safe and effective" for the intended use. (Samuels at 545.) For drugs with approved NDAs under the 1938 Act to retain these NDAs, they were required to demonstrate they were effective. (*Id*.; Weinberger v. Hynson, Wescott & Dunning, Inc, 412 U.S. 609, 612-15, 93 S.Ct. 2469, 37 L.Ed.2d 207 (1973).)

e. In 1972, the FDA established a new approval process for non-prescription drugs

In 1972, the FDA established a new approval process for non-prescription drugs. (21 CFR Part 330.) This process resulted in the establishment of over-the-counter ("OTC") monographs for various drug classifications including a monograph for anticaries drug products that do not require a prescription. (21 CFR Part 355.) The final rule for the anticaries drug monograph is in 60 FR 52473-510. Amendments to this final rule are in 60 FR 57927, 61 FR 52285-87, 64 FR 13296, and 68 FR 24879-80. This final rule, as amended, provides that all OTC anticaries drug products introduced to the market after April 7, 1997 must comply with general conditions in 21 CFR 330.1 and with anticaries monograph conditions in 21 CFR Part

[1] Congress specifically asked FDA to address the relationship of "fluoride in drinking water and drug(s)." (*Infra* at B 44.) The FDA responded, in part, stating "the Environmental Protection Agency regulates fluoride in the water supply." (*Id*.) But EPA had terminated its water additive program more than ten years earlier. (*Supra* at B 2-3.) So FDA was referring to EPA regulating the Maximum Contaminant Level ("MCL") for fluoride that triggers clean-up under the SDWA and was not referring to regulation of fluoride additives for health care purposes.

355; otherwise a NDA or ANDA is required.

> On or after [April 7, 1997] no OTC drug product that is subject to the monograph and that contains a nonmonograph condition . . . may be initially introduced . . . into interstate commerce unless it is the subject of an approved application or abbreviated application.

(60 FR 52474; 61 FR 52285.) Also, it should be noted that FDA regulations provide that any anticaries drug that includes hydrogen fluoride requires a NDA. (21 CFR 310.545(a)(2) and (b).) Typical specification sheets for water treatment certified Fluorosilicic Acid show a significant portion of the fluoride comes from hydrogen fluoride. (*Infra* at B 47.) Some of the fluoride in water treatment certified Sodium Fluoride also comes from hydrogen fluoride. (*Infra* at B 50.)

2. <u>All drinking waters are drugs when fluoridation chemicals are added with intent to prevent, mitigate and/or prophylactically treat tooth decay disease</u>

a. The FDCA explicitly makes articles drugs when intended for use in the treatment, mitigation and/or prevention of disease

> The term "drug" means
> (A) articles recognized in the official United States Pharmacopoeia . . .; and
> (B) **articles intended for use in the** diagnosis, cure, **mitigation, treatment, or prevention of disease in man** or other animals; and
> (C) articles (other than food) intended to affect the structure of any function of the body of man or other animals; and
> (D) articles intended for use as a component of any article specified in clause (A), (B), or (C). . . .

(21 USC 321(g)(1); *infra* at B 41; emphasis supplied.) The language quoted has not been amended since it was originally adopted in the 1938 Act. (52 Stat. 1041.)

b. Fluoridated drinking waters (bottled or tap (from public water systems)), and fluoridation chemical additives (whether or not certified under NSF/ANSI Standard 60) are drugs under 21 USC 321(g)(1) when the intended use is to aid in the prevention, mitigation and/or prophylactic treatment of dental caries disease (tooth decay, cavities)

Based on 21 USC 321(g)(1)(B) when fluoridated drinking water is intended to aid in the prevention, mitigation and/or prophylactic treatment of dental caries disease (tooth decay, cavities) it is a drug under the FDCA. **There is nothing in the FDCA that would suggest otherwise and HHS and FDA have not made the claim that there is.** Similarly, based on 21 USC 321(g)(1)(B) fluoridation chemical additives that are intended to aid in the prevention, mitigation and/or prophylactic treatment of dental caries disease are drugs under the FDCA. When fluoridation chemical additives are intended for use as a component of fluoridated drinking water, then these fluoridation chemical additives are also drugs under 21 USC 321(g)(1)(D). There is no provision in the FDCA that would cause either fluoridated tap water or fluoridated bottled water to not be considered a drug when the intended use is to aid in the prevention, mitigation and/or prophylactic treatment of dental caries disease (tooth decay, cavities).

c. It should be presumed that the intended use of fluoridation chemical additives and fluoridated waters (bottled or tap) using such additives is to aid in the prevention, mitigation and/or prophylactic treatment of dental caries disease (tooth decay, cavities)

Today, in almost every state, water fluoridation chemical additives are required to be certified to ANSI/NSF Standard 60. For example, in Washington State:

> Any treatment chemicals, with the exception of commercially retailed hypochlorite compounds such as unscented Clorox, Purex, etc., added to water intended for potable use must comply with ANSI/NSF Standard 60. The maximum application dosage recommendation for the product certified by the ANSI/NSF Standard 60 shall not be exceeded in practice.

WAC 246-290-220(3). NSF, an author of ANSI/NSF Standard 60, states in its 2008 NSF Fact Sheet on Fluoridation Chemicals:

> Water fluoridation . . . . Fluoride is added to water for the public health benefit of preventing and reducing tooth decay

(*Infra* at B 73.) In 2011, HHS confirmed its belief that:

> Community water fluoridation is the most cost-effective method of delivering fluoride for the prevention of tooth decay.

(76 FR 2386; *infra* at B 51.)

The FDA has concluded that the intended use is implied for fluoride additives to prevent tooth decay. The FDA finds that intended use "may be shown by the circumstances surrounding the distribution of the article." (21 CFR 801.4.) The FDA states:

> in some instances, the mere presence of certain therapeutically active ingredients could make a product a drug even in the absence of drug claims. In these cases, the intended use would be implied because of the known or recognized drug effects of the ingredient (e.g. fluoride in a dentifrice).

(59 FR 6088.) The intended use of added fluoride in drinking water is also implied and should be presumed. The FDA's interpretation of "intent" is entitled to "considerable deference." (Young v. Community Nutrition Institute, 476 U.S. 974, 981, 106 S.Ct. 2360, 90 L.Ed.2d 959 (1986).) The Washington State Board of Health states,

> The Board considers it self-evident that the purpose of water fluoridation is to help prevent tooth decay.

(*Infra* at B 52.)

The CDC states, "Tooth decay (dental caries) is an infectious, multifactorial disease." (Infra at B 54.) The FDA defines "dental caries" as "A disease of calcified tissues of teeth characterized by demineralization of the inorganic portion and destruction of the organic matrix" and defines "anticaries drug" as "A drug that aids in the prevention and prophylactic treatment of

dental cavities (decay, caries). (21 CFR 355.3(c) and (d).)

d. The language in 21 USC 321(g)(1)(B) defining drugs must be interpreted "as broad as its literal language indicates"

As early as 1916, the federal Supreme Court concurred that products that were otherwise defined as "foods" would be "drugs" under the federal statute[2] when labeling for the substance includes statements of therapeutic (including preventative) effect. (Seven Cases v. United States, 239 U.S. 510, 513-14, 36 S.Ct. 190, 60 L.Ed. 411 (1916).)

After the 1938 Act was adopted, the federal Supreme Court again concurred that "food products" will be "drugs" based on intended use and "labeling." (Kordel v. United States, 335 U.S. 345, 346, 69 S.Ct. 106, 93 L.Ed. 52 (1948).) In 1969, the federal Supreme Court, in finding a product was a drug, explained:

> Congress intended to define "drug" [in 21 USC 321(g)(1)(B)] far more broadly than does the medical profession. . . . The word "drug" is a term of art for the purposes of the Act, encompassing far more than the strict medical definition of that word.

(United States v. An Article of Drug . . . Bacto-Unidisk, 394 U.S. 784, 793, 89 S.Ct. 1410, 22 L.Ed.2d 726 (1969).) The Bacto-Unidisk Court continued:

> Congress fully intended that the Act's coverage be **as broad as its literal language indicates** - and, equally clear, broader than any strict medical definition might otherwise allow. . . . **the Food, Drug, and Cosmetic Act is to be given a liberal construction consistent with the Act's overriding purpose to protect the public health.**

(*Id*. at 798; emphasis supplied.) The Bacto-Unidisk Court finally directed,

> we must take care not to narrow the coverage of a statute short of the point where Congress indicated it should extend.

---

[2] The relevant portion of the federal statute are quoted *supra* at B 1.

(*Id*. at 801.)

In the construction of federal statutes, "the decisions of the Supreme Court of the United States are binding" upon all. (Beezer v. City of Seattle, 62 Wn.2d 569, 573, 383 P.2d 895 (1963).) Therefore, HHS and FDA and every court is required to construe the definition of drug as "articles intended for use in the . . . prevention of disease" as "broad as its literal language indicates." (*Supra*.)

e. Foods must be regulated as drugs if the "intended use" is to prevent disease

Interpretation of federal statutes by other federal courts are entitled to great weight. (Beezer at 573.) A long line of federal court cases has found that articles normally regulated as "foods" will be regulated as "drugs" if the intended use is to treat or prevent a disease:

> The word "drug" is defined in 21 U.S.C. s 321(g)(1)(B) to include:
>
> articles intended for use in the diagnosis, cure, mitigation, treatment, or prevention of disease in man or other animals . . .
>
> Thus, it is the intended use of an article which determines whether or not it is a "drug," and even the most commonly ingested foods and liquids are "drugs" within the meaning of the [FDCA] if their intended use falls within the definition of s 321(g)(1)(B).

Gadler v. United States, 425 F.Supp. 244, 246-47 (D.Minn. 1977); *see* Nutrilab, Inc. v. Schweiker, 713 F.2d 335, 336 (7th Cir. 1983); *see also* Bradley v. United States, 264 F.79 (5th Cir., 1920) where the court specifically found "mineral water" to be a "drug" when it is intended to treat disease.

In the determination of whether fluoridation products (fluoridated waters (tap or bottled) and fluoridation chemical additives) are drugs,

> the only question under the [FDCA] is whether the intended use of the product is to prevent disease, not whether the product actually prevents disease.

(United States v. Bowen, 172 F.3d 682, 686 (9th Cir. 1999).) Intent "may be derived or inferred from [any] relevant source." (National Nutritional Foods Ass'n v. Mathews, 557 F.2d 325, 334 (2nd Cir. 1977).) As discussed previously, it should be presumed that the "intended use" of fluoridation products is to prevent dental caries (tooth decay) disease. (*Supra* at B 6-8.)

f. The DSHEA further clarifies the intent of Congress that fluorides, which are minerals, that are added to drinking water to prevent the disease of dental caries, are drugs

Perhaps partly in response to the FDA's refusal to enforce the FDCA for fluoridated water supplies (*supra* at B 2), Congress adopted the DSHEA in 1994, with explicit statutory language that made fluoride a drug when used with intent to prevent disease. Fluoride, being a mineral, is a dietary supplement under DSHEA. (21 USC 321(ff)(1)(B); *infra* at B 42.) Minerals are normally regulated as foods except when they are drugs. (21 USC 321(ff)(postscript) ("**except** for purposes of [21 USC 321(g)(1) defining drugs] a dietary supplement shall be deemed to be a food;") *infra* at B 43 (emphasis supplied).)

g. Congress did not intend to exempt public water or water additives from the reach of federal drug laws

In 1974, Congress passed the Safe Drinking Water Act ("SDWA"). (88 Stat. 1661; codified at 42 USC 300f et seq.) The SDWA empowered the EPA to set standards for the control of contaminants in drinking water. (42 USC 300g-1(b); *see* In re Groundwater Cases, 154 Cal.App.4th 659, 677 (2007).) The SDWA authorizes EPA to adopt national primary drinking water regulations applicable to "public water systems." (42 USC 300f(1); *see* 42 USC

300f(4)(A).) Under the SDWA, national primary drinking water regulations identify contaminants that have adverse effects on human health and specify a maximum contaminant level ("MCL") for such contaminants. (42 USC 300f(1).) Pursuant to its authority under the SDWA, the EPA has since established MCLs for a wide variety of contaminants. (*See* 40 CFR Pt. 141 for substantive regulations, Pt. 142 for implementation regulations, and Pt. 143 for national secondary drinking water regulations that are not enforceable.) The fluoride MCL is 4.0 mg/l (four milligrams per liter which is 4 parts per million (ppm)). (40 CFR 141.62(b)(1).)

But there is no SDWA statutory provision or implementing regulation that addresses or sets standards for fluoridation chemical additives.[3] (SDWA; 40 CFR Part 141 et seq.) Therefore, there is no possible statutory conflict where Congress intended the SDWA to interfere with the FDCA or FDA authority to regulate drugs. If Congress wanted to exempt public drinking water from the definition of drugs in 21 USC 321(g)(1)(B) it certainly had the knowledge of how to do it (it had previously exempted "food" from subsection (1)(C)) and it certainly had the opportunity to do it in any one of the more than 20 significant amendments made to the FDCA since 1980. (*Infra* at B 56-57.) The SDWA did not explicitly or implicitly repeal any drug provision of the FDCA or any drug authority of the FDA.

h. Arguably, the 1979 EPA/FDA MOU has been terminated but never did restrict FDA authority over drugs

i. <u>The 1979 MOU</u>

In 1979, EPA and FDA entered into an MOU where FDA agreed not to enforce its food

[3] There is a SDWA statutory provision that directs the EPA to keep away from regulating drugs. (42 USC 300g-1(b)(11) ("No national primary drinking water regulation may require the addition of any substance for preventive health care purposes unrelated to contamination of drinking water."))

authority over public drinking water in exchange for EPA creating a federal regulatory drinking water additives program. (*Infra* at B 32-39.) In the FDCA, Congress gave FDA authority to regulate foods to ensure they are "safe" (21 USC 393(b)(2)(A)) and drugs to ensure they are "safe and effective" (21 USC 393(b)(2)(B)). Normally for drinking water, only food regulations would be applicable and prior to 1979, the FDA generally regulated drinking water as a food. (*Infra* at B 32 and B 37.) But after passage of the SDWA, EPA and FDA were concerned that FDA's "food" authority and EPA's "public drinking water" authority might result in "duplicative and inconsistent regulations" so they entered an MOU. (*Supra* at B 2-3, *Infra* at B 32.) In the MOU, FDA agreed not to use its "food" authority to regulate public drinking water, based on a commitment that EPA would adopt federal regulations to control additives in public drinking water. (*Supra* at B 2-3, *Infra* at B 32-33.)

There is no mention in the MOU that FDA would, or could, give up its "drug" authority over public drinking water and public drinking water additives. (Infra at B 32-39.) Congress required "drugs" to be "effective" (21 USC 393(b)(2)(B)) and Congress never gave EPA authority to regulate drug effectiveness. The MOU inartfully states:

> [EPA and FDA] have determined that the passage of the SDWA in 1974 implicitly repealed FDA's authority under the [FDCA] over water used for drinking water purposes.

(*Infra* at B 32.) Read in context with the other provisions of the MOU this can only possibly be true with respect to FDA's "food" authority and cannot be true with respect to FDA's "drug" authority. (*Infra* at B 32-34; *See* Board of Governors of the Federal Reserve System, 474 U.S. 361, 368, 106 S.Ct. 681, 88 L.Ed.2d 691 (1986) ("agency interpretation" cannot "alter the clearly expressed intent of Congress."))

In a subsequent section, the MOU states:

> [EPA and FDA] agreed that the Safe Drinking Water Act's passage in 1974 implicitly repealed FDA's jurisdiction over **drinking water as a "food"** under the [FDCA].

(*Infra* at B 33; emphasis supplied.) Thus the MOU itself clarifies that the MOU only was intended to address FDA's regulations regarding "food." The MOU also inartfully states:

> Under the agreement, EPA now retains exclusive jurisdiction over drinking water served by public water supplies, including any additives in such water.

(*Infra* at B 33.) In context of the whole agreement, EPA does not have exclusive jurisdiction when public drinking waters, and public drinking water additives, are "drugs" because Congress has given exclusive jurisdiction over drugs to the FDA. (21 USC 393(b)(2)(B); FDA v. Brown & Williamson Tobacco Corp., 529 U.S. 120, 126, 120 S.Ct. 1291, 146 L.Ed.2d 121 (2000).) Congress has clearly defined "drugs" in 21 USC 321(g)(1). Further EPA claims no authority that would give it jurisdiction over the determination of "effectiveness" of drugs. (*Infra* at B 32-35.)

ii. Arguably, the 1979 MOU is terminated

In 1988, EPA published in the Federal Register a "Notice"that it was terminating EPA's commitment to FDA to create a federal regulatory drinking water additives program. (53 FR 25586-89; *infra* at B 28-31.) In this 1988 Notice, EPA admits that it "does not currently regulate the levels of additives in drinking water." (*Infra* at B 28.) EPA explained that the "SDWA does not require EPA to control the use of specific additives in drinking water." (*Infra* at B 28.) It states,

> Resource constraints and the need to implement mandatory provisions of the SDWA precluded the Agency from implementing the comprehensive program originally

envisioned . . .

(*Infra* at B 29.) The Notice describes how EPA was cooperating with a private third-party organization to have that organization take over the development and monitoring of standards for public drinking water additives and explained that it would be "up to the States and utilities to determine the suitability of any 'third-party' certification." (*Infra* at B 28-30.) Then it announced that effective April 7, 1990, it would withdraw all EPA and predecessor agency lists of acceptable water additive products and all EPA and predecessor agency advisory opinions on drinking water additives. (*Infra* at B 31.) EPA stated that "Discontinuance of the additives program at EPA does not relieve the Agency of its statutory responsibilities." (*Infra* at B 31.)

Arguably, EPA's Federal Register published Notice that it was terminating its commitment to FDA to create a regulatory federal drinking water additives program was effective notice to FDA that EPA was exercising its option to terminate the MOU. *(Supra* at B 2-3.) Thus, arguably, the 1979 MOU was terminated by 1990 and EPA removed the cloud over FDA's "food" jurisdiction regarding public fluoridated water. FDA never lost "drug" jurisdiction over fluoridated water, but its policy, that it would not enforce this jurisdiction, remained in effect from 1952 to 1996. (*Supra* at B 2-3.)

i. The intent of Congress clearly establishes that water fluoridation products are drugs under the FDCA

In 1916, the federal Supreme Court concurred that Congress in adopting the 1906 Act directed that food be regulated as a drug when therapeutic (including preventative) effects are intended. (*Supra* at B 8.) In the 1938 Act, Congress significantly broadened, instead of limited, the definition of drugs. (*Compare supra* at B 1 and B 5.) In 1948, the federal Supreme Court

again concurred "food products" will be "drugs" depending on intent and "labeling." (*Supra* at B 8.)

In 1952, the FDA stated it would not enforce the FDCA for fluoride added to public water supplies. (*Supra* at B 2.) In 1969, the federal Supreme Court ruled that the FDCA definition of drugs is "as broad as its literal language indicates." (*Supra* at B 8-9.) In 1994, the Congress again specifically clarified that minerals will be drugs if they fall within the broad definition of drugs. (*Supra* at B 3-4 and 10.) In 1996, the FDA revoked its policy that it would not enforce the FDCA for fluoride added to public water supplies. (*Supra* at B 2.)

Every department and agency and court is bound by the intent of Congress as explained by the federal Supreme Court. (*Supra* at B 9.) Therefore, the FDA should find that water fluoridation products (fluoridated waters (tap or bottled) and fluoridation chemical additives) are drugs under federal law and regulation when the intended use is to aid in the prevention, mitigation, and/or prophylactic treatment of dental caries disease (tooth decay, cavities). And based on the history of fluoridation, it should be presumed that this is the intended use of water fluoridation products. (*Supra* at B 9-10.)

3. HHS, acting through the FDA, is responsible for regulating the addition of fluoride to public drinking water

_____ Despite the Federal Supreme Court ruling in Bacto-Unidisk (*supra* at B 8-9), HHS and FDA appear to now argue that certain fluoridation products (fluoridated public waters and fluoridation chemical additives) are not drugs. It is uncontested by HHS and FDA that these fluoridation products are articles intended to prevent dental caries disease in man. (*Supra* at B 6-8.) Under Bacto-Unidisk and other federal court rulings (*supra* at B 7 to B 9), these fluoridation

products are therefore within the definition of a "drug" in 21 USC 321(g)(1)(B).

However, HHS and FDA interpret the Safe Drinking Water Act of 1974 (SDWA) as removing HHS and FDA jurisdiction over these fluoridation products:

> *Congress did not intend for FDA to regulate the addition of fluoride to public drinking water for dental caries prevention as a drug under the FD&C Act. Instead, Congress intended that the U.S. Environmental Protection Agency (EPA) regulate fluoride in public drinking water as a potential contaminant under the Safe Drinking Water Act of 1974 (SDWA).*

(*Infra* at B 59 and B 66: B 58 to B 62 is a November 21, 2014 letter from HHS Principal Deputy Assistant Secretary for Health Dr. Wanda Jones to Ms. McEtheney; B 63 to B 68 is a December 23, 2013 Request for Review to Jill Warner, FDA Associate Commissioner for Special Medical Programs from Gerald Steel (FDA has not yet responded to this Request for Review).)

HHS and FDA argue that the SDWA provides:

> that within the limits thus set by EPA, state and local governments be permitted, but not required, to fluoridate public drinking water to help prevent dental caries.

(*Infra* at B 59 and B 66.) Thus, HHS and FDA argue that under their interpretation of the SDWA, FDA has no responsibility to regulate such fluoridation products that are articles that meet the definition of a drug in 21 USC 321(g)(1)(B).

The fundamental problem with this HHS and FDA interpretation of the SDWA is that it is in conflict with the EPA interpretation of the SDWA. The SDWA gives administrative authority to the EPA. (42 USC 300f(7 and 8).) Along with administrative authority comes the sole agency power to interpret the Act. Chevron USA v. NRDC, 467 U.S. 837, 842-45, 104 S.Ct. 2778, 81 L.Ed.2d 694 (1984).

Steven M. Neugeboren is the Associate General Counsel in charge of the Water Law Office of the EPA. The Water Law Office is responsible for interpreting the SDWA.[4] Mr. Neugeboren states:

> Under the Safe Drinking Water Act (SDWA), EPA is the lead federal agency with responsibility to regulate the safety of public water supplies. EPA does not have responsibility for substances added to water solely for preventative health care purposes, such as fluoride, other than [to meet maximum contaminant limits.] The Department of Health and Human Services (HHS), acting through the FDA, remains responsible for regulating the addition of drugs to water supplies for health care purposes.

(*Infra* at 69-70 - February 14, 2013 letter written on behalf of EPA Administrator Lisa Jackson to Gerald Steel.) Therefore the EPA's interpretation of the SDWA is that this Act does not affect the responsibility of the FDA "for regulating the addition of drugs to water supplies for health care purposes." Therefore HHS and FDA misinterpret Congressional intent when they state:

> Congress did not intend for FDA to regulate the addition of fluoride to public drinking water for dental caries prevention as a drug under the FD&C Act.

(Infra at B 59 and B 66.)

HHS and FDA are correct that the SDWA does give EPA lead responsibility for regulating the safety of public water supplies to protect against adverse health effects. Except for authorizing regulation of the maximum contaminant level for fluorides, the SDWA does not address state and local governments fluoridating public drinking water to help prevent dental caries. But the state and local governments which fluoridate must comply with all applicable laws and regulations including federal drug laws in the FDCA, state drug and fluoridation laws,

[4] http://www2.epa.gov/aboutepa/about-office-general-counsel-ogc#water

federal drug regulations, and state drug and fluoridation regulations. The EPA has determined that state fluoridation regulations are not related to the SDWA. (*Infra* at B 71-72 - November 17, 2011 letter written on behalf of EPA Region 10 Administrator to Gerald Steel.)

Under this analysis and the interpretations of the SDWA by the EPA: HHS and FDA should find that fluoridation products are drugs when they meet the definition of a drug in 21 USC 321(g)(1)(B). HHS, acting through the FDA, has responsibility to regulate these drugs to ensure that they are safe and effective.

4. FDA should request registration of all water fluoridation products as drugs pursuant to 21 CFR Part 207

It is requested that FDA request registration of all water fluoridation products as drugs pursuant to 21 CFR Part 207. In most states, lists of public water purveyors making fluoridated waters are available from State Health Departments. In most states, fluoridation chemical additives must be certified to meet ANSI/NSF Standard 60. (*Supra* at B 6.) There are only three organizations that certify products to ANSI/NSF Standard 60 and their web addresses are www.nsf.org/, www.ul.com/eph/, and www.wqa.org/. These organizations can be contacted to get current lists of ANSI/NSF Standard 60 certified fluoridation chemical additive products and manufacturers.

To facilitate determination of the legal drug status of these fluoridation products, it is requested that FDA request for each fluoridation product, for each year the product was marketed or proposed for future use, a copy of all certificates of analysis and product labeling (both on any packaging and from any other documents (electronic, print, or otherwise) describing the product or describing the purpose of using fluoride additives, or describing the conditions of use that are

recommended or suggested.) For water purveyors, the documents describing the purpose of fluoride additives, likely would include documents associated with the decision to begin fluoridation and documents, including materials sent to customers, that later describe on-going reasons for fluoridation. Because certification to ANSI/NSF Standard 60 began around 1990, it is expected that fluoridation chemical additive labeling was changed around that time to declare certification. It is likely that all fluoridation product manufactures will be required to get approved new drug applications or approved abbreviated new drug applications.

5. FDA should find that fluoridation products are not "safe and effective"

Once it accepts jurisdiction, FDA should find that fluoridation products are not safe and effective as drugs. While this is a subject that will only be addressed after HHS and FDA accept drug jurisdiction over fluoridation products, it is useful to point out the harms that HHS and FDA are allowing to occur because they have not accepted drug jurisdiction over fluoridation products.

An important overview was provided in the York Review in 2000 (M. McDonagh, P. Whiting, M. Bradley, et al., "A Systematic Review of Water Fluoridation," NHS Centre for Reviews and Dissemination, The University of York, Report 18 (2000) which is available at: (http://www.york.ac.uk/inst/crd/CRD_Reports/crdreport18.pdf). The potential harms explored by the York Review include dental fluorosis, hip fracture, other bone fractures, cancer, Down's syndrome, mortality, senile dementia, goitre, lowered IQ, hypersensitivity, and skeletal fluorosis. (York Review at 52, 54, 59-60.) The York Review concludes that except for dental fluorosis, no "confident statements" can be made regarding these "potential harms." (York Review at page xiv.) In other words, these other "potential harms" could not be ruled out by the available scientific literature.

a. Dental fluorosis is an out-of-control harm of water fluoridation

There is scientific consensus that fluoridated water causes dental fluorosis. HHS reported that 41% of people who were 12 to 15 years old in 1999 to 2004 had dental fluorosis with this dental fluorosis being moderate or severe for 3.6% of these people (one in twenty eight people). (76 FR 2385.) Even if water fluoridation is reduced to 0.7 mg/l fluoride as HHS now recommends, the number of people with dental fluorosis is likely to increase because in 1992 when these people were 0 to 8 years old, only 56% of the people in the United States received fluoridated water. Today a much higher percentage of people receive fluoridated water.

b. The FDA has already concluded that fluoride OTC products should not be swallowed except under professional supervision

The FDA has already concluded that fluoride OTC anti-cavity products should not be swallowed except under professional supervision. (21 CFR Part 355.) Fluoridation chemical additives are intended to be mixed with water and swallowed by everyone. At a minimum, fluoridated water is harmful to infants and children under 6. Warnings are required for OTC products to avoid swallowing by infants and even children under six. (21 CFR 355.50.) Bottled water regulations do not even allow a health claim for fluoridated water marketed to infants. (www.fda.gov/food/ingredientspackaginglabeling/labelingnutrition/ucm073602.htm)

c. York Review studies repeatedly show that artificial water fluoridation increases risk of hip fracture in people 65+ years old

The York Review was limited to review of human epidemiological studies of water fluoridation (around 1 mg/l fluoride). Over 3,200 primary studies were identified but only 9 studies met relevance criteria and measured risk of hip fracture for people 65+ years old in fluoridated areas compared to the risk in unfluoridated areas. (York Review at 10 and 48.) For

these 9 studies, there were only 4 analyses that produced statistically significant data (i.e. the relative risk of 1.0 was not in the 95% Confidence Interval). Each of these statistically significant analyses show an increased risk of hip fracture for people 65+ years old living in fluoridated areas. The studies are identified in the York Review at page 48 as:

Author (Year)	Sex	Relative Risk	95% Confidence Interval
Jacqmin-Gadda (1998)	Both	2.43	(1.1, 5.3)
Danielson (1992)	Women	1.27	(1.1, 1.5)
Jacobsen (1992)	Women	1.08	(1.06, 1.10)
Jacobsen (1992)	Men	1.17	(1.13, 1.22)

Relative Risk is defined as the risk of an adverse effect with exposure to a treatment (here fluoridated water) relative to risks for those who do not receive the treatment. (York Review at 99.) A ratio of 1.0 indicates no increased risk over receiving no treatment. (*Id*.) A ratio greater than 1.0 indicates the risk is higher in the group that did receive the treatment. (*Id*.) A ratio less than 1.0 indicates the risk of the adverse effect is higher in the group that did not receive treatment. (*Id*.) A Relative Risk of 1.27 means that there is a 27% higher risk of hip fractures when living in a fluoridated area (for 65+ year old women in the Danielson (1992) analysis).

Hip fracture for people 65+ years old is a significant health impact in the United States. "About 300,000 Americans are hospitalized for a hip fracture every year." (Connett (2010) at page 173.) The Irish Forum (2002) (Forum on Fluoridation (Dublin, Ireland: Stationery Office, 2002) online at http://fluoridealert.org/re/fluoridation.forum.2002.pdf found that "Fracture of the hip is a major cause of morbidity and mortality [disease and death] in persons 65 years of age and older."

> Aside from the fact that one in five patients die within 6 months of the fracture occurring, hip fractures lead to serious disability. Many basic functions such as dressing, climbing stairs, walking and transferring are markedly interfered with following a fracture. This can result in loss of both confidence and independence and an increased risk of development of medical complications.

(Irish Forum (2002) at 121.)

d. Fifty human studies agree that higher fluoride exposure is associated with a mental health impact that lowers IQ levels in children

Lowered IQ in persons who drink fluoridated water as infants and children is a significant mental health concern. The National Research Council (2006) states, "It is apparent that fluorides have the ability to interfere with the functions of the brain." (NRC, Fluoride in Drinking Water - A Scientific Review of EPA's Standards (Washington D.C.; The National Academies Press, 2006.) As of September, 2016, 50 of 57 human studies found elevated fluoride exposure is associated with reduced IQ and 45 animal studies have found fluoride exposure impairs the learning and/or memory capacity of animals. (http://fluoridealert.org/studies/brain01/)

> The lowest level at which IQ has been lowered (with borderline iodine deficiency) was at 0.88 ppm [fluoride in drinking water] (Lin et al., 1991) or at 1.26 ppm (without iodine as a complicating factor). It is very clear that there is no margin of safety to protect all children drinking water in the range 0.7 to 1.2 ppm.

Dec. 12, 2014 email from Paul Connett, PhD., then Director, Fluoride Action Network.

e. Drinking fluoridated water increases risk of hypothyroidism disorder

A large observational study was published in the online Journal Of Epidemiology and Community Health, a British Medical Journal (BMJ) publication, on February 24, 2015 that found rates of diagnosed hypothyroidism (underactive thyroid) were at least 30% higher in areas with artificial fluoridation. (Peckham (2015) -J Epidemiol Community Health doi:10.1136/jech-2014-204971.) The study states that thyroid dysfunction is a common endocrine disorder. The National

Research Council ((2006) at 223 called fluoride an endocrine disrupter and at 218 expresses concern about "the inverse correlation between asymptomatic hypothyroidism in pregnant mothers and the IQ of the offspring."

f. Boys drinking fluoridated water when they are 6 to 8 years old have a five to seven-fold greater risk of contracting bone cancer by the age of twenty

Regarding cancer, an unrefuted published primary study, Bassin (2006) (Bassin E. B. et al., "Age-specific Fluoride Exposure in Drinking Water and Osteosarcoma (United States)," Cancer Causes and Control 17, no. 4 (May 2006) 421-28) reports that boys who drink fluoridated water when they are 6 to 8 years old will have a five- to sevenfold greater risk of contracting osteosarcoma (bone cancer) by the age of twenty. This is a deadly disease. This result was first suggested by Perry Cohn in 1992. (*See* Connett (2010) at pages 187-94.) The twofold increase in cortical bone defects in the fluoridated city in the Kingston-Newburgh study (*supra* at B 20.) was described in 1955 and again in 1977 as being "strikingly similar to that of osteogenic sarcoma [now called osteosarcoma]." (*See* Connett (2010) at page 181-94.)

6. <u>FDA has correctly determined that fluoridated bottled water is a drug when there is a claim that "this drinking water is intended for use in the prevention of tooth decay disease"</u>

In a September 23, 2015 letter (B 74-75 hereto), the FDA found that fluoridated bottled water with 0.7 mg/l fluoride would be a drug if the claim is made that "this drinking water is intended for use in the prevention of tooth decay disease." In fact, fluoridated bottled water with this claim would be an "anticaries drug" as that term is defined by the FDA in 21 CFR 355.3(c) and (d). (*Supra* at B 7-8.) Such fluoridated bottled waters when introduced after April 7, 1997 would be required to have an approved New Drug Application (NDA) or Abbreviated NDA

(ANDA) because they would not be able to meet requirements of 21 CFR Part 355 which do not allow anticaries drugs to be swallowed without professional supervision. (*See supra* at B 4-5.) Under current law, it would be illegal to distribute such fluoridated bottled water in interstate commerce without an approved NDA or ANDA. Because such fluoridated bottled waters would be drugs, the fluoridation chemical additives, which are a component of such fluoridated bottled waters, would also be drugs. (21 USC 321(g)(1)(D).)

7. FDA must now find that fluoridated tap water is a drug when there is a claim that "this drinking water is intended for use in the prevention of tooth decay disease"

FDA must now find that fluoridated tap water is a drug when there is a claim that this drinking water is intended for use in the prevention of tooth decay disease. The FDA must also find that the fluoridation chemical additives, which are a component of such fluoridated tap waters, are also drugs. (USC 321(g)(1)(D).) The FDCA allows no distinction between fluoridated waters with the same contents whether they are served as drinking water either from a bottle or from a tap. Both are anticaries drugs under the FDCA if the drinking water is intended for use in the prevention of tooth decay disease. More generally, fluoridated drinking waters are anticaries drugs if the intended use is to aid "in the prevention and prophylactic treatment of dental cavities (decay, caries)." (21 CFR 355(3)(c).)

Today, as fluoridated water purveyors modify their fluoridated waters to meet the latest HHS recommendation to add fluoride to get 0.7 mg/l fluoride in the finished water, these water purveyors are making a new drug and are subject to new drug requirements for an approved NDA or ANDA and subject to the FDA requirements to show that their unique products are safe and effective.

The FDA can no longer rely on its prior reasoning (*Infra* at B 59 and B 66) that the intent of the SDWA was to eliminate FDA authority and responsibility under the FDCA to regulate substances that qualify as anticaries drugs under the USC and CFR. EPA is the agency with final agency authority to interpret the SDWA, and EPA interprets the SDWA to not remove the authority of HHS, acting through the FDA, regarding "regulating the addition of drugs to water supplies for health care purposes." (*Infra* at B 69.)

So while it is true that state and local governments may be permitted to fluoridate drinking waters to help prevent dental caries, they must do so in compliance with local, state, and federal laws and regulations which include federal requirements to consider such fluoridated waters to be drugs if the drinking waters are "intended for use in the prevention of tooth decay disease" or if the drinking waters otherwise meet the definition of drugs in section 201(g)(1) of the FDCA (21 USC 321(g)(1)). FDA, acting on behalf of HHS, has the authority and responsibility to regulate drugs by implementing the applicable federal laws and regulations and by adopting regulations when necessary to fulfill its responsibilities.

It is time for the FDA to be responsible and to require fluoridation products (fluoridated waters (tap or bottled) and fluoridation chemical additives) to be federally regulated as drugs when the intended use is prevention of tooth decay disease.

customs Form 4449 showing the name of the airport, date and time of arrival, date and time of departure and purpose of the visit. The permit shall be surrendered to the collector of customs at the port of final clearance for a foreign destination, who shall satisfy himself prior to the issuance of clearance that the aircraft received proper customs treatment while in this country. The permit shall then be returned to the collector of customs at the port of issue.

(2) A copy of the permit shall be retained by the collector at the port where issued. If within 60 days after the issuance of such permit the said collector does not receive a report of the outward clearance of the aircraft covered thereby, the matter shall be reported to the supervising customs agent for investigation.

(3) Civil aircraft registered in the United States arriving from a foreign country with passengers carried for hire or merchandise, after proper customs treatment of their cargo (passengers carried for hire or merchandise), may be allowed to proceed upon their identity being established.

This order shall become effective on the date of its publication in the FEDERAL REGISTER.

(R. S. 161, sec. 23, 5 Stat. 892, as amended, sec. 24, 43 Stat. 166, R. S. 251, secs. 624, 644, 46 Stat. 759, 761, secs. 201, 367, 58 Stat. 683, 706, sec. 7, 44 Stat. 57, as amended; 5 U. S. C. 22, 8 U. S. C. 102, 222, 19 U. S. C. 66, 1624, 1644, 42 U. S. C. 202, 270, 49 U. S. C. 177)

[SEAL] D. A. STRUBINGER,
Acting Commissioner of Customs.
JOHN S. GRAHAM,
Acting Secretary of the Treasury.
W. P. DEARING,
Acting Surgeon General,
U. S. Public Health Service.
JOHN L. THURSTON,
Acting Federal Security Administrator.
PHILIP B. PERLMAN,
Acting Attorney General.

JULY 17, 1952.

[F. R. Doc. 52-8054; Filed, July 22, 1952; 8:55 a. m.]

[T. D. 53046]

PART 10—ARTICLES CONDITIONALLY FREE, SUBJECT TO A REDUCED RATE, ETC.

SUPPLIES FOR VESSELS OF WAR

The Department of State has furnished the Treasury Department an up-to-date list of countries which permit the withdrawal of supplies free of duty and tax by vessels of war of the United States while in ports of those countries. Therefore, § 10.59 (d), Customs Regulations of 1943 (19 CFR 10.59 (d)), containing a list of countries whose vessels of war shall be accorded the privilege of withdrawing supplies free of customs duties and internal-revenue tax while in ports of the United States, as provided for in section 309 (a), Tariff Act of 1930, as amended, is further amended to read as follows:

§ 10.59 *Exemption from customs duties and internal revenue tax.* * * *

(d) The privilege shall be accorded to vessels of war of the following countries:

Argentina.
Australia.
Belgium.
Brazil.
Canada.
Chile.
Colombia.
Cuba.
Denmark.
The Dominican Republic.
Ethiopia.
Finland.
France.
Great Britain.
Greece.
Haiti.
India.
Ireland.
Mexico.
The Netherlands.
New Zealand.
Nicaragua.
Norway.
Panama.
The Philippines.
El Salvador.
Spain.
Sweden.
Thailand.
Turkey.
Union of South Africa.
Uruguay.
Venezuela.

(Sec. 5, 52 Stat. 1080; 19 U. S. C. 1309)

[SEAL] FRANK DOW,
Commissioner of Customs.

Approved: July 16, 1952.

JOHN S. GRAHAM,
Acting Secretary of the Treasury.

[F. R. Doc. 52-8025; Filed, July 22, 1952; 8:48 a. m.]

TITLE 21—FOOD AND DRUGS

Chapter I—Food and Drug Administration, Federal Security Agency

PART 3—STATEMENTS OF GENERAL POLICY OR INTERPRETATION

FLUORIDATED WATER AND PROCESSED FOODS CONTAINING FLUORIDATED WATER

Pursuant to section 3 of the Administrative Procedure Act (60 Stat. 237, 238; 5 U. S. C. 1002), the following statement of policy is issued:

§ 3.27 *Status of fluoridated water and foods prepared with fluoridated water under the Federal Food, Drug, and Cosmetic Act.* (a) The program for fluoridation of public water supplies recommended by the Federal Security Agency, through the Public Health Service, contemplates the controlled addition of fluorine at a level optimum for the prevention of dental caries.

(b) Public water supplies do not ordinarily come under the provisions of the Federal Food, Drug, and Cosmetic Act. Nevertheless, a substantial number of inquiries have been received concerning the status of such water under the provisions of the act and the status, in interstate commerce, of commercially prepared foods in which fluoridated water has been used.

(c) The Federal Security Agency will regard water supplies containing fluorine, within the limitations recommended by the Public Health Service, as not actionable under the Federal Food, Drug, and Cosmetic Act. Similarly, commercially prepared foods within the jurisdiction of the act, in which a fluoridated water supply has been used in the processing operation, will not be regarded as actionable under the Federal law because of the fluorine content of the water so used, unless the process involves a significant concentration of fluorine from the water. In the latter instance the facts with respect to the particular case will be controlling.

(Sec. 701, 52 Stat. 1055; 21 U. S. C. 371)

Dated: July 17, 1952.

[SEAL] JOHN L. THURSTON,
Acting Administrator.

[F. R. Doc. 52-8041; Filed, July 22, 1952; 8:50 a. m.]

TITLE 26—INTERNAL REVENUE

Chapter I—Bureau of Internal Revenue, Department of the Treasury

Subchapter C—Miscellaneous Excise Taxes

[T. D. 5920; Regs. 132]

PART 325—EXCISE AND SPECIAL TAX ON WAGERING

REGISTRY, RETURN AND PAYMENT OF TAX

Regulations 132 amended to require persons liable for special (occupational) wagering tax to file returns and pay tax before commencing taxable activity and to file supplemental returns advising of all agents or employees engaged to receive wagers or with respect to all persons for whom wagers are received.

On June 3, 1952, notice of proposed rule making regarding amendment of § 325.50 of Regulations 132 was published in the FEDERAL REGISTER (17 F. R. 4988). No objection to the rules proposed having been received, § 325.50 of Regulations 132 is amended to read as follows:

§ 325.50 *Registry, return, and payment of tax.* (a) No person shall engage in the business of accepting wagers subject to the 10 percent excise tax imposed by section 3285 of the Internal Revenue Code (see § 325.24) until he has filed a return on Form 11-C and paid the special tax imposed by section 3290. Likewise, no person shall engage in receiving wagers for or on behalf of any person engaged in such business until he has filed a return on Form 11-C and paid the special tax imposed by section 3290 of the Internal Revenue Code. Filing of successive applications and payment of tax by such persons are required on or before July 1 of each year thereafter during which taxable activity continues. The return, with remittance, shall be filed with the collector of internal revenue for the district in which is located the taxpayer's office or principal place of business. If such taxpayer resides in the United States, but has no office or principal place of business in the United States, the return shall be filed with the collector of internal revenue for the district in which he resides. If the taxpayer has no office, residence, or principal place of business in the United States, the return shall be filed with the Collector of Internal Revenue, Baltimore, Maryland. The collector, upon request, will furnish the taxpayer proper forms which shall be filled out and signed as indicated therein.

(b) Each return shall show the taxpayer's full name. A person doing business under an alias, style, or trade name shall give his true name, followed by his alias, style, or trade name. In the case of a partnership, association, firm,

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Title 21—Food and Drugs

CHAPTER I—FOOD AND DRUG ADMINISTRATION, DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

[Recodification Docket No. 9]

SUBCHAPTER C—DRUGS: GENERAL

Reorganization and Republication

The Commissioner of Food and Drugs, for the purposes of establishing an orderly development of informative regulations for the Food and Drug Administration, furnishing ample room for expansion of such regulations in years ahead, and providing the public and affected industries with regulations that are easy to find, read, and understand, has initiated a recodification program for Chapter I of Title 21 of the Code of Federal Regulations.

This is the ninth document in a series of recodification documents that will eventually include all regulations administered by the Food and Drug Administration.

This recodification document represents a reorganization of material remaining in Subchapter C—Drugs that has general applicability, rather than strictly human or animal use. In addition certain related sections under Parts 1 and 3 have been redesignated as part of the revised Subchapter C—Drugs: General.

The following table shows the relationship of the CFR section numbers under the former Subchapters A and C to their redesignation reflected in the new Parts 200 through 299:

Old Section	New Section	Old Section	New Section
1.100	299.5	3.21	250.102
1.101	201.6	3.22	200.101
1.101a	201.60	3.27	250.203
1.102	201.50	3.28	200.50
1.102a	201.61	3.29	201.307
1.102b	201.1	3.30	201.308
1.102c	201.51	3.35	201.303
1.102d	201.62	3.36	250.103
1.103	201.15	3.37	201.309
1.104	201.10	3.40	250.201
1.105	202.1	3.43	201.310
1.106(a)	201.5	3.44	201.311
1.106(b)	201.100	3.45	200.30
1.106(c)	201.105	3.48	250.106
1.106(d)	201.109	3.50	250.104
1.106(f)	201.110	3.52	250.107
1.106(g)	201.115	3.53	250.10
1.106(h)	201.116	3.56	201.405
1.106(i)	201.117	3.61	200.18
1.106(j)	201.119	3.62	299.4
1.106(k)	201.120	3.63	250.11
1.106(l)	201.122	3.64	250.12
1.106(m)	201.125	3.67	201.305
1.106(n)	201.127	3.71	250.100
1.106(o)	201.128	3.74	201.56
1.107	201.150	3.76	200.10
1.108(a) & (b)	201.16	3.77	299.35
1.108(c)	299.6	3.81	201.200
1.109	299.5	3.84	201.410
1.110	299.10	3.90	250.300
1.115	200.15	3.91	250.250
3.3	201.300	3.94	250.109
3.4	201.302	3.95	250.110
3.7	250.108	3.501	200.5
3.8	250.101	3.502	201.19
3.11	201.301	3.503	201.312
3.12	201.304	3.505	201.313
3.15	201.306	3.506	200.11
3.16	200.100	3.507	201.17
		3.508	201.18

Old Section	New Section	Old Section	New Section
3.509	201.314	133.11	211.58
3.510	201.315	133.12	211.110
3.512	200.31	133.13	211.60
3.513	200.7	133.14	211.62
3.514	201.55	133.15	211.115
3.515	201.160	133.100	225.1
3.516	250.105	133.101	225.20
3.518	201.161	133.102	225.30
132.1	207.3	133.103	225.10
132.2	207.20	133.104	225.42
132.3	207.21	133.105	225.102
132.4	207.22	133.106	225.40
132.5	207.25	133.107	225.80
132.6	207.30	133.108	225.58
132.7	207.31	133.109	225.110
132.8	207.35	133.110	225.115
132.9	207.37	133.200	226.1
132.10	207.26	133.201	226.20
132.11	207.39	133.202	226.30
132.31	207.40	133.203	226.10
132.51	207.65	133.204	226.42
133.1	210.3	133.205	226.102
133.2	211.1	133.206	226.40
133.3	211.20	133.207	226.80
133.4	211.30	133.208	226.58
133.5	211.10	133.209	226.110
133.6	211.42	133.210	226.115
133.7	211.101	133.300	229.25
133.8	211.40	138.1	299.3
133.9	211.55	138.2	299.20
133.10	211.80		

The changes being made are nonsubstantive in nature and for this reason notice and public procedure are not prerequisites to this promulgation. For the convenience of the user, the entire text of Parts 200, 201, 202, 207, 210, 211, 225, 226, 229, 250, 290, and 299 of Subchapter C is set forth below.

Dated: March 21, 1975.

SAM D. FINE,
Associate Commissioner for Compliance.

Therefore, 21 CFR is amended by redesignating portions of Parts 1 and 3 of Subchapter A and Parts 132, 133, and 138 of Subchapter C as Parts 200, 201, 202, 207, 210, 211, 225, 226, 229, 250, 290, and 299 of Subchapter C—Drugs: General, and republished to read as follows:

SUBCHAPTER C—DRUGS: GENERAL

Part

200—General

201—Labeling

202—Prescription Drug Advertising

207—Registration of Producers of Drugs and Listing of Drugs in Commercial Distribution

210—Current Good Manufacturing Practices in Manufacturing, Processing, Packing, or Holding of Drugs: General

211—Current Good Manufacturing Practice for Finished Pharmaceuticals

225—Current Good Manufacturing Practice for Medicated Feeds

226—Current Good Manufacturing Practice for Medicated Premixes

229—Current Good Manufacturing Practice for Certain Other Drug Products

250—Special Requirements for Specific Human Drugs

290—Controlled Drugs

299—Drugs; Official Names and Established Names

PART 200—GENERAL

Subpart A—General Provisions

Sec.

Subpart B—Manufacturing Procedures Affecting New Drug Status

Subpart C—Requirements for Specific Classes of Drugs

Subpart D—Suitability of Specific Drug Components

AUTHORITY: Sec. 701, 52 Stat. 1055; 21 U.S.C. 371, unless otherwise noted.

Subpart A—General Provisions

§ 200.5 Mailing of important information about drugs.

Manufacturers and distributors of drugs and the Food and Drug Administration occasionally are required to mail important information about drugs to physicians and others responsible for patient care. In the public interest, such mail should be distinctive in appearance so that it will be promptly recognized and read. The Food and Drug Administration will make such mailings in accordance with the specifications set forth in this section. Manufacturers and distributors of drugs are asked to make such mailings as prescribed by this section and not to use the distinctive envelopes for ordinary mail.

(a) Use first class mail and No. 10 white envelopes.

(b) The name and address of the agency or the drug manufacturer or distributor is to appear in the upper left corner of the envelope.

(c) The following statements are to appear in the far left third of the envelope front, in the type and size indicated, centered in a rectangular space approximately 3 inches wide and 2¼ inches high with an approximately ⅛-inch-wide border in the color indicated:

(1) When the information concerns a significant hazard to health, the statement:

IMPORTANT
DRUG
WARNING

The statement shall be in three lines, all capitals, and centered. "Important" shall be in 36 point Gothic Bold type. "Drug" and "Warning" shall be in 36 point Gothic Condensed type. The rectangle's

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code of federal regulations

Food and Drugs

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PARTS 200 TO 299

Revised as of April 1, 1995

CONTAINING
A CODIFICATION OF DOCUMENTS
OF GENERAL APPLICABILITY
AND FUTURE EFFECT

AS OF APRIL 1, 1995

With Ancillaries

Published by
the Office of the Federal Register
National Archives and Records
Administration

as a Special Edition of
the Federal Register

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ing of section 503(b) of the Federal Food, Drug, and Cosmetic Act unless it is labeled with the legend "Caution—Federal law prohibits dispensing without prescription."

(e) Any drug for oral ingestion intended, represented, or advertised for the prevention or treatment of pernicious anemia or which purports to contain any substance or mixture of substances described in paragraph (d) of this section (other than diagnostic drugs containing radioactive cyanocobalamin) will be regarded as misbranded under sections 502(f)(2) and (j) of the act unless its labeling bears a statement to the effect that some patients afflicted with pernicious anemia may not respond to the orally ingested product and that there is no known way to predict which patients will respond or which patients may cease to respond to the orally ingested products. The labeling shall also bear a statement that periodic examinations and laboratory studies of pernicious anemia patients are essential and recommended.

(f) Under section 409 of the Federal Food, Drug, and Cosmetic Act, intrinsic factor and intrinsic factor concentrate are regarded as food additives. No food additive regulation nor existing extension of the effective date of section 409 of the act authorizes these additives in foods, including foods for special dietary uses. Any food containing added intrinsic factor or intrinsic factor concentrate will be regarded as adulterated within the meaning of section 402(a)(2)(C) of the act.

(g) Regulatory action may be initiated with respect to any article shipped within the jurisdiction of the act contrary to the provisions of this policy statement after the 180th day following publication of this statement in the FEDERAL REGISTER.

§250.203 Status of fluoridated water and foods prepared with fluoridated water.

(a) The program for fluoridation of public water supplies recommended by the Department of Health and Human Services, through the Public Health Service (Centers for Disease Control), contemplates the controlled addition of fluorine at a level optimum for the prevention of dental caries.

(b) Public water supplies do not ordinarily come under the provisions of the Federal Food, Drug, and Cosmetic Act. Nevertheless, a substantial number of inquiries have been received concerning the status of such water under the provisions of the act and the status, in interstate commerce, of commercially prepared foods in which fluoridated water has been used.

(c) The Department of Health and Human Services will regard water supplies containing fluorine, within the limitations recommended by the Environmental Protection Agency, as not actionable under the Federal Food, Drug, and Cosmetic Act. Similarly, commercially prepared foods within the jurisdiction of the act, in which a fluoridated water supply has been used in the processing operation, will not be regarded as actionable under the Federal law because of the fluorine content of the water so used, unless the process involves a significant concentration of fluorine from the water. In the latter instance the facts with respect to the particular case will be controlling.

[40 FR 14033, Mar. 27, 1975, as amended at 48 FR 11426, Mar. 18, 1983]

Subpart D—Requirements for Drugs and Cosmetics

§250.250 Hexachlorophene, as a component of drug and cosmetic products.

(a) *Antibacterial component.* The use of hexachlorophene as an antibacterial component in drug and cosmetic products has expanded widely in recent years. It is used in such products because of its bacteriostatic action against gram-positive organisms, especially against strains of staphylococcus; however, hexachlorophene offers no protection against gram-negative infections. In addition, the antibacterial activity depends largely on repeated use. A notice published in the FEDERAL REGISTER of April 4, 1972 (37 FR 6775), invited data on OTC antimicrobial ingredients, including hexachlorophene, for review by an OTC Drug Advisory Review Panel to be convened under the procedures set forth in the FEDERAL REGISTER of May 11, 1972

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List of substances	Limitations
Monochlorobenzene Monochlorobenzene.	Not to exceed 500 parts per million as residual solvent in finished basic resin in paragraph (a)(1) of this section.
N-methyl-2-pyrrolidone.	Not to exceed 0.01 percent (100 parts per million) as residual solvent in finished basic resin in paragraph (a)(2) of this section.

* * * * *

Dated: May 17, 1996.

Fred R. Shank,

Director, Center for Food Safety and Applied Nutrition.

[FR Doc. 96-14697 Filed 6-10-96; 8:45 am]

BILLING CODE 4160-01-F

Food and Drug Administration

21 CFR Parts 200, 250, and 310

[Docket No. 95N-0310]

Revocation of Obsolete Regulations

AGENCY: Food and Drug Administration, HHS.

ACTION: Final rule.

SUMMARY: The Food and Drug Administration (FDA) is revoking certain regulations that are obsolete or are no longer necessary to achieve public health goals. These regulations were among those identified for revocation in a page-by-page review conducted in response to the Administration's "Reinventing Government" initiative, which seeks to streamline government to ease the burden on regulated industry and consumers.

EFFECTIVE DATE: July 11, 1996.

FOR FURTHER INFORMATION CONTACT: Christine F. Rogers, Center for Drug Evaluation and Research (HFD-7), Food and Drug Administration, 7500 Standish Pl., Rockville, MD 20855, 301-594-2041.

SUPPLEMENTARY INFORMATION:

I. Background

In the Federal Register of October 13, 1995 (60 FR 53480), FDA published a proposed rule to revoke certain regulations. This was done in response to the President's order to all Federal agencies to conduct a page-by-page review of all their regulations and to "eliminate or revise those that are outdated or otherwise in need of reform." The proposed rule contained a section-by-section analysis of all the regulations (21 CFR parts 100, 101, et al.) that FDA intended to revoke. This final rule pertains only to those regulations (21 CFR parts 200, 250, and 310) pertaining exclusively to the Center for Drug Evaluation and Research. No comments were received in response to the proposal to revoke these regulations.

II. Analysis of Impacts

FDA has examined the impacts of the final rule under Executive Order 12866 and the Regulatory Flexibility Act (Pub. L. 96-354). Executive Order 12866 directs agencies to assess all costs and benefits of available regulatory alternatives and, when regulation is necessary, to select regulatory approaches that maximize net benefits (including potential economic, environmental, public health and safety, and other advantages; distributive impacts; and equity). The agency believes that this final rule, which is the revocation of certain regulations that are obsolete or are no longer necessary, is consistent with the regulatory philosophy and principles identified in the Executive Order. In addition, the final rule is not a significant regulatory action as defined by the Executive Order and so is not subject to review under the Executive Order.

The Regulatory Flexibility Act requires agencies to analyze regulatory options that would minimize any significant impact of a rule on small entities. Because this final rule is the revocation of certain regulations that are obsolete or are no longer necessary, the agency is not aware of any adverse impact this final rule will have on any small entities, and the agency certifies that the final rule will not have a significant economic impact on a substantial number of small entities. Therefore, under the Regulatory Flexibility Act, no further analysis is required.

III. Environmental Impact

The agency has determined under 21 CFR 25.24(a)(9) that this action is of a type that does not individually or cumulatively have a significant effect on the human environment. Therefore, neither an environmental assessment nor an environmental impact statement is required.

List of Subjects

21 CFR Part 200

Drugs, Prescription drugs.

21 CFR Part 250

Drugs.

21 CFR Part 310

Administrative practice and procedure, Drugs, Labeling, Medical devices, Reporting and recordkeeping requirements.

Therefore, under the Federal Food, Drug, and Cosmetic Act (21 U.S.C. 301 *et seq.*) and under authority delegated to the Commissioner of Food and Drugs, 21 CFR parts 200, 250, and 310 are amended as follows:

PART 200—GENERAL

1. The authority citation for 21 CFR part 200 continues to read as follows:

Authority: Secs. 201, 301, 501, 502, 503, 505, 506, 507, 508, 515, 701, 704, 705 of the Federal Food, Drug, and Cosmetic Act (21 U.S.C. 321, 331, 351, 352, 353, 355, 356, 357, 358, 360e, 371, 374, 375).

2. Sections 200.100 and 200.101 are removed and the heading for subpart D is reserved.

PART 250—SPECIAL REQUIREMENTS FOR SPECIFIC HUMAN DRUGS

3. The authority citation for 21 CFR part 250 continues to read as follows:

Authority: Secs. 201, 306, 402, 502, 503, 505, 601(a), 602(a) and (c), 701, 705(b) of the Federal Food, Drug, and Cosmetic Act (21 U.S.C. 321, 336, 342, 352, 353, 355, 361(a), 362(a) and (c), 371, 375(b)).

§ 250.104 [Removed]

4. Section 250.104 *Status of salt substitutes under the Federal Food, Drug, and Cosmetic Act* is removed.

§ 250.203 [Removed]

5. Section 250.203 *Status of fluoridated water and foods prepared with fluoridated water* is removed.

PART 310—NEW DRUGS

6. The authority citation for 21 CFR part 310 continues to read as follows:

Authority: Secs. 201, 301, 501, 502, 503, 505, 506, 507, 512-516, 520, 601(a), 701, 704, 705, 721 of the Federal Food, Drug, and Cosmetic Act (21 U.S.C. 321, 331, 351, 352, 353, 355, 356, 357, 360b-360f, 360j, 361(a), 371, 374, 375, 379e); secs. 215, 301, 302(a), 351, 354-360F of the Public Health Service Act (42 U.S.C. 216, 241, 242(a), 262, 263b-263n).

§ 310.101 [Removed]

7. Section 310.101 *FD&C Red No. 4; procedure for discontinuing use in new drugs for ingestion; statement of policy* is removed.

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ENVIRONMENTAL PROTECTION AGENCY

[OW-FRL-3410-1]

Drinking Water Technical Assistance; Termination of the Federal Drinking Water Additives Program

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: The Environmental Protection Agency (EPA), Office of Drinking Water (ODW), has operated an advisory program that gives technical assistance to concerned parties on the use of drinking water additives. On May 17, 1984, EPA proposed to terminate major elements of this Federal program and to assist in the establishment of a private-sector program which would offer assistance in evaluating drinking water additives. 49 FR 21004. EPA solicited proposals from qualified nongovernmental, nonprofit organizations for assistance under a cooperative agreement to establish a credible and efficient program in the private sector.

On September 17, 1985, EPA selected a consortium consisting of the National Sanitation Foundation (NSF), the American Water Works Association Research Foundation (AWWARF), the Conference of State Health and Environmental Managers (COSHEM), and the Association of State Drinking Water Administrators (ASDWA) to receive funds under a cooperative agreement to develop the private-sector program. EPA believes that the NSF-led program has proceeded satisfactorily. NSF Standard 60, covering many direct additives, was adopted on December 7, 1987; and NSF Standard 61, covering indirect additives, was adopted on June 3, 1988. Other standards are forthcoming. The NSF-led program has begun offering testing, certification, and listing services, as described in 49 FR 21004, for certain classes of products covered by these standards. Accordingly, as the NSF-led program becomes operational, EPA will phase out its activities in this area, as described in this notice.

DATE: Any written comments on implementing this notice should be submitted to the address below by September 6, 1988.

ADDRESSES: Submit comments to: Mr. Arthur H. Perler, Chief, Science and Technology Branch, Office of Drinking Water (WH-550D), U.S. Environmental Protection Agency, 401 M Street, SW., Washington, DC 20460. A copy of all comments will be available for review during normal business hours at the U.S. Environmental Protection Agency, Criteria and Standards Division, Science and Technology Branch, Room 931ET, 401 M Street, SW., Washington, DC 20460. For further information on the NSF-led private-sector program, including standards development and testing, certification, and listing services, contact: Director, Drinking Water Additives Program, National Sanitation Foundation, P.O. Box 1468, Ann Arbor, MI 48106; or call (313) 769-8010. For information on alternative testing, certification, and listing programs, contact individual State regulatory authorities or the American Water Works Association, Technical and Professional Department, 6666 Quincy Avenue, Denver CO, 80235, or call (303) 794-7711. For information on the directory of products certified as meeting the criteria in a NSF standard, contact the American Water Works Association Research Foundation, 6666 Quincy Avenue, Denver CO, 80235, or call (303) 794-7711.

FOR FURTHER INFORMATION CONTACT: Mr. Arthur H. Perler, Chief, Science and Technology Branch, Office of Drinking Water (WH-550D), U.S. Environmental Protection Agency, 401 M Street, SW., Washington, DC 20460, or call (202) 382-2022.

I. Introduction

The Safe Drinking Water Act (SDWA) (42 U.S.C. 300f *et seq.*) provides for enhancement of the safety of public drinking water supplies through the establishment and enforcement of national drinking water regulations. The Environmental Protection Agency (EPA) has the primary responsibility for establishing the regulations, and the States have the primary responsibility for enforcing such regulations. The regulations control contaminants in drinking water which may have any adverse effect on public health. Section 1412, 42 U.S.C. 300g-1. The regulations include maximum contaminant levels (MCLs) or treatment techniques and monitoring requirements for these contaminants. Sections 1401 and 1412; 42 U.S.C. 300f and 300g-1. EPA also promulgates monitoring requirements for unregulated contaminants. Section 1445; 42 U.S.C. 300j-4. In addition, EPA has broad authorities to provide technical assistance and financial assistance (e.g., grants, cooperative agreements) to States and to conduct research. Sections 1442, 1443, 1444; 42 U.S.C. 300j-1, 300j-2, 300j-3.

The Agency has established MCLs for a number of harmful contaminants that occur naturally or pollute public drinking water supplies. In addition to such contaminants, there is a possibility that drinking water supplies may be contaminated by compounds "added" to drinking water, either directly or indirectly, in the course of treatment and transport of drinking water. Public water systems use a broad range of chemical products to treat water supplies and to maintain storage and distribution systems. For instance, systems may directly add chemicals such as chlorine, alum, lime, and coagulant aids in the process of treating water to make it suitable for public consumption. These are known as "direct additives." In addition, as a necessary function of maintaining a public water system, storage and distribution systems (including pipes, tanks, and other equipment) may be fabricated from or painted, coated, or treated with products which may leach into or otherwise enter the water. These products are known as "indirect additives." Except to the extent that direct or indirect additives consist of ingredients or contain contaminants for which EPA has promulgated MCLs, EPA does not currently regulate the levels of additives in drinking water.

In 1979, EPA executed a Memorandum of Understanding (MOU) with the U.S. Food and Drug Administration (FDA) to establish and clarify areas of authorities with respect to control of additives in drinking water. 44 FR 42775, July 20, 1979. FDA is authorized to regulate "food additives" pursuant to the Federal Food, Drug, and Cosmetic Act (FFDCA). (21 U.S.C. 301 *et seq.*). Both agencies acknowledged in the MOU that "passage of the SDWA in 1974 repealed FDA's authority under the FFDCA over water used for drinking water purposes." The MOU stated that FDA would continue to have authority for taking regulatory action under the FFDCA to control additives in bottled drinking water and in water used in food and for food processing. The MOU went on to say that EPA had authority to control additives in public drinking water supplies.

While the SDWA does not require EPA to control the use of specific additives in drinking water, EPA has provided technical assistance to States and public water systems on the use of additives through the issuance of advisory opinions on the acceptability of many additive products. EPA has provided this technical assistance pursuant to its discretionary authority in section 1442(b)(1) to "collect and make available information pertaining to research, investigations and demonstrations with respect to

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providing a dependable safe supply of drinking water together with appropriate recommendations in connection therewith." EPA has additional authorities under the Toxic Substances Control Act (TSCA) (15 U.S.C. 2601 *et seq.*) and the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) (7 U.S.C. 136 *et seq.*) that could be used to control additives in drinking water. TSCA authorizes EPA to regulate a new chemical substance before it is manufactured or any existing chemical substance before it is manufactured or processed for a use that EPA has determined to be a "significant new use." Although an additive product might come within the jurisdiction of TSCA, EPA has never invoked this authority. EPA has used its authority under FIFRA to control the use of pesticides, disinfectants, and certain other additives. For a more complete discussion of these authorities, see the MOU. 44 FR 42776.

In 1980, EPA declared a moratorium on the issuance of new advisory opinions on additives pending a review of past advisory opinions and the establishment of uniform test protocols and decision criteria. However, between 1980 and 1984, EPA continued to issue advisory opinions in cases where the new additive products were virtually identical to products previously reviewed. Resource constraints and the need to implement mandatory provisions of the SDWA precluded the Agency from implementing the comprehensive program originally envisioned for the issuance of additives advisory opinions. Thus, the Agency was not able to review the technical data supporting previous submissions (approximately 2,300 products from 525 manufacturers) nor was it able to develop test protocols or decision criteria for the consistent evaluation of new products. The result has been long delays in processing manufacturer petitions, inability to review and accept completely new products, and acceptance of products simply because they were virtually identical to older products. Hence, few products have been thoroughly evaluated for the safety of their formulations based on the latest scientific information.

Recognizing the need for continuing technical assistance in evaluating additive products and for providing advice to States and public water systems on the toxicological aspects of additive products, the Agency proposed to terminate its attempts to institute a formal advisory program, and to solicit proposals from nongovernmental, nonprofit organizations to establish such a program in the private sector. The Agency believed that the proposal to assist in the establishment of a private-sector program was consistent with, and would best serve the goals of, the SDWA.

On May 17, 1984, EPA formally announced its intention to transfer the program to the private sector, which would function as to many other voluntary product-standard programs. 49 FR 21004. This was accomplished by requesting proposals from qualified organizations or consortia of organizations for the competitive award of a cooperative agreement designed to provide incentive for the establishment of a private-sector program. The 1984 notice stated that:

- EPA expected the activity to be self-supporting.
- EPA would maintain an active interest in the development of the program, without assuming responsibility for or directing its approach.
- EPA would continue to establish regulations under the SDWA, FIFRA, and/or TSCA, as needed, for chemicals in treated, distributed drinking water that may originate as additives.
- Establishment of such a program would be consistent with the Administration's initiatives in the area of regulatory reform and offered an opportunity for an innovative alternative to regulation.

The May 1984 notice requested public comments on the proposal and solicited applications from qualified nongovernmental, nonprofit organizations for partial funding of the developmental phase of the program under a cooperative agreement. The response to the solicitation for comments indicated strong public support for the proposed approach. EPA received 106 public comments on the proposal. All but six supported this "third-party" approach. However, despite the Agency's open competition, EPA received only one application for financial assistance. The applicant was a consortium, led by the National Sanitation Foundation, which included the American Water Works Association Research Foundation, the Conference of State Health and Environmental Managers, and the Association of State Drinking Water Administrators. This single proposal met all of the basic criteria articulated in the May 1984 notice. Furthermore, EPA believed that the single applicant was very likely to succeed, because it represented an organization experienced in private-sector consensus standard-setting, State regulators, and water utilities.

EPA awarded the cooperative agreement to the NSF consortium on September 17, 1985, and committed funding of $185,000 to NSF over a three-year period. The non-Federal (consortium and participating industry) contribution during the first three years of the program was projected to be approximately $1.4 million.

The NSF program has the following major objectives:

- To develop systematic, consistent, and comprehensive voluntary consensus standards for public health safety evaluation of all products (previously EPA-accepted as well as new) intended for use in drinking water systems.
- To obtain broad-based participation in the standard-setting program from industry, States, and utilities.
- To provide for regular periodic review, update, and revision of the standards.
- To undertake needed research, testing, evaluation, and inspections and to provide the followup necessary to maintain the program.
- To establish a separate program for testing, evaluation, certification, and listing of additive products.
- To widely disseminate information about the program, and to make information about conforming products available to users.
- To maintain the confidentiality of all proprietary information.
- To fully establish the third-party program on a self-supporting basis.

NSF's established standard-setting process utilizes a tiered structure. Each standard is drafted by a task group and then presented to a Joint Committee, which includes 12 industry, 12 user, and 12 regulatory members. Following successful Joint Committee balloting, standards are reviewed by the Council of Public Health Consultants, which is a high level advisory group consisting of technical and policy experts from regulatory agencies and academia.

NSF has established task groups to develop standards for the product categories listed below. Each task group includes a member representing the regulatory agencies and a member representing the utilities. All manufacturers expressing interest in a particular product task group may participate as members of that group. Therefore, task group membership is predominately manufacturers. In addition, a group of health effects consultants is addressing the toxicological and risk considerations for various product categories. NSF's role in the standard-setting process is administrative, that is, to bring together experts from government, industry,

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utilities, users, and other relevant groups so that a standard which reflects a consensus of these interests can be developed. In addition, NSF staff provide technical leadership and laboratory support. Product categories and correspoding task groups are:

- Protective Materials.
- Chemicals for Corrosion and Scale Control, Softening, Precipitation, Sequestering, and pH Adjustment.
- Coagulation and Flocculation Chemicals.
- Miscellaneous Treatment Chemicals.
- Joining and Sealing materials.
- Process Media.
- Pipes and Related Products.
- Disinfection and Oxidation Chemicals.
- Mechanical Devices.

All of the task groups have made satisfactory progress during the term of the cooperative agreement. In addition, the health effects consultants have endorsed the bases of the standards. Standards have been drafted for all product categories, and final standards were published and implemented as follows:

Standard 60, December 1987

- Chemicals for Corrosion and Scale Control, Softening, Precipitation, Sequestering, and pH Adjustment.
- Disinfection and Oxidation Chemicals.
- Miscellaneous Treatment Chemicals (selected).

Standard 61, June 1988

- Process Media.

Development of the remaining standards is on schedule, and publication and implementation are expected on the following schedule:

Standards 60 and 61, expected October 1988

- Protective Materials.
- Coagulation and Flocculation Chemicals.
- Miscellaneous Treatment Chemicals (additional).
- Joining and Sealing Materials.
- Pipes and Related Products.
- Mechanical Devices.

EPA believes that the NSF program is successfully pursuing all of its objectives. Furthermore, the program is strongly supported by user and regulatory sectors. AWWARF, COSHEM, ASDWA, the Great Lakes Upper Mississippi River Board, the American Water Works Association (AWWA) (including the Utilities and Standards Councils and the Regulatory Agencies Division), and the Association of Metropolitan Water Agencies, among others, have voiced strong support for the third-party program. The AWWA recently joined the NSF-led consortium and urged EPA to support national uniform accreditation of certifying entities for additives products. To date, more than 60 manufacturers are full participants in the standard-setting program.

The cooperative agreement between EPA and the consortium requires NSF to establish both a standard-setting program and a service for testing, certification, and listing. These are completely separate activities. EPA's intent is to support the development of a widely accepted uniform standard for each category of products while encouraging the development of competing sources for testing, certification, and listing. The cooperative agreement assures that at least one sound and reliable product-evaluation service will be available to manufacturers, i.e., the consortium. However, the consortium's standards will allow for entities other than NSF to be evaluators of products.

EPA recognizes the authority and responsibility of the individual States to determine the acceptability of drinking water additives. Hence, it is up to the States and utilities to determine the suitability of any "third-party" certification. AWWARF will maintain a directory of products approved by all organizations claiming to conduct evaluations under Standards 60 and 61. However, AWWARF will not judge the competence or reliability of these organizations.

II. Announcement of Phase-Down of EPA's Additives Program

During the developmental phase of the NSF consortium's program, EPA has continued to review products and process requests for advisory opinions on a limited basis. The May 1984 notice stated that, "EPA does not intend to develop further interim administrative procedures, testing protocols or decision criteria for future evaluation of additive products. The use of existing informal criteria will continue until a third-party or alternative program is operational * * *. EPA may not be able to process all requests for opinions on additive products before the establishment of a cooperative agreement with a third party. The large volume of currently pending requests makes it unlikely that additional requests will be completely processed by that date." Likewise, EPA, in its acknowledgment letters to manufacturers requesting opinions on new products, explains that the Agency is, "* * * making a concerted effort to process petitions as quickly as possible. However, EPA may not be able to process your request for an opinion on an additive product before the establishment of an alternative program as described in the Federal Register, Vol. 49, No. 97, 21003–8, May 17, 1984." Product reviews and issuance of advisory opinions have been limited to:

- Products composed entirely of other products which EPA had previously determined to be acceptable;
- Products composed entirely of ingredients which have been determined to be acceptable by EPA or the FDA, or other Federal agencies, for addition to potable water or aqueous foods;
- Products composed entirely of ingredients listed in the "Water Chemicals Codex," National Academy of Sciences, November 1982, and in the "Water Chemicals Codex: Supplementary Recommendations for Direct Additives," National Academy of Sciences, 1984;
- Certain other products of particular interest to EPA or to other Federal agencies; and
- Products which, if effectively excluded from the marketplace by lack of approval, might jeopardize public health or safety.

Continued processing of petitions during the development of the private-sector program minimized disruption of the marketplace from the viewpoint of manufacturers whose business depended in part on EPA acceptance of products, users who required water treatment products for the production of safe drinking water, and State officials who rely on the advice of EPA.

EPA believes that NSF is moving expeditiously and on schedule toward the full establishment of a third-party program covering products intended for use in drinking water systems. Priorities for standards development and implementation of a testing, certification, and listing program for various product categories have been based upon need, interest, complexity, and availability of information for developing standards. Direct drinking water additives were assigned high priority for the following reasons: (1) Use of direct additives is widespread in drinking water systems, so there are large population exposures to these chemicals; (2) as direct additives to drinking water, they present greater potential for water contamination than indirect mechanisms (e.g., migration from protective paints in pipes and storage tanks); and (3) the National Academy of Sciences' *Water Chemicals Codex* provided a good starting point for development of standards.

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As originally planned, EPA is beginning to phase out the Agency's additives evaluation program. Thus, EPA will not accept new petitions or requests for advisory options after the date of this notice. While EPA will continue to process requests which are pending and those received on or before July 7, 1988, petition evaluations not completed by October 4, 1988, will be returned to the submitter. After that date, EPA will no longer evaluate additive products.

Petitions which are completely evaluated by October 5, 1988, will be added to the quarterly list of acceptable products published shortly after that date. That quarterly list will be the last such list issued by EPA. On April 7, 1990, EPA will withdraw its list of acceptable products, and the list and the advisories on these additives will expire. This means that: (1) The various lists published by EPA under the titles *Report on Acceptable Drinking Water Additives, Report on Coagulant Aids for Water Treatment, Report on Concrete Coatings/Admixture for Water Treatment, Report on Detergents, Sanitizers and Joint Lubricants for Water Treatment, Report on Evaporative Suppressants for Water Treatment, Report on Liners/Grouts/Hoses and Tubings for Water Treatment, Report on Miscellaneous Chemicals for Water Treatment, Report on Protectivce Paints/Coatings for Water Treatment,* and any and all other lists of drinking water products issued by EPA or its predecessor agencies regarding drinking water additives will be invalid after April 7, 1990; and (2) advisory opinions on drinking water additives issued by EPA and predecessor agencies will be invalid after that date.

EPA believes that, while in the past every effort has been made to provide the best possible evaluations, all products should be evaluated against carefully developed and considered nationally uniform standards. Many of the currently listed products were evaluated and accepted up to 20 years ago and have not been reevaluated since that time. Numerous products have been accepted because they were virtually identical to or were repackagings of older products. The result is that few products have been completely evaluated for the safety of their original or current formulations vis-a-vis the latest toxicological, chemical, and engineering information. A uniform evaluation of all products, old and new, will result in consistent quality of products, and will assure fair and equitable treatment to all manufacturers and distributors.

Henceforth, parties desiring to have existing or new products evaluated against the NSF standards should contact NSF or other organizations offering such evaluations. To contact NSF about the drinking water additives program write to: David Gregorka, National Sanitation Foundation, P.O. Box 1468, Ann Arbor, MI 48106, or call (313) 769-8010. Information on alternatives to NSF evaluation may be obtained by contacting State regulatory agencies or the AWWA, Technical and Professional Department, 6666 Quincy Avenue, Denver Co, 80235, or call (303) 794-7711, which is addressing certifier accreditation.

EPA believes that the 21 months between today and the expiration date of EPA's last list is sufficient time for manufacturers to submit their products to NSF or other certification entities for evaluation. The first NSF list will be published prior to April 7, 1990, thereby preventing any disruption in the marketplace. Furthermore, NSF had indicated that it will consider current EPA and other regulatory evaluations when evaluating products in order to ensure a smooth transition. States may choose to rely on the last EPA quarterly list of products until their individual programs for accepting private-sector certification are fully implemented.

Parties desiring to market drinking water additive products are reminded that the individual States have the authority to regulate the sale and/or use of specific products as they see fit. Thus, reliance upon a particular standard or organization to certify that a product complies with a particular standard must be acceptable to the State in which the supplier wishes to do business.

Discontinuation of the additives program at EPA does not relieve the Agency of its statutory responsibilities. If contamination resulting from third-party sanctioned products occurs or seems likely, EPA will address that issue with appropriate drinking water regulations or other actions authorized under the SDWA. EPA is a permanent member of the NSF program Steering Committee, and senior EPA staff and management will continue to participate in this and other programs designed to assure that high-quality products are employed in the treatment of public drinking water. Also, the Agency will continue to sponsor research on contaminants introduced in public water supplies during water treatment, storage, and distribution.

III. Comments

Although this notice does not include a proposed or final regulation, EPA welcomes comments and suggestions that would assist the Agency in implementing the additives program phasedown. Please address all comments and suggestions to: Mr. Arthur H. Perler, Chief, Science and Technology Branch, Office of Drinking Water (WH-550D), U.S. Environmental Protection Agency, 401 M Street, SW., Washington, DC 20460.

Date: June 16, 1988.

William Whittington,

Acting Assistant Administrator for Water.

[FR Doc. 88-15232 Filed 7-6-88; 8:45 am]

BILLING CODE 6560-50-M

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Federal facilities. Prior to making a final recommendation to the Administrator, U.S. EPA, the Regional Administrator, Region V, is providing opportunity for public comment on the State of Wisconsin request. Any interested person may comment upon the State request by writing to the U.S. EPA, Region V Office, 230 South Dearborn Street, Chicago, Illinois 60604, Attention: Permit Branch. Such comments will be made available to the public for inspection and copying. All comments or objections received by August 22, 1979, will be considered by U.S. EPA before taking final action on the Wisconsin request for authority to issue permits to Federal facilities.

The State's request, related documents, and all comments received are on file and may be inspected and copied (@ 20 cents/page) at the U.S. EPA, Region V Office, in Chicago.

Copies of this notice are available upon request from the Enforcement Division of U.S. EPA, Region V, by contacting Dorothy A. Price, Public Notice Clerk (312–353–2105), at the above address.

Dated: July 13, 1979.

John McGuire,
Regional Administrator.

[FR Doc. 79-22872 Filed 7-19-79; 8:45 am]

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

Food and Drug Administration

ENVIRONMENTAL PROTECTION AGENCY

[FRL 1275–4]

Drinking Water Technical Assistance; Implementation Plan for Control of Direct and Indirect Additives to Drinking Water and Memorandum of Understanding Between the Environmental Protection Agency and the Food and Drug Administration

AGENCY: Environmental Protection Agency and Food Drug Administration.

ACTION: Notice.

SUMMARY: The Food and Drug Administration (FDA) and the Environmental Protection Agency (EPA) have executed a memorandum of understanding (MOU) with regard to the control of direct and indirect additives to and substances in drinking water. The purpose of the MOU is to avoid the possibility of overlapping jurisdiction between EPA and FDA with respect to control of drinking water additives. The agreement became effective on June 22, 1979.

ADDRESS: Submit comments to: Victor J. Kimm, Deputy Assistant Administrator for Drinking Water, Environmental Protection Agency (WH–550), Washington, D.C. 20460.

FOR FURTHER INFORMATION CONTACT: David W. Schnare, Ph.D., Office of Drinking Water (WH–550), Environmental Protection Agency, Washington, D.C. 20460, (202) 755–5643; or Gary Dykstra, Enforcement Policy Staff (HFC–22), Food and Drug Administration, 5600 Fishers Lane, Rockville, MD 20857, (301) 443–3470.

SUPPLEMENTARY INFORMATION: In the spirit of interagency cooperation and to avoid the possibility of overlapping jurisdiction over additives and other substances in drinking water, FDA and EPA have entered into a memorandum of understanding to avoid duplicatve and inconsistent regulation. In brief, the memorandum provides that EPA will have primary responsibility over direct and indirect additives and other substances in drinking water under the Safe Drinking Water Act, the Toxic Substances Control Act, and the Federal Insecticide, Fungicide and Rodenticide Act. FDA will have responsibility for water, and substances in water, used in food and for food processing and for bottled water under the Federal Food, Drug and Cosmetic Act.

Pursuant to the notice published in the Federal Register of October 3, 1974, (39 FR 35697) stating that future memoranda of understanding, and agreements between FDA and others would be published in the Federal Register, the following memorandum of understanding is issued:

Memorandum of Understanding Between the Environmental Protection Agency and the Food and Drug Administration

I. Purpose

This Memorandum of Understanding establishes an agreement between the Environmental Protection Agency (EPA) and the Food and Drug Administration (FDA) with regard to the control of direct and indirect additives to and substances in drinking water.

EPA and FDA agree:

(1) That contamination of drinking water from the use and application of direct and indirect additives and other substances poses a potential public health problem;

(2) That the scope of the additives problem in terms of the health significance of these contaminants in drinking water is not fully known;

(3) That the possibility of overlapping jurisdiction between EPA and FDA with respect to control of drinking water additives has been the subject of Congressional as well as public concern;

(4) That the authority to control the use and application of direct and indirect additives to and substances in drinking water should be vested in a single regulatory agency to avoid duplicative and inconsistent regulation;

(5) That EPA has been mandated by Congress under the Safe Drinking Water Act (SDWA), as amended, to assure that the public is provided with safe drinking water;

(6) That EPA has been mandated by Congress under the Toxic Substances Control Act (TSCA) to protect against unreasonable risks to health and the environment from toxic substances by requiring, *inter alia*, testing and necessary restrictions on the use, manufacture, processing, distribution, and disposal of chemical substances and mixtures;

(7) That EPA has been mandated by Congress under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended, to assure, *inter alia*, that when used properly, pesticides will perform their intended function without causing unreasonable adverse effects on the environment; and,

(8) That FDA has been mandated by Congress under the Federal Food, Drug, and Cosmetic Act (FFDCA), as amended, to protect the public from, *inter alia*, the adulteration of food by food additives and poisonous and deleterious substances.

It is the intent of the parties that;

(1) EPA will have responsibility for direct and indirect additives to and other substances in drinking water under the SDWA, TSCA, and FIFRA; and,

(2) FDA will have responsibility for water, and substances in water, used in food and for food processing and responsibility for bottled drinking water under the FFDCA.

II. Background

(A) *FDA Legal Authority.* "Food" means articles used for food or drink for man or other animals and components of such articles. (FFDCA § 201(f)). Under Section 402, *inter alia*, a food may not contain any added poisonous or deleterious substance that may render it injurious to health, or be prepared, packed or handled under unsanitary conditions. Tolerances may be set, under Section 403, limiting the quantity of any substance which is required for the production of food or cannot be avoided in food. FDA has the authority under Section 409 to issue food additive regulations approving, with or without conditions, or denying the use of a "food additive." That term is defined in Section 201(s) to include any substance the intended use of which results or may reasonably be expected to result, directly or indirectly, in its becoming a component or otherwise affecting the characteristics of any food, if such substance is not generally recognized as safe.

In the past, FDA has considered drinking water to be a food under Section 201(f). However, both parties have determined that the passage of the SDWA in 1974 implicitly repealed FDA's authority under the FFDCA over water used for drinking water purposes. Under the express provisions of Section 410

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of the FFDCA. FDA retains authority over bottled drinking water. Furthermore, all water used in food remains a food and subject to the provisions of the FFDCA. Water used for food processing is subject to applicable provisions of FFDCA. Moreover, all substances in water used in food are added substances subject to the provisions of the FFDCA, but no substances added to a public drinking water system before the water enters a food processing establishment will be considered a food additive.

(B). *EPA Legal Authority.* The SDWA grants EPA the authority to control contaminants in drinking water which may have any adverse effect on the public health, through the establishment of maximum contaminant levels (MCLs) or treatment techniques, under Section 1412, which are applicable to owners and operators of public water systems. The expressed intent of the Act was to give EPA exclusive control over the safety of public water supplies. Public water systems may also be required by regulation to conduct monitoring for unregulated contaminants under Section 1445 and to issue public notification of such levels under Section 1414(c).

EPA's direct authority to control additives to drinking water apart from the existence of maximum contaminant levels or treatment techniques is limited to its emergency powers under Section 1431. However, Section 1442(b) of the act authorizes EPA to "collect and make available information pertaining to research, investigations, and demonstrations with respect to providing a dependably safe supply of drinking water together with appropriate recommendations therewith."

TSCA gives EPA authority to regulate chemical substances, mixtures and under some circumstances, articles containing such substances or mixtures. Section 4 permits EPA to require testing of a chemical substance or mixture based on possible unreasonable risk of injury to health or the environment, or on significant or substantial human or environmental exposure while Section 8 enables EPA to require submission of data showing substantial risk of injury to health or the environment, existing health and safety studies, and other data. For new chemical substances, and significant new uses of existing chemical substances, Section 5 requires manufacturers to provide EPA with premanufacturing notice. Under Section 6 the manufacture, processing, distribution, use, and disposal of a chemical substance or mixture determined to be harmful may be restricted or banned. Although Section 3(2)(B) of TSCA excludes from the definition of "chemical substance" food and food additives as defined under FFDCA, the implicit repeal by the SDWA of FDA's authority over drinking water enables EPA to regulate direct and indirect additives to drinking water as chemical substances and mixtures under TSCA.

The FIFRA requires EPA to set restrictions on the use of pesticides to assure that when used properly, they will not cause unreasonable adverse effects on the environment. EPA may require, *inter alia*, labeling which specifies how, when, and where a pesticide may be legally used. In addition, EPA has, under Section 409 of the FFDCA, required FIFRA registrants at times to obtain a food additive tolerance before using a pesticide in or around a drinking water source. Such tolerances establish further restrictions on the use of a pesticide which are enforceable against the water supplier as well as the registrant of the pesticide.

III. Terms of Agreement

(A) EPA's responsibilities are as follows:

(1) To establish appropriate regulations, and to take appropriate measures, under the SDWA and/or TSCA, and FIFRA, to control direct additives to drinking water (which encompass any substances purposely added to the water), and indirect additives (which encompass any substances which might leach from paints, coatings or other materials as an incidental result of drinking water contact), and other substances.

(2) To establish appropriate regulations under the SDWA to limit the concentrations of pesticides in drinking water; the limitations on concentrations and types of pesticides in water are presently set by EPA through tolerances under Section 409 of the FFDCA.

(3) To continue to provide technical assistance in the form of informal advisory opinions on drinking water additives under Section 1442(b) of the SDWA.

(4) To conduct and require research and monitoring and the submission of data relative to the problem of direct and indirect additives in drinking water in order to accumulate data concerning the health risks posed by the presence of these contaminants in drinking water.

(B). FDA's responsibilities are as follows:

(1) To take appropriate regulatory action under the authority of the FFDCA to control bottled drinking water and water, and substances in water, used in food and for food processing;

(2) To provide assistance to EPA to facilitate the transition of responsibilities, including:

(a) To review existing FDA approvals in order to identify their applicability to additives in drinking water.

(b) To provide a mutually agreed upon level of assistance in conducting literature searches related to toxicological decision making.

(c) To provide a senior toxicologist to help EPA devise new procedures and protocols to be used in formulating advice on direct and indirect additives to drinking water.

IV. Duration of Agreement

This Memorandum of Understanding shall continue in effect unless modified by mutual consent of both parties or terminated by either party upon thirty (30) days advance written notice to the other.

This Memorandum of Understanding will become effective on the date of the last signature.

Dated: June 13, 1979.

Douglas M. Costle,

Administrator, Environmental Protection Agency.

Dated: June 22, 1979.

Donald Kennedy,

Administrator, Food and Drug Administration.

Implementation Plan

EPA is concerned that direct and indirect additives may be adding harmful trace chemical contaminants into our Nation's drinking water during treatment, storage and distribution. Direct additives include such chemicals as chlorine, lime, alum, and coagulant aides, which are added at the water treatment plant. Although these chemicals themselves may be harmless, they may contain small amounts of harmful chemicals if their quality is not controlled. Indirect additives include those contaminants which enter drinking water through leaching, from pipes, tanks and other equipment, and their associated paints and coatings. This notice is being published in the Federal Register to solicit public comment on EPA's implementation plan to assess and control direct and indirect additives in drinking water.

Legal Authorities

EPA and the Food and Drug Administration (FDA) signed a Memorandum of Understanding which recognizes that regulatory control over direct and indirect additives in drinking water is placed in EPA. The two agencies agreed that the Safe Drinking Water Act's passage in 1974 implicitly repealed FDA's jurisdiction over drinking water as a 'food' under the Federal Food, Drug and Cosmetic Act (FFDCA). Under the agreement, EPA now retains exclusive jurisdiction over drinking water served by public water supplies, including any additives in such water. FDA retains jurisdiction over bottled drinking water under Section 410 of the FFDCA and over water (and substances in water) used in food or food processing once it enters the food processing establishment.

In implementing its new responsibilities, EPA may utilize a variety of statutory authorities, as appropriate. The authorities are identified in Appendix A.

Under the Safe Drinking Water Act, EPA has authority to set and enforce maximum contaminant levels and treatment techniques in drinking water for ubiquitous contaminants, to conduct research, to offer technical assistance to States and to protect against imminent

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hazards should such situations arise. Under the Toxic Substances Control Act, EPA has authority to review all new chemicals proposed for use related to drinking water, to mandate toxicological testing of existing and new chemicals where there is evidence that such materials may pose an unreasonable risk to health and the environment as well as authority to limit some or all uses of harmful chemicals. Pesticide use is regulated by EPA under the Federal Insecticide, Fungicide and Rodenticide Act. Thus, EPA believes it has adequate authority to deal with additives to drinking water where they may pose a problem.

Past Actions

For more than ten years, the Public Health Service and other organizations which have become part of EPA have provided advisory opinions on the toxicological safety of a variety of additives to drinking water. These historical informal opinions reflect a variety of information provided by manufacturers and reflect changing toxicological concerns over the years. As such, they will require detailed review over the next few years.

General Approach

EPA intends to begin its responsibility over additives to drinking water with a series of analytical studies to determine the composition and significance of the health risks posed by contaminants related to direct and indirect additives to drinking water. A first step in this process will be monitoring studies of the contaminants actually getting into drinking water from generic categories of additives like bulk chemicals, paints and coatings, pipes and equipment.

In the initial six to twelve months, EPA will develop interim administrative procedures, testing protocols, and decision criteria for future toxicological advisories to the States. These will be distributed for public comment once they are developed. All existing opinions will remain in effect until a general review of past opinions can be undertaken using the new procedures. During this development phase, no new opinions will be rendered unless a proposed product can be shown to be virtually identical to a product for which an opinion has already been rendered, on the basis of chemical formulation and production process. New products or new uses of existing products which are proposed for use in drinking water will be subject to the pre-manufacture notice procedures of TSCA.

A more detailed outline of the steps to be taken by EPA follows.

1. *Problem Definition.*—EPA will contract for *in situ* monitoring to determine use patterns and the contribution of trace contaminants to drinking water from:

a. bulk chemicals.

b. generic classes of paints and coatings.

c. pipes and equipment.

d. coagulant aids.

EPA has already contracted with the National Academy of Sciences to develop a CODEX system of quality control standards for chemicals (direct additives) used in the treatment of drinking water. This effort will take about three years to complete. When finished, the CODEX system, modeled on the existing FDA-inspired CODEX system for chemicals used in processing food, will be largely self-enforcing.

For the indirect additives listed in items b and c above, considerable effort will be expended to identify the trace contaminants involved before the related health risks can be fully evaluated and appropriate recommendations for future use can be assessed.

2. *Review of Past Advisories.*—The same data base derived from *in situ* monitoring will serve as a basis for a structured reassessment of past toxicological advisories which will be conducted by generic classes of use e.g., paints, coagulant aides, etc. Past opinions will be reviewed to insure conformance with and satisfaction of new test protocols and decision criteria that will be developed.

3. *Future Toxicological Advisories.*—Once initial procedures, test protocols and decision criteria are developed, EPA will resume offering toxicological opinions to the States.

General Policy

In assessing additives to drinking water, EPA will be guided by a policy of reducing public health risks to the degree it is feasible to do so. In such determinations, EPA will evaluate the risks and benefits associated with the materials of concern and their substitutes. Economic impacts of agency actions will also be analyzed.

Notwithstanding these procedures, EPA would use its authorities to protect against any direct or indirect additive to drinking water when data and information indicate that the use of any additive may pose an undue risk to public health.

Implementation

To fulfill this program, resources from the Office of Drinking Water, the Office of Research and Development, and the Office of Toxic Substances will be used. In addition, EPA looks forward to the cooperation of FDA and other Federal regulatory bodies. EPA intends to involve interested industry groups, independent testing groups, State regulatory bodies, interested members of the public, and industry standards groups, in a continued effort to ensure the safety of the Nation's drinking water.

Finally, EPA may recommend specialized legislative authority to regulate additives to drinking water should a situation arise for which legal authorities prove inadequate.

Lead responsibility for this new Federal initiative will be in EPA's Office of Drinking Water. Public comments on any or all aspects of the proposed program are requested, and should be directed to the address given in the opening sections of this notice.

Dated: July 13, 1979.

Thomas C. Jorling,

Assistant Administrator for Water and Waste Management.

Appendix A

Safe Drinking Water Act

Section 1412—establishment of national primary drinking water regulations applicable to public water systems to control contaminants in drinking water which may have any adverse effect on human health. This may include maximum contaminant levels, treatment techniques, monitoring requirements, and quality control and testing procedures.

Section 1431—use of emergency powers where a contaminant which is present in water, or is likely to enter a public water system, may present an imminent and substantial endangerment to the health of persons.

Section 1445—establishment of monitoring and reporting requirements applicable to public water systems.

Section 1450—authority to prescribe such regulations as are necessary or appropriate to carry out the Administrator's functions under the Act.

Toxic Substances Control Act

Section 4—testing of chemical substances and mixtures.

Section 5—pre-manufacture notice required for new chemicals or significant new uses.

Section 6—regulation of hazardous chemical substances and mixtures which pose an unreasonable risk of injury to health or the environment, including restrictions on manufacture, processing, distribution, and use.

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Section 7—imminent hazards authority including seizure and other relief through civil court action.

Section 8—reporting and retention of information as required by the Administrator, including health and safety studies and notice to the Administrator of substantial risks.

Section 10—research and development. Development of systems for storing, retrieving and disseminating data.

Section 11—inspections and subpenas and other enforcement and general administration provisions therein.

Federal Insecticide, Fungicide and Rodenticide Act

Section 3—registration of pesticides, including imposition of restrictions and labeling requirements.

Section 6—suspension and cancellation procedures.

[FR Doc. 79-22222 Filed 7-18-79; 8:45 am]

BILLING CODE 6560-01-M

BILLING CODE 4110-03-M

FEDERAL COMMUNICATIONS COMMISSION

[Report No. A-1a]

FM Broadcasting Applications Accepted for Filing and Notification of Cut-off Date; Erratum

Released: July 12, 1979.

The FM Application listed below was inadvertently included on the acceptance/cut-off notice, Report No. A-1, BC Mimeo No. 18676, released on June 25, 1979.

BPH-790108AE (New): Creston, Pennsylvania, Sherlock-Hart Broadcasting, Inc.
Req.: 94.3 MHz, Channel 232A
ERP: 0.600 kW, HAAT: 600 feet.

Accordingly, the application is removed from the acceptance/cutoff list and the August 8, 1979, cutoff date is deleted.

Federal Communications Commission.

William J. Tricarico,

Secretary.

[FR Doc. 79-22422 Filed 7-19-79; 8:45 am]

BILLING CODE 6712-01-M

FEDERAL LABOR RELATIONS AUTHORITY

Official Time of Employees Involved in Negotiating Collective Bargaining Agreements

AGENCY: Federal Labor Relations Authority.

ACTION: Notice Relating to Official Time.

SUMMARY: This notice principally relates to the interpretation of section 7131 of the Federal Service Labor-Management Relations Statute (92 Stat. 1214) on the questions of whether employees who are on official time under this section while representing an exclusive representative in the negotiation of a collective bargaining agreement are entitled to payments from agencies for their travel and per diem expenses, and whether the official time provisions of section 7131(a) of the Statute encompass all negotiations between an exclusive representative and an agency, regardless of whether such negotiations pertain to the negotiation or renegotiation of a basic collective bargaining agreement. The notice further invites interested persons to address the impact, if any, of section 7135(a)(1) of the Statute (92 Stat. 1215) on such interpretation, and to submit written comments concerning these matters.

DATE: Written comments must be submitted by the close of business on August 24, 1979, to be considered.

ADDRESS: Send written comments to the Federal Labor Relations Authority, 1900 E Street, NW., Washington, D.C. 20424.

FOR FURTHER INFORMATION CONTACT: Harold D. Kessler, Deputy Executive Director, 1900 E Street, NW., Washington, D.C. 20424, (202) 632-3920.

SUPPLEMENTARY INFORMATION: The Federal Labor Relations Authority was established by Reorganization Plan No. 2 of 1978, effective January 1, 1979 (43 FR 36037). Since January 11, 1979, the Authority has conducted its operations under the Federal Service Labor-Management Relations Statute (92 Stat. 1191).

Upon receipt of requests and consideration thereof, the Authority has determined, in accordance with 5 CFR 2410.3(a) (1978) and sections 7105 and 7135(b) of the Statute (92 Stat. 1196, 1215), that an interpretation is warranted concerning section 7131 of the Statute (92 Stat. 1214). Interested persons are invited to express their views in writing on this matter, as more fully explained in the Authority's notice set forth below:

To Heads of Agencies, Presidents of Labor Organizations and Other Interested Persons

The Authority has received a request from the American Federation of Government Employees (AFGE) for a statement of policy and guidance concerning whether employees representing an exclusive representative in the negotiation of a collective bargaining agreement are entitled to payments from agencies for their travel and per diem expenses under the official time provisions of section 7131 of the Federal Service Labor-Management Relations Statute (92 Stat. 1214). Additionally, the National Federation of Federal Employees (NFFE) has requested a major policy statement as to the application of the official time provisions of section 7131(a) of the Statute (92 Stat. 1214) to all negotiations between an exclusive representative and an agency, regardless of whether such negotiations pertain to the negotiation or renegotiation of a basic collective bargaining agreement. AFGE has raised a similar issue in its request.

The Authority hereby determines, in conformity with 5 CFR 2410.3(a) (1978) and section 7135(b) of the Statute (92 Stat. 1215), as well as section 7105 of the Statute (92 Stat. 1196), that an interpretation of the Statute is warranted on the following:

(1) Whether employees who are on official time under section 7131 of the Statute while representing an exclusive representative in the negotiation of a collective bargaining agreement are entitled to payments from agencies for their travel and per diem expenses.

(2) Whether the official time provisions of section 7131(a) of the Statute encompass all negotiations between an exclusive representative and an agency, regardless of whether such negotiations pertain to the negotiation or renegotiation of a basic collective bargaining agreement.

Before issuing an interpretation on the above, the Authority, pursuant to 5 CFR 2410.6 (1978) and section 7135(b) of the Statute (92 Stat. 1215), solicits your views in writing. You are further invited to address the impact, if any, of section 7135(a)(1) of the Statute (92 Stat. 1215) on the above matters and to submit your views as to whether oral argument should be granted. To receive consideration, such views must be submitted to the Authority by the close of business on August 24, 1979.

Issued, Washington, D.C., July 13, 1979.

Federal Labor Relations Authority.

Ronald W. Haughton,

Chairman.

Henry B. Frazier III,

Member.

[FR Doc. 79-22440 Filed 7-19-79; 8:45 am]

BILLING CODE 6325-01-M

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MOU number: 225-79-2001

Memorandum of Understanding

Between
The Environmental Protection Agency

and

The Food and Drug Administration

I. Purpose:

This Memorandum of Understanding establishes an agreement between the Environmental Protection Agency (EPA) and the Food and Drug Administration (FDA) with regard to the control of direct and indirect additives to and substances in drinking water.

EPA and FDA agree:

A. That contamination of drinking water from the use and application of direct and indirect additives and other substances poses a potential public health problem;

B. That the scope of the additives problem in terms of the health significance of these contaminants in drinking water is not fully known;

C. That the possibility of overlapping jurisdiction between EPA and FDA with respect to control of drinking water additives has been the subject of Congressional as well as public concern;

D. That the authority to control the use and application of direct and indirect additives to and substances in drinking water should be vested in a single regulatory agency to avoid duplicative and inconsistent regulation;

E. That EPA has been mandated by Congress under the Safe Drinking Water Act (SDWA), as amended, to assure that the public is provided with safe drinking water;

F. That EPA has been mandated by Congress under the Toxic Substances Control Act (TSCA) to protect against unreasonable risks to health and the environment from toxic substances by requiring, inter alia, testing and necessary restrictions on the use, manufacture, processing, distribution, and disposal of chemical substances and mixtures;

G. That EPA has been mandated by Congress under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended, to assure, inter alia, that when used properly, pesticides will perform their intended function without causing unreasonable adverse effects on the environment; and,

H. That FDA has been mandated by Congress under the Federal Food, Drug, and

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Cosmetic Act (FFDCA), as amended, to protect the public from, inter alia, the adulteration of food by food additives and poisonous and deleterious substances.

It is the intent of the parties that:

A. EPA will have responsibility for direct and indirect additives to and other substances in drinking water under the SDWA, TSCA, and FIFRA; and,

B. FDA will have responsibility for water, and substances in water, used in food and for food processing and responsibility for bottled drinking water under the FFDCA.

II. Background:

A. FDA Legal Authority

"Food" means articles used for food or drink for man or other animals and components of such articles. (FFDCA Section 201(f)). Under Section 402, inter alia, a food may not contain any added poisonous or deleterious substance that may render it injurious to health, or be prepared, packed or handled under unsanitary conditions. Tolerances may be set, under Section 406, limiting the quantity of any substance which is required for the production of food or cannot be avoided in food. FDA has the authority under Section 409 to issue food additive regulations approving, with or without conditions, or denying the use of a "food additive." That term is defined in Section 201(s) to include any substance the intended use of which results or may reasonable be expected to result, directly or indirectly, in its becoming a component or otherwise affecting the characteristics of any food, if such substance is not generally recognized as safe.

In the past, FDA has considered drinking water to be a food under Section 201(f). However, both parties have determined that the passage of the SDWA in 1974 implicitly repealed FDA's authority under the FFDCA over water used for drinking water purposes. Under the express provisions of Section 410 of the FFDCA, FDA retains authority over bottled drinking water. Furthermore, all water used in food remains a food and subject to the provisions of the FFDCA. Water used for food processing is subject to applicable provisions of FFDCA. Moreover, all substances in water used in food are added substances subject to the provisions of the FFDCA, but no substances added to a public drinking water system before the water enters a food processing establishment will be considered a food additive.

B. EPA Legal Authority

The SDWA grants EPA the authority to control contaminants in drinking water which may have any adverse effect on the public health, through the establishment of maximum contaminant levels (MCLs) or treatment techniques, under Section 1412, which are applicable to owners and operators of public water systems. The expressed intent of the Act was to give EPA exclusive control over the safety of public water supplies. Public water systems may also be required by regulation to conduct monitoring for unregulated contaminants under Section 1445 and to issue public notification of such levels under Section 1414(c).

EPA's direct authority to control additives to drinking water apart from the existence of maximum contaminant levels or treatment techniques is limited to its emergency powers under Section 1431. However, Section 1442(b) of the Act authorizes EPA to "collect and make available information pertaining to research, investigations, and demonstrations with respect to providing a dependably safe supply of drinking water together with appropriate recommendations therewith."

TSCA gives EPA authority to regulate chemical substances, mixtures and under some circumstances, articles containing such substances or mixtures. Section 4 permits EPA

to require testing of a chemical substance or mixture based on possible unreasonable risk of injury to health or the environment, or on significant or substantial human or environmental exposure while Section 8 enables EPA to require submission of data showing substantial risk of injury to health or the environment, existing health and safety studies, and other data. For new chemical substances, and significant new uses of existing chemical substances, Section 5 requires manufacturers to provide EPA with pre-manufacturing notice. Under Section 6 the manufacture, processing, distribution, use, and disposal of a chemical substance or mixture determined to be harmful may be restricted or banned. Although Section 3(2)(B) of TSCA excludes from the definition of "chemical substance" food and food additives as defined under FFDCA, the implicit repeal by the SDWA of FDA's authority over drinking water enables EPA to regulate direct and indirect additives to drinking water as chemical substances and mixtures under TSCA.

The FIFRA requires EPA to set restrictions on the use of pesticides to assure that when used properly, they will not cause unreasonable adverse effects on the environment. EPA may require, inter alia labeling which specifies how, when, and where a pesticide may be legally used. In addition, EPA has, under Section 409 of the FFDCA, required FIFRA registrants at times to obtain a food additive tolerance before using a pesticide in or around a drinking water source. Such tolerances establish further restrictions on the use of a pesticide which are enforceable against the water supplier as well as the registrant of the pesticide.

III. Terms of Agreement:

A. EPA's responsibilities are as follows:

1. To establish appropriate regulations, and to take appropriate measures, under the SDWA and/or TSCA, and FIFRA, to control direct additives to drinking water (which encompass any substances purposely added to the water), and indirect additives (which encompass any substance which might leach from paints, coatings or other materials as an incidental result of drinking water contact), and other substances.

2. To establish appropriate regulations under the SDWA to limit the concentrations of pesticides in drinking water; the limitations on concentrations and types of pesticides in water are presently set by EPA through tolerances under Section 409 of the FFDCA.

3. To continue to provide technical assistance in the form of informal advisory opinions on drinking water additives under Section 1442(b) of the SDWA.

4. To conduct and require research and monitoring and the submission of data relative to the problem of direct and indirect additives in drinking water in order to accumulate data concerning the health risks posed by the presence of these contaminants in drinking water.

B. FDA's responsibilities are as follows:

1. To take appropriate regulatory action under the authority of the FFDCA to control bottled drinking water and water, and substances in water, used in food and for food processing.

2. To provide assistance to EPA to facilitate the transition of responsibilities, including:

a) To review existing FDA approvals in order to identify their applicability to additives in drinking water.

b) To provide a mutually agreed upon level of assistance in conducting literature searches related to toxicological decision making.

c) To provide a senior toxicologist to help EPA devise new procedures and protocols to be used in formulating advice on direct and indirect additives to drinking water.

IV. Duration of Agreement:

This Memorandum of Understanding shall continue in effect unless modified by mutual consent of both parties or terminated by either party upon thirty (30) days advance written notice to the other.

This Memorandum of Understanding will become effective on the date of the last signature.

Approved and Accepted for the Environmental Protection Agency

Signed by: Douglas P. Costle
Administrator
Environmental Protection Agency

Date: June 12, 1979

Approved and Accepted for the Food and Drug Administration

Signed by: Donald Kennedy
Administrator
Food and Drug Administration

Date: June 22, 1979

Domestic MOUs

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FDA Website Management Staff

§ 321. Definitions; Generally.

Archive

United States Statutes

Title 21. Food and Drugs

Chapter 9. FEDERAL FOOD, DRUG, AND COSMETIC ACT

Subchapter II. DEFINITIONS

Current through P.L. 111-290

§ 321. Definitions; Generally

For the purposes of this chapter-

(a) (1) The term "State", except as used in the last sentence of section **372 (a)** of this title, means any State or Territory of the United States, the District of Columbia, and the Commonwealth of Puerto Rico.

(2) The term "Territory" means any Territory or possession of the United States, including the District of Columbia, and excluding the Commonwealth of Puerto Rico and the Canal Zone.

(b) The term "interstate commerce" means

(1) commerce between any State or Territory and any place outside thereof, and

(2) commerce within the District of Columbia or within any other Territory not organized with a legislative body.

(c) The term "Department" means Department of Health and Human Services.

(d) The term "Secretary" means the Secretary of Health and Human Services.

(e) The term "person" includes individual, partnership, corporation, and association.

(f) The term "food" means

(1) articles used for food or drink for man or other animals,

(2) chewing gum, and

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(3) articles used for components of any such article.

(g) (1) The term "drug" means

(A) articles recognized in the official United States Pharmacopoeia, official Homoeopathic Pharmacopoeia of the United States, or official National Formulary, or any supplement to any of them; and

(B) articles intended for use in the diagnosis, cure, mitigation, treatment, or prevention of disease in man or other animals; and

(C) articles (other than food) intended to affect the structure or any function of the body of man or other animals; and

(D) articles intended for use as a component of any article specified in clause (A), (B), or (C). A food or dietary supplement for which a claim, subject to sections **343 (r)(1)(B)** and **343 (r)(3)** of this title or sections **343 (r)(1)(B)** and **343 (r)(5)(D)** of this title, is made in accordance with the requirements of section **343 (r)** of this title is not a drug solely because the label or the labeling contains such a claim. A food, dietary ingredient, or dietary supplement for which a truthful and not misleading statement is made in accordance with section **343 (r)(6)** of this title is not a drug under clause (C) solely because the label or the labeling contains such a statement.

(2) The term "counterfeit drug" means a drug which, or the container or labeling of which, without authorization, bears the trademark, trade name, or other identifying mark, imprint, or device, or any likeness thereof, of a drug manufacturer, processor, packer, or distributor other than the person or persons who in fact manufactured, processed, packed, or distributed such drug and which thereby falsely purports or is represented to be the product of, or to have been packed or distributed by, such other drug manufacturer, processor, packer, or distributor.

(h) The term "device" (except when used in paragraph (n) of this section and in sections **331 (i)**, **343 (f)**, **352 (c)**, and **362 (c)** of this title) means an instrument, apparatus, implement, machine, contrivance, implant, in vitro reagent, or other similar or related article, including any component, part, or accessory, which is-

(1) recognized in the official National Formulary, or the United States Pharmacopeia, or any supplement to them,

(2) intended for use in the diagnosis of disease or other conditions, or in the cure, mitigation, treatment, or prevention of disease, in man or other animals, or

(3) intended to affect the structure or any function of the body of man or other animals, and

which does not achieve its primary intended purposes through chemical action within or on the body of man or other animals and which is not dependent upon being metabolized for the achievement of its primary intended

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(dd) For purposes of sections **335a** and **335b** of this title, the term "drug product" means a drug subject to regulation under section **355**, **360b**, or **382** of this title or under section **262** of title 42.

(ee) The term "Commissioner" means the Commissioner of Food and Drugs.

(ff) The term "dietary supplement"-

(1) means a product (other than tobacco) intended to supplement the diet that bears or contains one or more of the following dietary ingredients:

(A) a vitamin;

(B) a mineral;

(C) an herb or other botanical;

(D) an amino acid;

(E) a dietary substance for use by man to supplement the diet by increasing the total dietary intake; or

(F) a concentrate, metabolite, constituent, extract, or combination of any ingredient described in clause (A), (B), (C), (D), or (E);

(2) means a product that-

(A)

(i) is intended for ingestion in a form described in section **350 (c)(1)(B)(i)** of this title; or

(ii) complies with section **350 (c)(1)(B)(ii)** of this title;

(B) is not represented for use as a conventional food or as a sole item of a meal or the diet; and

(C) is labeled as a dietary supplement; and

(3) does-

(A) include an article that is approved as a new drug under section **355** of this title or licensed as a biologic under section **262** of title 42 and was, prior to such approval, certification, or license, marketed as a dietary supplement or as a food unless the Secretary has issued a regulation, after notice and comment, finding that the article, when used as or in a dietary supplement under the conditions of use and dosages set forth in the labeling for such dietary supplement, is unlawful under section **342 (f)** of

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this title; and

(B) not include-

(i) an article that is approved as a new drug under section **355** of this title, certified as an antibiotic under section **357** of this title, or licensed as a biologic under section **262** of title 42, or

(ii) an article authorized for investigation as a new drug, antibiotic, or biological for which substantial clinical investigations have been instituted and for which the existence of such investigations has been made public,

which was not before such approval, certification, licensing, or authorization marketed as a dietary supplement or as a food unless the Secretary, in the Secretary's discretion, has issued a regulation, after notice and comment, finding that the article would be lawful under this chapter.

Except for purposes of paragraph (g) and section 350f of this title, a dietary supplement shall be deemed to be a food within the meaning of this chapter.

(gg) The term "processed food" means any food other than a raw agricultural commodity and includes any raw agricultural commodity that has been subject to processing, such as canning, cooking, freezing, dehydration, or milling.

(hh) The term "Administrator" means the Administrator of the United States Environmental Protection Agency.

(ii) The term "compounded positron emission tomography drug"-

(1) means a drug that-

(A) exhibits spontaneous disintegration of unstable nuclei by the emission of positrons and is used for the purpose of providing dual photon positron emission tomographic diagnostic images; and

(B) has been compounded by or on the order of a practitioner who is licensed by a State to compound or order compounding for a drug described in subparagraph (A), and is compounded in accordance with that State's law, for a patient or for research, teaching, or quality control; and

(2) includes any nonradioactive reagent, reagent kit, ingredient, nuclide generator, accelerator, target material, electronic synthesizer, or other apparatus or computer program to be used in the preparation of such a drug.

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DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Food and Drug Administration
Rockville MD 20857

DEC 21 2000

The Honorable Ken Calvert
Chairman
Subcommittee on Energy and Environment
Committee on Science
House of Representatives
Washington, D.C. 20515-6301

Dear Mr. Chairman:

Thank you for the letter of May 8, 2000, to Dr. Jane E. Henney, Commissioner of Food and Drugs, regarding the use of fluoride in drinking water and drug products. We apologize for the delay in responding to you.

We have restated each of your questions, followed by our response.

1. **If health claims are made for fluoride-containing products (e.g. that they reduce dental caries incidence or reduce pathology from osteoporosis), do such claims mandate that the fluoride-containing product be considered a drug, and thus subject the product to applicable regulatory controls?**

Fluoride, when used in the diagnosis, cure, mitigation, treatment, or prevention of disease in man or animal, is a drug that is subject to Food and Drug Administration (FDA) regulation. FDA published a final rule on October 6, 1995, for anticaries drug products for over-the-counter (OTC) human use (copy enclosed). This rule establishes the conditions under which OTC anticaries drug products are generally recognized as safe and effective and not misbranded. The rule has provisions for active ingredients, packaging conditions, labeling, and testing procedures that are required by manufacturers in order to market anticaries products. A new drug application (NDA) may be filed for a product containing fluoride that does not meet the provisions stated in the final rule. As you know, the Environmental Protection Agency regulates fluoride in the water supply.

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2. Are there any New Drug Applications (NDA) on file, that have been approved, or that have been rejected, that involve a fluoride-containing product (including fluoride-containing vitamin products) intended for ingestion with the stated aim of reducing dental caries? If any such NDA's have been rejected, on what grounds were they rejected? If any such NDA have been approved, please provide the data on safety and efficacy that FDA found persuasive.

No NDAs have been approved or rejected for fluoride drugs meant for ingestion. Several NDAs have been approved for fluoride topical products such as dentifrices and gels. Fluoride products in the form of liquid and tablets meant for ingestion were in use prior to enactment of the Kefauver-Harris Amendments (Drug Amendments of 1962) to the Food, Drug, and Cosmetic Act in which efficacy became a requirement, in addition to safety, for drugs marketed in the United States (U.S.). Drugs in use prior to 1962 are being reviewed under a process known as the drug efficacy study implementation (DESI). The DESI review of fluoride-containing products has not been completed.

3. Does FDA consider dental fluorosis a sign of over exposure to fluoride?

Dental fluorosis is indicative of greater than optimal ingestion of fluoride. In 1988, the U.S. Surgeon General reported that dental fluorosis, while not a desirable condition, should be considered a cosmetic effect rather than an adverse health effect. Surgeon General M. Joycelyn Elders reaffirmed this position in 1994.

4. Does FDA have any action-level or other regulatory restriction or policy statement on fluoride exposure aimed at minimizing chronic toxicity in adults or children?

The monograph for OTC anticaries drug products sets acceptable concentrations for fluoride dentifrices, gels and rinses (all for topical use only). This monograph also describes the acceptable dosing regimens and labeling including warnings and directions for use. FDA's principal safety concern regarding fluoride in OTC drugs is the incidence of fluorosis in

children. Children under two years of age do not have control of their swallowing reflex and do not have the skills to expectorate toothpaste properly. Young children are most susceptible to mild fluorosis as a result of improper use and swallowing of a fluoride toothpaste. These concerns are addressed in the monograph by mandating maximum concentrations, labeling that specifies directions for use and age restrictions, and package size limits.

Thanks again for contacting us concerning this matter. If you have further questions, please let us know.

Sincerely,

Melinda K. Plaisier
Associate Commissioner
for Legislation

Enclosure
"Final Rule/Federal Register - October 6, 1995
Over-the-Counter Anticaries Drug Products"

Web site administrator's note:
To perform query to access this document
Enter: http://www.access.gpo.gov/su_docs/aces/aces140.html
Enter: checkmark for 1995 Volume 60
Enter: On: 10/06/95
Enter: Search terms: anticaries

Fluorosilicic Acid

Fluorosilicic Acid (Hydrofluorosilicic Acid, HFS, FSA)

Technical Data Sheet

CHEMICAL ANALYSIS	SPECIFICATION	TYPICAL ANALYSIS
H_2SiF_6, %	23-25	23.5
Heavy Metals (as Pb), %		< 0.02
HF, %	1.0 max	0.5
Color, APHA	100 max	< 20
P_2O_5, %		< 0.2

Product meets ANSI/AWWA Standard B703-06, and is certified by NSF International or Classified by UL to ANSI/NSF Standard 60. Maximum use level for potable water treatment is 6.0 mg/L.

PHYSICAL PROPERTIES

Physical Description	Aqueous solution, water white to straw-yellow, corrosive acid, irritating to skin and having pungent odor.
Molecular Weight	144.08
Specific Gravity 23% solution @ 75°F	1.212
Boiling Point of Aqueous 23% Solution	221°F (Decomposes)
Freezing Point of Aqueous 23% Solution	5°F (approx.)
Freezing Point of Aqueous 25% Solution	-4°F
pH of 1%, H_2SiF_6	1.2

CONTAINERS

Tank truck, rubber or plastic-lined	40,000 lb (approx.)
Tank car, rubber or plastic-lined	196,000 lb net (approx.)

DOT AND FREIGHT DESCRIPTION

Hazardous Material Description	Fluorosilicic acid
Haz. Mat. Class, I.D.#, Packaging Group	8, UN 1778, PG II
Freight Classification	Hydrofluorosilicic Acid
Principal CAS Number	16961-83-4
RQ	None
Placard	Corrosive
Label	Corrosive

Responsible Care®
Good Chemistry at Work

CGR#3323 HFS-0205 Revised 0707

www.solvaychemicals.us 1.800.765.8292

SOLVAY Fluorides

SOLVAY

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Fluorosilicic Acid

Fluorosilicic Acid (Hydrofluorosilicic Acid, HFS, FSA)

Technical Data Sheet

Use in public Water Treating Plants:

The reduction in dental caries by adjusting the fluoride content of public water supplies is a matter of common knowledge today, half a century following the first installation in Grand Rapids, Michigan. Approximately 170 million people in over three thousand communities are now drinking fluoride-treated water from water purification plants where fluoridation is currently practiced. Fluoridation is concerned with the controlled introduction to water of the fluoride ion. Other materials in the fluoride compound simultaneously introduced into the water with the fluoride ions are carriers which provide no benefits and are nontoxic. The addition of one part per million of fluoride requires that the product be soluble, of definite concentration and have high purity standards. In conformity with the American Water Works Association standard B703-94, the term fluorosilicic acid has replaced the more technical designation of hydrofluosilicic acid. After the original work with sodium fluoride proved the effectiveness of fluoride on tooth health and a broad fluoridation program was envisaged, new sources of fluoride and economics of their use were investigated. Fluorosilicic acid is a high purity source of fluoride. It is simpler to use than any other chemical approved for water fluoridation purposes, primarily because it is a liquid and can therefore be accurately measured and fed with a minimum of equipment. In contrast to powdered or granular chemicals, it presents no dust problems, no measuring problems and handling requires a minimum of labor. Today most of the large cities and many small ones are fluoridating with fluorosilicic acid. It is readily available in tank cars or tank trucks and can also be supplied in 15-gallon carboys and 55-gallon drums. The addition of fluorosilicic acid to a water supply can be readily controlled to give a total fluoride (F) level of one part per million which has been established as effective for reducing tooth decay. It should be used in accordance with procedures approved by each state's department of health.

Acid Characteristics:

Fluorosilicic acid is a transparent, clear to straw-colored, corrosive liquid having the chemical formula of H_2SiF_6. It is manufactured in modern rubber-lined equipment producing an acid of high commercial purity. Commercial water solutions of the acid are available, having concentration of between 23% and 25% H_2SiF_6. Fluorosilicic acid is generally believed not to exist in the vapor phase, but only in solution. Upon vaporizing, it decomposes into hydrofluoric acid (HF) and silicon tetrafluoride. This equilibrium exists at the surface of strong solutions of fluorosilicic acid and if stored in glass containers, the small concentration of hydrofluoric acid may very slowly attack the glass above the solution level. For this reason, it is generally shipped in polyethylene containers rather than glass carboys. A 23% fluorosilicic acid-water solution weighs 10.1 pounds per gallon at 75°F, and has a fluoride (F) content of 18.20%.

CGR#3323 HFS-0205 Revised 0707

www.solvaychemicals.us 1.800.765.8292

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Fluorosilicic Acid (Hydrofluorosilicic Acid, HFS, FSA)

Technical Data Sheet

Installation:
In a typical large plant installation, rubber-lined vented storage tanks are usually mounted outside the building with the tanks ranging in size from 4,500 to 6,500 gallon capacities. These tanks, equipped with recording level gauges, feed the acid through plastic piping or tubing to the dosage unit. Feeding is regulated by controlled volume pumps. Metering is used for accurate flow records. Fluorosilicic acid may be handled in rubber-lined, saran or other available corrosive-resistant equipment as suggested below:

Pipes and lines	-	rubber, saran or polyethylene
Pumps	-	Lucite, saran or Hastelloy
Valves	-	rubber-lined or polyethylene-lined
Tanks	-	rubber-lined, saran or polyethylene-lined

Acid should be pumped by positive diaphragm proportioning pumps.

Operation procedure:
The drum or drums of fluorosilicic acid should be mounted on a platform of sufficient size and capacity to permit weighing the amount used each day. Proportioning pumps deliver an accurate volume, but for small pumping rates, the dosage may be more satisfactorily regulated by periodic weighing of the drum. Whenever a drum of fluorosilicic acid is replaced on the scale, the time and weight should be recorded in the daily operating log. Whenever dosage is changed to a varying pumpage, the time and feeder setting should be recorded in the daily log.

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SHANGHAI MINTCHEM DEVELOPMENT CO., LTD

Specification Sheet

Sodium Fluoride

Physical Properties:

Formula	**Na F**	**Molecular Weight**	**41.99**	**CAS NO**	**7681-49-4**
U.N-NO	**1690**	**Class**	**6.1**	**H.S-NO**	**2826110010**

Character: White crystal or powder. Relative Density 2.558. It's odorless. Soluble in water and HF. Insoluble in ethanol. Mellting point 993ºC and boiling point 1695ºC. Non flammable but toxic.

Chemical Parameters:

NO.	Technological Specification	Granular Standard%		Powder Standard%	
1	NaF purity	98.5%min		98.5%min	
2	Sodium Carbonate	0.5%max.		0.5%max.	
3	Na2SiF6	1.5%max		1.5%max	
4	Silicon Dioxide	0.5%max.		0.5%max.	
5	Sulphate	0.3%max.		0.3%max.	
6	HF	0.1%max,		0.1%max,	
7	H2O(moisture)	0.5%max.		0.5%max.	
8	Heavy Metal(As Pb)	0.04%max.		0.04%max.	
9	Available Fluoride	43.8%min.		43.8%min.	
10	Water Insoluble matter	0.6%max.		0.6%max.	
11	Particle Size	-20 mesh	98%min	+80 mesh	4 % max
12		+100 mesh	50%min	+200 mesh	25 % max
13		-325 mesh			

APPLICATION: It is mainly used as fluxing agent, timber preservative and water treatment etc.

PACKAGE: Packing in plastic weaved bag 25kg each. TRANSPORTATION: DG, Class 6.1, UN 1690

MANUFACTURER: SHANGHAI MINTCHEM DEVELOPMENT CO., LTD

上海办 **Shanghai Office**

上海市浦东新区牡丹路 89 弄 4 号 602 室
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mid 1980's[7] (Evans R.W, Stamm JW., 1991). Across all age groups more than 90% of fluorosis cases were very mild or mild. (Evans R.W, Stamm JW., 1991). The study did not include measures of fluoride intake. Concurrently, dental caries prevalence did not increase. (Lo ECM *et al,* 1990). Although not fully generalizable to the current U.S. context, these findings, along with those from the 1986–87 survey of U.S. schoolchildren, suggest that risk of fluorosis can be reduced and caries prevention maintained toward the lower end (*i.e.,* 0.7 mg/L) of the 1962 USPHS recommendations for fluoride concentrations for community water systems.

Relationship of fluid intake and ambient temperature among children and adolescents in the United States:

The 1962 USPHS recommendations stated that community drinking water should contain 0.7–1.2 mg/L [ppm] fluoride, depending on the ambient air temperature of the area. These temperature-related guidelines were based on studies conducted in two communities in California in the early 1950's. Findings indicated that a lower fluoride concentration was appropriate for communities in warmer climates because children drank more tap water on warm days (Galagan DJ, 1953; Galagan DJ and Vermillion JR, 1957; Galagan DJ *et al,* 1957). Social and environmental changes, including increased use of air conditioning and more sedentary lifestyles, have occurred since the 1950's, and thus, the assumption that children living in warmer regions drink more tap water than children in cooler regions may no longer be valid.

Studies conducted since 2001 suggest that fluid intake in children does not increase with increases in ambient air temperature (Sohn W, *et al,* 2001; Beltrán-Aguilar ED, *et al,* 2010b). One study conducted among children using nationally representative data from 1988 to 1994 did not find an association between fluid intake and ambient air temperature (Sohn W, *et al,* 2001). A similar study using nationally representative data from 1999 to 2004 also found no association between fluid intake and ambient temperature among children or adolescents (Beltrán-Aguilar ED, *et al,* 2010b). These recent findings demonstrating a lack of an association between fluid intake among children and adolescents and ambient temperature support use of a single target concentration for community water fluoridation in all temperature zones of the United States.

Conclusions

HHS recommends an optimal fluoride concentration of 0.7 mg/L for community water systems based on the following information:

- Community water fluoridation is the most cost-effective method of delivering fluoride for the prevention of tooth decay;
- In addition to drinking water, other sources of fluoride exposure have contributed to the prevention of dental caries and an increase in dental fluorosis prevalence;
- Significant caries preventive benefits can be achieved and risk of fluorosis reduced at 0.7 mg/L, the lowest concentration in the range of the USPHS recommendation.
- Recent data do not show a convincing relationship between fluid intake and ambient air temperature. Thus, there is no need for different recommendations for water fluoride concentrations in different temperature zones.

Surveillance Activities

CDC and the National Institute of Dental and Craniofacial Research (NIDCR), in coordination with other Federal agencies, will enhance surveillance of dental caries, dental fluorosis, and fluoride intake with a focus on younger populations at higher risk of fluorosis to obtain the best available and most current information to support effective efforts to improve oral health.

Process

The U.S. Department of Health and Human Services (HHS) convened a Federal inter-departmental, inter-agency panel of scientists (Appendix A) to review scientific evidence related to the 1962 USPHS Drinking Water Standards related to recommendations for fluoride concentrations in drinking water in the United States and to update these proposed recommendations. Panelists included representatives from the Centers for Disease Control and Prevention, the National Institutes of Health, the Food and Drug Administration, the Agency for Healthcare Research and Quality, the Office of the Assistant Secretary for Health, the U.S. Environmental Protection Agency, and the U.S. Department of Agriculture. The panelists evaluated existing recommendations for fluoride in drinking water, systematic reviews of the risks and benefits from fluoride in drinking water, the epidemiology of dental caries and fluorosis in the U.S., and current data on fluid intake in children, aged 0 to 10 years, across temperature gradients in the U.S. Conclusions were reached and are summarized along with their rationale in this proposed guidance document. This guidance will be advisory, not regulatory, in nature. Guidance will be submitted to the **Federal Register** and will undergo public and stakeholder comment for 30 days, after which HHS will review comments and consider changes.

Dated: January 7, 2011.

Kathleen Sebelius,

Secretary.

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[7] Fluorosis prevalence ranged from 64% (SE = 4.1) to 47% (SE = 4.5) based on the upper right central incisor only.

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STATE OF WASHINGTON

WASHINGTON STATE BOARD OF HEALTH

PO Box 47990 • Olympia, Washington 98504-7990

November 16, 2010

Mr. William Osmunson, DDS, MPH, President
Washington Action for Safe Water
1418 – 112th Ave NE, Suite 200
Bellevue, WA 98004

Dear Dr. Osmunson:

This letter provides formal notice that the Washington State Board of Health has denied your petition for rule making received on October 7, 2010 to add an intent statement in two places in WAC 246-290-460, regarding water fluoridation. The suggested statement was "with the intent to prevent dental caries." This was the fifth petition for rule making you submitted to the Board this year regarding this rule.

The Board's intent for setting an "optimal" fluoride concentration in WAC 246-290-460 is part of its requirement to "adopt rules for group A public water systems...to assure safe and reliable public drinking water and to protect the public health" under RCW 43.20.050(2)(a). The Board follows guidelines of the Centers for Disease Control and Prevention (CDC) regarding setting an appropriate level of fluoride in drinking water if the directors of a water system decide to fluoridate under the authority of RCW 57.08.012. The CDC promotes community water fluoridation as one of the ten great public health achievements of the twentieth century. It says fluoridation is the single most effective public health measure to prevent tooth decay. The Board supports this and other positions of the CDC. The Board considers it self evident that the purpose of water fluoridation is to help prevent tooth decay. The Board does not consider it efficient use of public resources to initiate and complete a rule making process to add to the rule the language requested by the petitioner.

The Board handled your request as a petition for rule making under RCW 34.05.330 and Board Policy 2005-001, Responding to Petitions for Rule Making. The statute requires the Board to respond within 60 days of receipt. RCW 34.05.330(3) allows a person to appeal a petition's denial to the Governor within 30 days. The Board's policy allows the Board Chair to respond to a petition for rule making without the petition being placed on a meeting agenda for full Board consideration. If you have questions about this decision, please contact Craig McLaughlin, Executive Director of the Board, at 360-236-4106 or craig.mclaughlin@doh.wa.gov.

Sincerely,

Keith Higman
Chair

cc: Michelle Davis, Department of Health
Gregg Grunenfelder, Department of Health
State Board of Health Members

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CDC
CENTERS FOR DISEASE CONTROL AND PREVENTION

August 17, 2001 / Vol. 50 / No. RR-14

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WEEKLY REPORT

Recommendations and Reports

Recommendations for Using Fluoride to Prevent and Control Dental Caries in the United States

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Centers for Disease Control and Prevention (CDC)
Atlanta, GA 30333

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Recommendations for Using Fluoride to Prevent and Control Dental Caries in the United States

Summary

Widespread use of fluoride has been a major factor in the decline in the prevalence and severity of dental caries (i.e., tooth decay) in the United States and other economically developed countries. When used appropriately, fluoride is both safe and effective in preventing and controlling dental caries. All U.S. residents are likely exposed to some degree to fluoride, which is available from multiple sources. Both health-care professionals and the public have sought guidance on selecting the best way to provide and receive fluoride. During the late 1990s, CDC convened a work group to develop recommendations for using fluoride to prevent and control dental caries in the United States. This report includes these recommendations, as well as a) critical analysis of the scientific evidence regarding the efficacy and effectiveness of fluoride modalities in preventing and controlling dental caries, b) ordinal grading of the quality of the evidence, and c) assessment of the strength of each recommendation.

Because frequent exposure to small amounts of fluoride each day will best reduce the risk for dental caries in all age groups, the work group recommends that all persons drink water with an optimal fluoride concentration and brush their teeth twice daily with fluoride toothpaste. For persons at high risk for dental caries, additional fluoride measures might be needed. Measured use of fluoride modalities is particularly appropriate during the time of anterior tooth enamel development (i.e., age <6 years).

The recommendations in this report guide dental and other health-care providers, public health officials, policy makers, and the public in the use of fluoride to achieve maximum protection against dental caries while using resources efficiently and reducing the likelihood of enamel fluorosis. The recommendations address public health and professional practice, self-care, consumer product industries and health agencies, and further research. Adoption of these recommendations could further reduce dental caries in the United States and save public and private resources.

INTRODUCTION

Dental caries (i.e., tooth decay) is an infectious, multifactorial disease afflicting most persons in industrialized countries and some developing countries (*1*). Fluoride reduces the incidence of dental caries and slows or reverses the progression of existing lesions (i.e., prevents cavities). Although pit and fissure sealants, meticulous oral hygiene, and appropriate dietary practices contribute to caries prevention and control, the most effective and widely used approaches have included fluoride use. Today, all U.S. residents are exposed to fluoride to some degree, and widespread use of fluoride has been a major factor in the decline in the prevalence and severity of dental caries in the United States and other economically developed countries (*1*). Although this decline is a major public

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Fluoridated Drinking Water and Processed Beverages and Food

Fluoridated drinking water contains a fluoride concentration effective for preventing dental caries; this concentration can occur naturally or be reached through water fluoridation, which is the controlled addition of fluoride to a public water supply. When fluoridated water is the main source of drinking water, a low concentration of fluoride is routinely introduced into the mouth. Some of this fluoride is taken up by dental plaque; some is transiently present in saliva, which serves as a reservoir for plaque fluoride; and some is loosely held on the enamel surfaces (*76*). Frequent consumption of fluoridated drinking water and beverages and food processed in fluoridated areas maintains the concentration of fluoride in the mouth.

Estimates of fluoride intake among U.S. and Canadian adults have ranged from ≤1.0 mg fluoride per day in nonfluoridated areas to 1–3 mg fluoride per day in fluoridated areas (*77–80*). The average daily dietary fluoride intake for both children and adults in fluoridated areas has remained relatively constant for several years (*11*). For children who live in optimally fluoridated areas, this average is approximately 0.05 mg/kg/day (range: 0.02–0.10); for children who live in nonfluoridated areas, the average is approximately half (*11*). In a survey of four U.S. cities with different fluoride concentrations in the drinking water (range: 0.37–1.04 ppm), children aged 2 years ingested 0.41–0.61 mg fluoride per day and infants aged 6 months ingested 0.21–0.54 mg fluoride per day (*81,82*).

In the United States, water and processed beverages (e.g., soft drinks and fruit juices) can provide approximately 75% of a person's fluoride intake (*83*). Many processed beverages are prepared in locations where the drinking water is fluoridated. Foods and ingredients used in food processing vary in their fluoride content (*11*). As consumption of processed beverages by children increases, fluoride intake in communities without fluoridated water will increase whenever the water source for the processed beverage is fluoridated (*84*). In fluoridated areas, dietary fluoride intake has been stable because processed beverages have been substituted for tap water and for beverages prepared in the home using tap water (*11*).

A study of Iowa infants estimated that the mean fluoride intake from water during different periods during the first 9 months of life, either consumed directly or added to infant formula or juice, was 0.29–0.38 mg per day, although estimated intake for some infants was as high as 1.73 mg per day (*85*). As foods are added to an infant's diet, replacing some of the formula prepared with fluoridated water, the amount of fluoride the infant receives typically decreases (*86*). The Iowa study also reported that infant formula and processed baby food contained variable amounts of fluoride. Since 1979, U.S. manufacturers of infant formula have voluntarily lowered the fluoride concentration of their products, both ready-to-feed and concentrates, to <0.3 ppm fluoride (*87*).

Drinking Water

Community Water. During the 1940s, researchers determined that 1 ppm fluoride was the optimal concentration in community drinking water for climates similar to the Chicago area (*88,89*). This concentration would substantially reduce the prevalence of dental caries, while allowing an acceptably low prevalence (i.e., 10%–12%) of very mild and mild enamel fluorosis and no moderate or severe enamel fluorosis. Water fluoridation for caries control began in 1945 and 1946, when the fluoride concentration was

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U.S. Food and Drug Administration

Regulatory Information

Significant Amendments to the FD&C Act

Significant Amendments to the FD&C Act:

Since 1980, listed chronologically; date shown is when the Public Law was approved. "Summary" indicates link to a summary of the law; other links are to full text. Provisions of these Public Laws are incorporated into the FD&C Act.

- Infant Formula Act of 1980 (summary)[1]
  Public Law (PL) 96-359 (Oct. 26, 1980)
- Orphan Drug Act[2]
  PL 97-414 (Jan. 4, 1983)
- Drug Price Competition and Patent Term Restoration Act of 1984 (summary)[3]
  PL 98-417 (Sept. 24, 1984)
- Prescription Drug Marketing Act of 1987[4]
  PL 100-293 (Apr. 22, 1988)
- Generic Animal Drug and Patent Term Restoration Act of 1988 (summary)[5]
  PL 100-670 (Nov. 16, 1988)
- Nutrition Labeling and Education Act of 1990 (summary)[6]
  PL 101-535 (Nov. 8, 1990)
- Safe Medical Devices Act of 1990 (summary)[7]
  PL 101-629 (Nov. 28, 1990)
- Medical Device Amendments of 1992 (summary)[8]
  PL 102-300 (June 16, 1992)
- Prescription Drug Amendments of 1992; Prescription Drug User Fee Act of 1992[9]
  PL 102-571 (Oct. 29, 1992)
- Animal Medicinal Drug Use Clarification Act (AMDUCA) of 1994[10]
  PL 103-396 (Oct. 22, 1994)
- Dietary Supplement Health and Education Act of 1994[11]
  PL 103-417 (Oct. 25, 1994)
- FDA Export Reform and Enhancement Act of 1996[12]
  PL 104-134 (April 26, 1996)
- Food Quality Protection Act of 1996[13]
  PL 104-170 (Aug. 3, 1996)
- Animal Drug Availability Act of 1996[14]
  PL 104-250 (Oct. 9, 1996)
- Food and Drug Administration Modernization Act (FDAMA) of 1997[15]
  PL 105-115 (Nov. 21, 1997)
- Best Pharmaceuticals for Children Act[16]
  PL 107-109 (Jan. 4, 2002)
- Medical Device User Fee and Modernization Act (MDUFMA) of 2002[17]
  PL 107-250 (Oct. 26, 2002)
- Animal Drug User Fee Act of 2003[18]
  PL 108-130 (Nov. 18, 2003)
- Pediatric Research Equity Act of 2003[19]
  PL 108-155 (Dec. 3, 2003)
- Minor Use and Minor Species Animal Health Act of 2004[20]
  PL 108-282 (Aug. 2, 2004)
- Dietary Supplement and Nonprescription Drug Consumer Protection Act[21]
  PL 109-462 (Dec. 22, 2006)
- Food and Drug Administration Amendments Act (FDAAA) of 2007[22]
  PL 110-85 (Sept. 27, 2007)
- Family Smoking Prevention and Tobacco Control Act (Public Law 111-31)[23]
  PL 111-31 (June 22, 2009)
- FDA Food Safety Modernization Act[24]
  PL 111-353 (Jan. 4, 2011)

Links on this page:

1. http://thomas.loc.gov/cgi-bin/bdquery/z?d096:HR06940:@@@L|TOM:/bss/d096query.html|#summary
2. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/OrphanDrugAct/default.htm

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3. http://thomas.loc.gov/cgi-bin/bdquery/z?d098:SN01538:@@@D&summ2=m&|TOM:/bss/d098query.html|
4. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/PrescriptionDrugMarketingActof1987/default.htm
5. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/ucm147135.htm
6. http://thomas.loc.gov/cgi-bin/bdquery/z?d101:HR03562:@@@D&summ2=3&|TOM:/bss/d101query.html|
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11. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/ucm148003.htm
12. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/ucm148005.htm
13. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/ucm148008.htm
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15. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/FDAMA/default.htm
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17. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/MedicalDeviceUserFeeandModernizationActMDUFMAof2002/default.htm
18. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/AnimalDrugUserFeeActof2003/default.htm
19. http://frwebgate.access.gpo.gov/cgi-bin/getdoc.cgi?dbname=108_cong_public_laws&docid=f:publ155.108
20. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/MinorUseandMinorSpeciesAnimalHealthActof2004/default.htm
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22. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/FoodandDrugAdministrationAmendmentsActof2007/default.htm
23. http://www.gpo.gov/fdsys/pkg/PLAW-111publ31/pdf/PLAW-111publ31.pdf
24. /RegulatoryInformation/Legislation/FederalFoodDrugandCosmeticActFDCAct/SignificantAmendmentstotheFDCAct/ucm244718.htm

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NOV 21 2014

Dear Ms. McElheney:

Thank you for your correspondence concerning fluoridation of drinking water. Your letter requests that I take a number of actions related to fluoridation. These include instructing the Food and Drug Administration (FDA) to advise fluoridation manufacturers to submit New Drug Applications; instructing the Centers for Disease Control and Prevention (CDC) to stop "promotion. . . of any and all drugs, including the ingestion of fluoride products, not FDA CDER approved"; sponsoring a review of fluoride's neurotoxicity by the National Research Council; and supporting a prospective randomized control trial of the effectiveness of ingesting hydrofluorosilicic acid.

For nearly 70 years, community water fluoridation (CWF) has been a safe and healthy way to effectively prevent tooth decay. CDC has recognized water fluoridation as one of ten great public health achievements of the 20th century. CDC works with national partners, states, communities, and water operators to ensure that the U.S. population has access to optimally fluoridated water to prevent tooth decay.

However, fluoride ingestion while teeth are developing can result in a range of visually detectable changes in the tooth enamel, called dental fluorosis. The prevalence of mild to moderate dental fluorosis in the United States has increased in recent years. Fluoride in drinking water is one of several available fluoride sources. In 2011, the Department of Health and Human Services (HHS) proposed that the recommended level of fluoride in drinking water be set at 0.7 mg/L. This will reduce the chance for children's teeth to develop dental fluorosis, while still preventing tooth decay. The previous U.S. Public Health Service recommendations for fluoride levels ranged from 0.7mg/L to 1.2 mg/L, depending on average maximum regional air temperature. The new recommendation is based on recent findings that in the U.S., outdoor temperature does not determine water intake.

HHS expects that the final recommendations to reduce the optimal fluoride level will be publicly available soon. CDC, in collaboration with the National Institute of Dental and Craniofacial Research (NIDCR), will monitor the impact of these changes through enhanced surveillance of dental caries (tooth decay) and dental fluorosis in the National Health and Nutrition Examination Survey (NHANES).

Your specific requests are addressed below.

Instruct FDA CDER to no longer defer regulatory action. FDA CDER to send a letter to fluoridation manufacturers advising them to make FDA CDER NDA (New Drug Application) as required by Congress in the US FD&C Act.

FDA has provided the following information regarding your request:

> *FDA has determined that Congress did not intend for FDA to regulate the addition of fluoride to public drinking water for dental caries prevention as a drug under the FD&C Act. Instead, Congress intended that the U.S. Environmental Protection Agency (EPA) regulate fluoride in public drinking water as a potential contaminant under the Safe Drinking Water Act of 1974 (SDWA), Public Law No. 93-523, 88 Stat. 1660 (codified as amended at 42 U.S.C. 300f et seq.) to protect against adverse health effects, and that within the limits thus set by EPA, state and local governments be permitted, but not required, to fluoridate public drinking water to help prevent dental caries. Thus, FDA does not require NDAs for fluoridated public drinking water.*

Instruct the CDC to stop the promotion (internet and education) of any and all drugs, including the ingestion of fluoride products, not FDA CDER approved.

Section 317M of the Public Health Service Act, codified at 42 U.S.C. § 247b-14, authorizes the Secretary of HHS, acting through the Director of the CDC, to make grants to States and Indian tribes for the purpose of increasing the resources available for community water fluoridation. This includes funds to develop educational materials on the benefits of fluoridation. CDC's Division of Oral Health leads an effort to improve the oral health of the nation and reduce inequalities in oral health. This includes encouraging the use of proven strategies to prevent oral disease, such as the effective use of fluoride products and community water fluoridation.

Sponsor a review of the scientific evidence on fluoride's neurotoxicity by the National Academy of Science's National Research Council. The review should include studies listed at www.FluorideAlert.org/issues/health/brain.

The NRC reviewed the toxicity of fluoride as recently as 2006, when it reviewed the Environmental Protection Agency's drinking water standard for fluoride as a contaminant. (See *Fluoride in Drinking Water: A Scientific Review of EPA's Standards.*) More recently and of more relevance to community water fluoridation is the systematic review undertaken by the Community Preventive Services Task Force (Task Force) in 2013. The Task Force is an independent, nonfederal, unpaid panel of public health and prevention experts that provides evidence-based findings and recommendations about community preventive services, programs, and policies to improve health. Its members represent a broad range of research, practice, and policy expertise in community preventive services, public health, health promotion, and disease prevention. In its report, *Preventing Dental Caries: Community Water Fluoridation*, the Task Force noted, "Overall, the body of evidence indicates that Community Water Fluoridation is an effective intervention for reducing caries at the population level. At the optimal fluoride concentration, associated risks are predominantly the milder forms of fluorosis that are only detectable under clinical examination." The report further stated, "In addition, there is no evidence that CWF (Community Water Fluoridation) results in severe dental fluorosis."

Sponsor a quality published independent prospective randomized controlled trial (RTC), of the effectiveness of ingesting hydrofluorosilicic acid (fluoridation), including blood serum and urine concentrations of fluoride.

As stated above, the effectiveness and safety of community water fluoridation was reaffirmed by the Community Preventive Services Task Force in 2013 following a systematic evidence review. Studies on the effectiveness of adjusting fluoride in community water to the optimal concentration cannot be designed as randomized clinical trials. Random allocation of study subjects is not possible when a community begins to fluoridate the water because all residents receiving community water have access to and are exposed to this source of fluoride. Furthermore, clinical studies cannot be conducted double-blind because both study subjects and researchers usually know whether a community's water has been fluoridated. In addition, it would not be possible to find control subjects with no fluoride exposure because fluorides are ubiquitous in the environment.

Although I am not able to fulfill your requests, I appreciate the information you provided to me and my staff. I will keep your concerns in mind as HHS continues to consider community water fluoridation.

A copy of this response is being shared with Dr. Hirzy, Mr. Nidel, Dr. Connett, Ms. Smith, and Dr. Osmunson.

Sincerely,

Wanda K. Jones, DrPH
Principal Deputy Assistant Secretary for Health

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Jill McElheney
Chris Nidel, Nidel Law 1615 New Hampshire Ave., NW, Washington, DC 20009. 202-558-2030
Bill Hirzy PhD Fluoride Action Network
Paul Connett PhD President, Fluoride Action Network
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54 Ponder Point, Sandpoint, Idaho 83864 bill@teachingsmiles.com

September 4, 2014

Wanda Jones
Jonathan Beeton
Office of the Assistant Secretary for Health
U.S. Department of Health and Human Services
Sandra.Howard@HHS.GOV
202-690-7778

For the health and safety of the public:

1. Instruct FDA CDER to no longer defer regulatory action. FDA CDER to send a letter to fluoridation manufacturers advising them to make FDA CDER NDA (New Drug Application) as required by Congress in the US FD&C Act.

a. In 1975, Drug Digest reported FDA CDER (Center for Drug Evaluation and Research) protected the public by withdrawing NDA (New Drug Application) for fluoride supplements (pills). FDA CDER must do the same for artificial fluoridation drug manufacturers. There is no difference in intent or efficacy between fluoride in pills and fluoridated water. But there is a significant difference in freedom of choice, labeling, and oversight.

b. **HHS** would incur no cost to request FDA CDER to take regulatory action.

c. **FDA CDER** would incur no cost to send a letter to artificial fluoridation drug manufacturers requiring them to gain NDA as required by law.

d. **FD&C Act** protects the public by requiring ***manufacturers*** to gain NDA, not the FDA nor patients. The FDA CDER is to evaluate and regulate substances used with the intent to prevent disease or listed in the official US Pharmacopoeia as a drug. Fluoride is used with intent to prevent disease and listed in the USP. The FDA has testified to Congress and the public that fluoride, when used with the intent to prevent disease, is a drug.

e. **CDC and Surgeon General** actively promote fluoridation for the manufacturers but do not determine scientifically the safety or efficacy of fluoridation or any drugs. Cities and water districts rely on the CDC and Surgeon General assuming they are correct.

f. **EPA** is prohibited by Congress from regulating the addition of any substance to water intended to treat humans. Fluoride is a protected pollutant and the EPA assumes efficacy.

g. **Excess exposure:** Of greatest concern is EPA's confirming in their Dose Response Analysis (DRA) that all infants on formula with fluoridated water are at risk. The DRA reports about a third of children under the age of 7 and all infants on formula made with fluoridated water will be ingesting too much fluoride under the proposed RfD (Reference Dose) and HHS proposed 0.7 ppm artificial fluoridation. Infants and children are being harmed. Excess exposure is confirmed with 41% of children now having dental fluorosis a biomarker of excess fluoride ingestion. An NDA would provide a legend, caution, warnings, and dosage, reducing risks.

h. Over 60 requests and petitions have been made to the FDA CDER since 2009 and the requests, petitions, and complaints have been made. These have been ignored, no answer, or pending for years.

2. **Instruct the CDC to stop the promotion (internet and education) of any and all drugs, including the ingestion of fluoride products, not FDA CDER approved.**

3. **Sponsor a review of the scientific evidence on fluoride's neurotoxicity by the National Academy of Science's National Research Council. The review should include studies listed at www.FluorideAlert.org/issues/health/brain**

Of most concern are the more than 30 human studies finding harm to brains. The question is no longer whether fluoridation causes neurological damage and lower IQ, the question is how much fluoride and at what age damage is caused.

Neurological harm is one of the reasons Israel recently banned fluoridation. Most developed countries have rejected fluoridation due to ethics, lack of efficacy and risks.

4. **Sponsor a quality published independent prospective randomized controlled trial (RCT), of the effectiveness of ingesting hydrofluorosilicic acid (fluoridation), including blood serum and urine concentrations of fluoride.**

a. Quality research is essential and in 60 years of fluoridation, not one published prospective randomized controlled trial of fluoridation has been done. Current reviews of the low quality research available are biased, serious unknowns are not controlled and even known confounding factors are often not controlled.

b. The results of a well-designed RCT could allow HHS to tailor public health policy on fluoridation to optimize benefits and minimize costs. This is in line with the goals of "Obamacare": evidence-based public health policy.

c. Most research on fluoridation have numerous problems which include:

- Not one Randomized Controlled Trial
- Socioeconomic status usually not controlled
- Inadequate size
- Difficulty in diagnosing decay
- Delay in tooth eruption
- Diet: Vitamin D, calcium, strontium, sugar, variables.
- Total exposure of Fluoride and measured blood and/or urine fluoride concentration
- Oral hygiene habits
- Not evaluating life time benefit
- Estimating or assuming subject actually drinks the fluoridated water.
- Dental treatment expenses
- Breast feeding and infant formula
- Fraud or gross errors.
- Genetics

Sincerely,

Jill McElheney
Chris Nidel JD
Bill Hirzy PhD
Paul Connett PhD
Bill Osmunson DDS, MPH

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Gerald Steel PE
Attorney at Law
7303 Young Rd. NW
Olympia WA 98502
360.867.1166 Phone

December 23, 2013

Ms. Jill Warner,
Acting Assoc. Commissioner
WO32, Room 5162
10903 New Hampshire Ave
Silver Spring, MD 20993

RE: Request for Review pursuant to 21 CFR 10.75 – Kailin System Public Drinking Water with Sodium Fluoride – Your file: RFD130073

Dear Ms. Warner:

On September 27, 2013, Leigh Hayes sent me the FDA determination (Attachments A-1 to A-3 hereto) wherein FDA states that it has determined that "Congress did not intend for FDA to regulate the addition of fluoride to public drinking water for dental caries prevention as a drug under the Federal Food, Drug, and Cosmetic Act (FD&C Act)." As a consequence, FDA has responded to our Request for Designation (RFD130073) by finding that our proposed fluoridated public drinking water is not a drug under the FD&C Act. On December 4, 2013, Leigh Hayes informed me that we can request review under 21 CFR 10.75. We hereby submit a Request for Review under 21 CFR 10.75 of the determinations regarding RFD130073.

The FDA has a long history of protecting the public from unsafe and ineffective drugs. Generally, state and local governments do not have the capability or staff to determine if articles or substances intended for preventative health care purposes are safe and effective. HHS, generally acting through the FDA, is the only regulatory body that has the authority to implement the FD&C Act in interstate commerce and protect the public from such articles and substances that are not safe and effective. So we ask the FDA to review its determination that our proposed "fluoridated public drinking water" is not a drug under the FD&C Act.

I believe that the FDA has accepted our statement of facts as accurate. Sodium Fluoride, as a water additive certified under industrial ANSI/NSF Standard 60 is intended for use in the prevention of tooth decay disease in man. (RFD130073 – our RFD at pages 1 and A-1.) This chemical with this intended use is square within the literal language included in the definition of a drug by Congress in 21 USC 321(g)(1)(B). (RFD130073 – our RFD at page 6.) When this chemical is added to our public drinking water, this chemical retains its intended use (prevention of tooth decay disease in man). The purpose of adding this chemical to our public drinking water is to deliver this chemical in drinking water for its intended use. As we stated, our "fluoridated public drinking water" is "intended for use in the prevention of dental caries (tooth decay) disease in man." (RFD130073 – our RFD at page 1.) With this statement, our "fluoridated public drinking water" is square within the literal language included in the definition of a drug in 21 USC 321(g)(1)(B).

RFD130073 provided a letter signed by EPA Water Law Office Associate General Counsel Steven M. Neugeboren, which was sent to me in 2013 on behalf of the EPA Administrator, and

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which states the EPA official position that, "The Department of Health and Human Services (HHS) acting through the FDA, remains responsible for regulating the addition of drugs to water supplies for health care purposes." (RFD130073 – our RFD at page A-8 to A-9.) In RFD130073, we also cited to the Federal Supreme Court ruling in *United States v. An Article of Drug . . . Bacto-Unidisk* (*Bacto-Unidisk*), 394 U.S. 784, 793-801, 89 S.Ct. 1410, 22 L.Ed.2d 726 (1969) which found that the definition of "drug" in 21 USC 321(g)(1)(B) is "as broad as its literal language indicates." (RFD130073 – our RFD at page 6.) There can be no doubt that under the facts presented, ANSI/NSF Standard 60 certified Sodium Fluoride alone and our proposed fluoridated public drinking water are within the literal plain language of the definition of a drug in 21 USC 321(g)(1)(B). Therefore we continue to assert that such Sodium Fluoride and the proposed fluoridated public drinking water are drugs under federal law and are under the jurisdiction of FDA CDER.

I think we can assume that in 1974 Congress was aware of the definition of "drug" in 21 USC 321(g)(1)(B) and aware of the 1969 federal Supreme Court ruling in *Bacto-Unidisk*. I find no plain language in the 1974 SDWA (as amended) that seeks to carve out an exemption from the plain language of 21 USC 321(g)(1)(B) for fluoride water additives or fluoridated public drinking water when the intended use is for the prevention of dental caries disease in man. The challenged determination incorrectly claims that the "text" of the SDWA includes such [plain] language. It does not. The challenged determination also incorrectly claims support from the legislative history of the SDWA. The legislative history of the SDWA cannot be used by FDA to modify the plain language definition of "drug" in 21 USC 321(g)(1)(B) or modify the *Bacto-Unidisk* Court's interpretation of that drug definition. We request that you reverse the determination made for RFD130073 because the SDWA does not carve out an exemption from the plain language of 21 USC 321(g)(1)(B).

We claim that the intent of Congress is clear in 21 USC 321(g)(1)(B) as interpreted by *Bacto-Unidisk* that under our facts, ANSI/NSF Standard 60 certified Sodium Fluoride alone and our proposed fluoridated public drinking water are drugs under the FD&C Act. To further support our claim, we cited to 21 USC 321ff ("Dietary Supplement Health and Education Act of 1994") that states that minerals [such as fluoride public water additives] are foods except when they meet the definition of a drug. (RFD130073 – our RFD at page 6.) This 1994 statute did not exempt minerals that meet the definition of a "drug" in 21 USC 321(g)(1)(B) from being drugs just because the minerals were being added to public water supplies. This subsequent Congressional enactment supports our claim.

The federal Supreme Court in *FDA v. Brown & Williamson Tobacco Corp.* (*Tobacco Corp.*), 529 U.S. 120, 120 S.Ct. 1291, 146 L.Ed.2d 121 (2000) further supports our claim and refutes the claim in the determination regarding Congressional intent of 21 USC 321(g)(1)(B). The *Tobacco Corp.* Court found that reading the FD&C Act as a whole, as well as in conjunction with Congress' subsequent tobacco-specific legislation, it is plain that Congress has not given the FDA the authority to regulate tobacco products as customarily marketed. (*Tobacco Corp.* at 120 and 131-61.) "As customarily marketed" means "without manufacturer claims of therapeutic benefit." (*Id.* at 120.) But the *Tobacco Corp.* Court found that while the FDA did not generally have authority to regulate tobacco under the FD&C Act, there was a "well-established exception of when the manufacturer makes express claims of therapeutic benefit." (*Id.* at 158.) Therapeutic benefit refers to uses identified in 21 USC 321(g)(1)(B). We are making an express claim of therapeutic benefit for our proposed fluoridated public drinking water.

In the instant case, Congress has not shown that it has created a distinct regulatory scheme addressing the subject of purposely adding fluoride to public drinking water. But even if it did

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have such a distinct regulatory scheme, FDA still has authority and responsibility under the FD&C Act to regulate fluoride added to public drinking water when it is added for the "therapeutic benefit" of preventing tooth decay disease. Similarly, FDA has authority and responsibility under the FD&C Act to regulate our fluoridated public drinking water because our water is fluoridated with the intent to prevent tooth decay disease. The FDA can point to no relevant federal caselaw where products that are intended for use in the prevention of disease in man are not regulated by the FD&C Act independent of other Congressional enactments.

Therefore under 21 CFR 10.75(a)(3) and 21 CFR 10.75(c)(1) and (2) along with 21 CFR 10.75(d) we request review and if it is concluded that our proposed ANSI/NSF Standard 60 fluoride water additives and our proposed fluoridated public drinking water are drugs, we again request that you designate our proposed fluoridated public drinking as a drug regulated by CDER.

Respectfully submitted,

Gerald Steel, PE, Attorney at Law
geraldsteel@yahoo.com

Attachments: A-1 to A-3

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DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Food and Drug Administration
Silver Spring, MD 20993

Office of Combination Products
WO 32, Room 5129
10903 New Hampshire Avenue
Silver Spring, MD 20993

September 27, 2013

Eloise Kailin
Owner and Manager
Gerald Steel
Attorney
Kailin Public Water System
160 Kane Lane
Sequim, WA 98382

Re: Request for Designation
Kailin Public Drinking Water System with Sodium Fluoride
Our file: RFD130073
Dated: July 22, 2013
Received: July 23, 2013
Filed: July 29, 2013

Dear Dr. Kailin and Mr. Steel:

The United States (U.S.) Food and Drug Administration (FDA) has completed its review of the request for designation (RFD) for the Kailin Public Drinking Water System with Sodium Fluoride that you submitted on behalf of Kailin Public Water System. We have determined that Congress did not intend for FDA to regulate the addition of fluoride to public drinking water for dental caries prevention as a drug under the Federal Food, Drug, and Cosmetic Act (FD&C Act). Instead, Congress intended that the U.S. Environmental Protection Agency (EPA) regulate fluoride in public drinking water as a potential contaminant under the Safe Drinking Water Act of 1974 (SDWA) to protect against adverse health effects, and that within the limits thus set by EPA, state and local governments be permitted, but not required, to fluoridate drinking water to help prevent dental caries. Thus, we are not designating your fluoridated public drinking water as a drug under the FD&C Act.

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Description

In your RFD, you seek designation of your specific public fluoridated drinking water as a drug under the FD&C Act. You assert that you will submit a New Drug Application (NDA) for your fluoridated public drinking water that "will be composed of our public drinking water with an added fluoridation product certified to meet ANSI/NSF Standard 60...: Sodium Fluoride with a maximum addition of 2.3 mg/L....The public drinking water system is registered with the Washington State Department of Health as PWS ID# AC982. It is a neighborhood system with multiple approved connections. The source water comes from a well as is typical for public water systems in Washington State and currently there is a transmission pipeline from the well to a tank that maintains water pressure for the system in an acceptable range. A distribution system which starts at the tank serves all of the individual residential and commercial connections. There are pressure zones in the distribution system where pressure reducers are used to lower water pressure for connections at lower elevations. All individual connections to the distribution system are made in a manner approved by the Washington State Department of Health."

The RFD explains that "...the transmission line will be rerouted to a small fluoridation building where fluoridation will occur and the fluoridated water will be transmitted to the tank that maintains water pressure. This public water system is required to meet standard specifications for public water systems in Washington State as established by the Washington State Board of Health." The RFD states that the addition of the fluoridation materials "...will be metered into flowing water in a manner to maintain the specified chemical concentration rates. The Sodium Fluoride will be injected using an up-draft fluoride saturator. The injection rate into the transmission line in the control house will be controlled using a 4 to 20 milliamperes signal from the main water meter so that finished fluoridation levels are close to 0.7 mg/L. Fluoride levels will be manually checked twice daily." Finally, with regard to packaging of the product, the RFD asserts that "[t]his system does not have conventional packaging. [The company proposes] that [it] will negotiate with CDER regarding adequate labeling. For example, [the company] will propose that drug facts and warning approved by CDER will be sent out with each billing for each connection."

You recommend that your fluoridated public drinking water designed to aid in the prevention and prophylactic treatment of dental caries disease be classified as a drug and that it be assigned to FDA's Center for Drug Evaluation and Research (CDER) for premarket review and regulation.

Product Classification

We have considered the information in the RFD and discussed the issues with staff from CDER, the Center for Food Safety and Applied Nutrition, the Department of Health and Human Services, HHS's Office of the General Counsel, and the EPA.

A-2 B67

After careful consideration, we conclude that Congress did not intend for FDA to regulate the addition of fluoride to public drinking water for dental caries prevention as a drug under the FD&C Act. Instead, Congress intended that EPA regulate fluoride in public drinking water as a potential contaminant under the SDWA to protect against adverse health effects, and that within the limits thus set by EPA, state and local governments be permitted, but not required, to fluoridate public drinking water to help prevent dental caries. The SDWA gives EPA certain authorities with respect to the regulation of public drinking water, including the authority to promulgate national primary drinking water regulations that set maximum contaminant levels (MCLs) for contaminants that EPA determines may have an adverse effect on human health. Pursuant to its authority under the SDWA, EPA has codified a primary MCL for fluoride at 40 CFR § 141.62(b)(1) and a secondary MCL for fluoride at 40 CFR § 143.3.

The historical context surrounding the passage of the SDWA indicates that Congress was aware in 1974 that many localities were adding fluoride to public drinking water to help prevent dental caries. They were also aware that FDA had a codified policy of not regulating such fluoride as a drug, so long as the levels were within certain recommended limits. Based on the text and legislative history of the SDWA, we have concluded that Congress did not intend for FDA to regulate fluoride in public drinking water for the purpose of helping to prevent dental caries as a drug under the FD&C Act. Instead, Congress set up a regime under which EPA would set upper limits for fluoride to protect against adverse health effects, and EPA would not have the authority to mandate or ban the use of fluoride to help prevent dental caries. The decision of whether or not to add fluoride to public drinking water to help prevent dental caries (within the limits set by EPA) was left to state and local authorities, as it had been before 1974. Since the passage of the SDWA, this division of federal and state/local oversight has continued.

Conclusion

For the reasons explained above, we have determined that Congress did not intend for FDA to regulate fluoride in public drinking water to help prevent dental caries as a drug under the FD&C Act, and we therefore are not designating your fluoridated public drinking water as a drug.

If you have any other questions about this letter, please feel free to contact me. You may reach us at the above address or by email at combination@fda.gov.

Sincerely,

Leigh Hayes

Leigh Hayes
Product Assignment Officer

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A-3

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
GENERAL COUNSEL

February 14, 2013

Gerald Steel, PE
7303 Young Road NW
Olympia, WA 98502

Dear Mr. Steel:

This is in response to your letter of December 28, 2012 to EPA Administrator Lisa Jackson in which you asked several questions about the status of an MOU between EPA and the Federal Drug Administration (FDA) published in 1979. I am replying on behalf of her.

Your first question is whether, from the viewpoint of EPA, the purpose of a 1979 Memorandum of Understanding (MOU) between EPA and the Federal Drug Administration (FDA) was "to take away from FDA, and give to EPA, responsibility for regulating public drinking water additives intended for preventative health care purposes and unrelated to contamination of public drinking water?" Your second question is whether, if that was the purpose of the 1979 MOU, the MOU was terminated through a subsequent Federal Register notice.

The answer to your first question is no, so there is no need to address your second question. The purpose of the MOU was not to shift any responsibilities between the Agencies. Rather, it was to help facilitate effective coordination of our respective legal authorities. Under the Safe Drinking Water Act (SDWA), EPA is the lead federal agency with responsibility to regulate the safety of public water supplies. EPA does not have responsibility for substances added to water solely for preventative health care purposes, such as fluoride, other than to limit the addition of such substances to protect public health or to prevent such substances from interfering with the effectiveness of any required treatment techniques. SDWA Section 1412(b)(11); see also A Legislative History of the Safe Drinking Water Act, Committee Print, 97th Cong, 2d Session (February 1982) at 547. The Department of Health and Human Services (HHS), acting through the FDA, remains responsible for regulating the addition of drugs to water supplies for health care purposes.

The 1979 MOU was intended to address contamination of drinking water supplies as a result of direct or indirect additives to drinking water, not to address the addition of substances solely for preventative health purposes. 44 Fed. Reg. 42775 (July 20, 1979) ("EPA and FDA agree: (1) that *contamination* of drinking water from the use and application of direct and indirect additives and other substances poses a potential public health problem...")(emphasis added). It was intended to avoid potentially duplicative regulation of "food", which FDA had, in the past, considered to include drinking water. 44 Fed. Reg. 42775 (July 20, 1979). The MOU did not address drugs or other substances added to water for health care purposes.

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I hope that this has adequately answered your inquiry. Please do not hesitate to contact Carrie Wehling of my staff (202-564-5492) if you have further questions about this.

Sincerely,

Steven M. Neugeboren
Associate General Counsel
Water Law Office

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 10
1200 Sixth Avenue, Suite 900
Seattle, WA 98101-3140

OFFICE OF
WATER AND
WATERSHEDS

NOV 17 2011

Mr. Gerald Steel, PE
Attorney-at-Law
7303 Young Road NW
Olympia, Washington 98502

Dear Mr. Steel:

I am responding to your letter dated November 7, 2011, on behalf of Dennis J. McLerran, Regional Administrator, U S. Environmental Protection Agency (EPA). In your communication you have asked the EPA to send you a letter that answers the question "Are [Washington Administrative Code] WAC 246-290-220(3) and 246-290-460 part of implementation of requirements of the Federal Safe Drinking Water Act in Washington State, or are they unrelated to the requirements of the Federal Safe Drinking Water Act in Washington State?"

A concise answer to your question is that the provisions at WAC 246-290-220(3) and 246-290-460 are not related to the requirements of the Federal Safe Drinking Water Act in Washington State. An explanation as to why this is the case follows.

The requirements for a State drinking water primacy program are spelled out in Section 1413 of the Federal Safe Drinking Water Act (SDWA) (42 U.S.C. § 300g-2). Section 1413(a) specifies that a State has primary enforcement responsibility (primacy) for public water systems during any period for which the EPA Administrator determines that such State:

(1) has adopted drinking water regulations that are no less stringent than the national primary drinking water regulations i.e., the regulations promulgated at 40 CFR Part 141 (see http://ecfr.gpoaccess.gov/cgi/t/text/text-idx?tpl=/ecfrbrowse/Title40/40tab_02.tpl);

(2) has adopted and is implementing adequate procedures for the enforcement of such State regulations as the Administrator may require by regulation;

(3) will keep such records and make such reports with respect to its activities as the Administrator may require by regulation;

(4) if it permits variances and/or exemptions from the requirements of its drinking water regulations, permits such variances and exemptions under conditions and in a manner which is not less stringent than the conditions under, and the manner in which variances and exemptions may be granted under SDWA sections 1415 and 1416;

(5) has adopted and can implement an adequate plan for the provision of safe drinking water under emergency circumstances; and

(6) has adopted authority for administrative penalties, unless the constitution of the State prohibits the adoption of such authority.

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The EPA's role in SDWA section 1413(b) requires the Administrator to promulgate regulations that establish how the States may apply for primacy, how the Administrator will make primacy determinations and the manner in which the Administrator may determine that the primacy agency is no longer meeting the primacy requirements. These primacy implementing regulations can be found at 40 CFR 142.10 – 40 CFR 142.12. (See enclosure and/or website provided above.) 40 CFR Part 142.10 describes the requirements of a State primacy program. 40 CFR Part 142.11 describes the documents a State must submit to the EPA for an initial determination of primacy. 40 CFR 142.12 describes the contents of a State request for approval of a State's revised primacy program. This must take place whenever the EPA adopts a new or revised drinking water rule. As per 40 CFR 142.12(c) a State must submit for EPA approval a copy of their regulations and a document we refer to as a crosswalk. The crosswalk is a side-by-side comparison of the new or revised Federal requirements in 40 CFR Parts 141 and 142 and the corresponding State authorities, including citations to the specific statutes and administrative regulations (see enclosed example of a crosswalk page). EPA will only make a determination that a State's revised drinking water primacy program can be approved if the State's regulations are as stringent as the Federal regulations and the State continues to maintain all required authorities as per SDWA Section 1413.

WAC 246-290-220(3) requires treatment chemicals with the exception of commercially retailed hypochlorite compounds added to water intended for potable use to comply with ANSI/NSF Standard 60 and also specifies that the maximum application dosage recommendation for the product certified by the ANSI/NSF Standard 60 shall not be exceeded in practice. The Department of Health (DOH), which is the State of Washington's drinking water primacy agency has never submitted WAC 246-290-220(3) to the EPA for approval as there is no analogous provision in the National Primary Drinking Water Regulations at 40 CFR Part 141, and neither the other statutory provisions mentioned above, nor the primacy implementing provisions at 40 CFR Part 142 require that language, such as is found in WAC 246-290-220(3), be part of a State primacy program.

WAC 246-290-460 addresses fluoridation practices, should a community choose to provide fluoridation. DOH has never submitted WAC 246-290-460 to the EPA for approval as there are no analogous provisions in the National Primary Drinking Water Regulations at 40 CFR Part 141, and neither the other statutory provisions mentioned above, nor the primacy implementing provisions at 40 CFR Part 142 require that a State primacy program regulate fluoridation practices.

For the reasons stated in the above paragraphs, I can assert that that the provisions at WAC 246-290-220(3) and 246-290-460 were not required to be submitted by the State or approved by the EPA and these provisions are not related to the requirements of the Federal Safe Drinking Water Act.

I hope this response answers your questions satisfactorily. If you have additional questions, please contact Marie Jennings, our Manager for the Drinking Water Unit at (260) 553-1893.

Sincerely,

Michael A. Bussell, Director
Office of Water & Watersheds

Enclosures

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February 2008

NSF Fact Sheet on Fluoridation Chemicals

Introduction

This fact sheet provides information on the fluoride containing water treatment additives that NSF has tested and certified to NSF/ANSI Standard 60: Drinking Water Chemicals - Health Effects. According to the latest Association of State Drinking Water Administrators Survey on State Adoption of NSF/ANSI Standards 60 and 61, 45 states require that chemicals used in treating potable water must meet Standard 60 requirements. If you have questions on your state's requirements, or how the NSF/ANSI Standard 60 certified products are used in your state, you should contact your state's Drinking Water Administrator.

Water fluoridation is the practice of adjusting the fluoride content of drinking water. Fluoride is added to water for the public health benefit of preventing and reducing tooth decay and improving the health of the community. The U.S. Centers for Disease Control and Prevention is a reliable source of information on this important public health intervention. For more information please visit www.cdc.gov/fluoridation/.

NSF certifies three basic products in the fluoridation category:

1. Fluorosilicic Acid (aka Fluosilicic Acid or Hydrofluosilicic Acid).
2. Sodium Fluorosilicate (aka Sodium Silicofluoride).
3. Sodium Fluoride.

NSF Standard 60

Products used for drinking water treatment are evaluated to the criteria specified in NSF/ANSI Standard 60. This standard was developed by an NSF-led consortium, including the American Water Works Association (AWWA), the American Water Works Association Research Foundation (AWWARF), the Association of State Drinking Water Administrators (ASDWA), and the Conference of State Health and Environmental Managers (COSHEM). This group developed NSF/ANSI Standard 60, at the request of the US EPA Office of Water, in 1988. The NSF Joint Committee on Drinking Water Additives continues to review and maintain the standard annually. This committee consists of representatives from the original stakeholder groups as well as other regulatory, water utility and product manufacturer representatives.

Standard 60 was developed to establish minimum requirements for the control of potential adverse human health effects from products added directly to water during its treatment, storage and distribution. The standard requires a full formulation disclosure of each chemical ingredient in a product. It also requires a toxicology review to determine that the product is safe at its maximum use level and to evaluate potential contaminants in the product. The standard requires testing of the treatment chemical products, typically by dosing these in water at 10 times the maximum use level, so that trace levels of contaminants can be detected. A toxicology evaluation of test results is required to determine if any contaminant concentrations have the potential to cause adverse human health effects. The standard sets criteria for the establishment of single product allowable concentrations (SPAC) of each respective contaminant. For contaminants regulated by the U.S. EPA, this SPAC has a default level not to exceed ten-percent of the regulatory level to provide protection for the consumer in the unlikely event of multiple sources of the contaminant, unless a lower or higher number of sources can be specifically identified.

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DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Food and Drug Administration
Silver Spring, MD 20993

Office of Combination Products
WO 32, Room 5129
10903 New Hampshire Avenue
Silver Spring, MD 20993

September 23, 2015

Mr. Gerald Steel, PE
Attorney-At-Law
7303 Young Road, NW
Olympia, WA 98502

Re: "Submittal of Three Requests for Designation
for Libera Bottled Fluoridated Water each using a Different Fluoridation Chemical"
Dated: September 2, 2015
Received: September 2, 2015

Dear Mr. Steel:

For the reasons discussed below, we disagree that our previous legal reasoning is, as you indicate below, "no longer valid." As we have previously communicated to you, and as stated in the preamble to 21 CFR Part 3, Part 3 "does not apply to foods, veterinary products, or cosmetics" (56 FR 58754), and jurisdictional questions concerning a product that may be within the jurisdiction of the Center for Food Safety and Applied Nutrition (CFSAN) are outside the scope of 21 CFR part 3 and section 563 of the FD&C Act. In contrast to your characterization, the Center for Food Safety and Applied Nutrition's (CFSAN's) recent communication to you (Letter from F. Billingslea dated August 7, 2015, attached) does not state that your proposed bottled water product with the claim discussed below ("this drinking water is intended for use in the prevention of tooth decay disease") is "not a food under their [CFSAN's] jurisdiction." Instead, Ms. Billingslea stated that this proposed labeling statement "is not an authorized claim on food labeling under Section 403(r) of the Act." Ms. Billingslea further recommended that you contact Ms. Barbara Gould in FDA's Center for Drug Evaluation and Research (CDER) if you wished to market a bottled water product with this claim.

Ms. Billingslea recommended contacting CDER because you propose to market your product with a therapeutic claim. Your proposed claim would establish that your product is intended to prevent disease. Therefore, your proposed product (if marketed with your proposed claim) would be a drug as that term is defined in section 201(g)(1)(B) of the Federal Food, Drug, and Cosmetic Act (the Act).

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However, the fact that your proposed product (if marketed with your proposed claim) would be a drug under the Act does not mean that your product is not also a food. To the contrary, the definitions of "food" and "drug" under the Act are not mutually exclusive. *See, e.g., Nutrilab v. Schweiker*, 713 F.2d 335, 336 (7th Cir. 1983). It is commonplace for FDA to take regulatory action with respect to food products that are promoted for conditions that cause the products to be drugs as well as foods.

Accordingly, we believe that our previous legal reasoning continues to apply, and your most recent requests fall outside the scope of the regulation and statutory provisions that authorize requests for designation. As a result, we are not treating your submissions regarding fluoridated bottled water as requests for designation. Instead, we are treating them as informal inquiries.

We hope it is helpful for you to know that your proposed product (if marketed with your proposed claim) would be both a food and a drug under the Act. We note that if you were to remove your proposed claim ("This drinking water is intended for use in the prevention of tooth decay disease"), your product would not be a drug under the Act unless there was other evidence to establish its status as a drug. As Ms. Billingslea discussed in her letter of August 7, your other proposed claim – "fluoride added" – would not render your product a drug. You can also reference Ms. Billingslea's letter for information about a health claim that may be used on certain bottled water products.

As Ms. Billingslea stated in her letter of August 7, we recommend that you contact Ms. Barbara Gould in CDER if you wish to market your bottled water product with your proposed claim about the prevention of tooth decay.

Patricia A. Hansen, Ph.D.

Acting Director
Office of Nutrition, Labeling and Dietary Supplements
CFSAN
FDA

Leigh Hayes

Leigh Hayes

Product Assignment Officer
Office of Combination Products
FDA

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CFSAN/Office of Nutritional Products, Labeling, and Dietary Supplements
October 14, 2006

Health Claim Notification for Fluoridated Water and Reduced Risk of Dental Caries

Under section 403(r)(3)(C) (21 U.S.C. §343(r)(3)(C)) of the Federal Food, Drug, and Cosmetic Act (Act), a manufacturer may submit to the Food and Drug Administration (FDA) a notification of a health claim based on an authoritative statement from an appropriate scientific body of the United States Government or the National Academy of Sciences (NAS) or any of its subdivisions. The notification must be submitted to FDA at least 120 days before the food is introduced into interstate commerce. The claim may be made after 120 days from the date of submission of the notification until such time as 1) FDA issues a regulation prohibiting or modifying the claim or finding that the requirements for making the claim have not been met, or 2) a district court in an enforcement proceeding has determined that the requirements for making the claim have not been met.

On June 16, 2006, the FDA received a notification (the June 16 notification) from the law firm of Covington and Burling regarding a health claim for the relationship between fluoridated water and a reduced risk of dental caries. The 120-day period from the date of submission of the June 16 notification was October 14, 2006. Therefore, after October 14, 2006, manufacturers may use the claim specified in the notification, as modified by the notifier in a letter to FDA dated October 13, on the label and in labeling of any food product that meets the eligibility criteria described below, unless or until FDA or a court acts to prohibit the claim.

The June 16 notification cites statements from several sources as authoritative statements for the claim. FDA reviewed the sources and cited statements in their context and in light of existing authorized health claims and current science. The following three statements are considered authoritative for purposes of this notification.

Recommendation for Using Fluoride to Prevent and Control Dental Caries in the U.S. (Centers for Disease Control, 2001):

> "Widespread use of fluoride has been a major factor in the decline in the prevalence and severity of dental caries (i.e., tooth decay) in the United States and other economically developed countries. When used appropriately, fluoride is both safe and effective in preventing and controlling dental caries. All U.S. residents are likely exposed to some degree of fluoride, which is available from multiple sources." (Summary section, page 1)
>
> "Continue and extend fluoridation of community drinking water: Community water fluoridation is a safe, effective, and inexpensive way to prevent dental caries. This modality benefits persons in all age groups and of all SES, ..." (Recommendation section, page 24)

Oral Health in America: A Report of the Surgeon General (2000):

> "Community water fluoridation is safe and effective in preventing dental caries in both children and adults. Water fluoridation benefits all residents served by community water supplies regardless of their social or economic status. Professional and individual measures, including the use of fluoride mouth rinses, gels, dentifrices, and dietary supplements and the application of dental sealants, are additional means of preventing dental caries." (Executive summary)

Review of Fluoride: Benefits and Risks (Public Health Service, 1991):

> "Extensive studies over the past 50 years have established that individuals whose drinking water is fluoridated show a reduction in dental caries. Although the comparative degree of measurable benefit has been reduced recently as other fluoride sources have become available in non-fluoride areas, the benefits of water fluoridation are still clearly evident." (Conclusions section, page 87)

According to the June 16 notification and the letter to FDA dated October 13, the food eligible to bear the claim is bottled water meeting the standards of identity and quality set forth in 21 CFR 165.110, containing greater than 0.6 and up to 1.0 mg/L *total* fluoride, and meeting all general requirements for health claims (21 CFR 101.14) with the exception of minimum nutrient contribution (21 CFR 101.14 (e)(6)). The claim language is: "Drinking fluoridated water may reduce the risk of [dental caries or tooth decay]." In addition, the health claim is not intended for use on bottled water products specifically marketed for use by infants.

The notification and materials regarding the claim are publicly available from the FDA Division of Dockets Management (Docket No.2006Q-0418). Persons interested in these documents may view them at the Division of Dockets Management from 9am to 4pm, Monday through Friday at 5630 Fishers Lane, room 1061, Rockville, MD 20852. The Division of Dockets Management may be contacted at 301-827-6860. FDA also intends to make the documents available on the Dockets web site at http://www.fda.gov/ohrms/dockets/dockets/dockets.htm, under Docket No. 2006Q-0418.

FDA U.S. FOOD & DRUG ADMINISTRATION

Inspections, Compliance, Enforcement, and Criminal Investigations

DS Waters of America, LP 6/8/09

Department of Health and Human Services

Public Health Service
Food and Drug Administration
5100 Paint Branch Parkway
College Park, MD 20740

JUN - 8 2009

WARNING LETTER

CERTIFIED MAIL
RETURN RECEIPT REQUESTED

Mr. Stewart E. Allen
Mr. Dillon Schickli
Chief Executive Officers
DS Waters of America, Inc.
5660 New Northside Drive, Suite 500
Atlanta, GA 30328

Re: CFSAN-OC-WL09-03

Dear Mr. Allen and Schickli:

The Food and Drug Administration (FDA) has reviewed the label for the 1 Gal (3.78L) package of your NURSERY@ Purified Water with added fluoride, and your website at www.nurserywater.com in April 2009. Based on our review, we have found that your product label has serious violations of the Federal Food, Drug, and Cosmetic Act (the Act) and the applicable regulations in Title 21, Code of Federal Regulations (21 CFR). You can find the Act and these regulations through links on FDA's Internet website at www.fda.gov.

Your significant violations are as follows:

Your product is misbranded within the meaning of section 403(r)(1)(B) of the Act [21 U.S.C. 343(r)(1)(B)], because it bears an unauthorized health claim in its labeling. We have determined that your website www.nurserywater.com. the address for which appears on your product's label, is labeling for your product under section 201(m) of the Act [21 U.S.C. 321(m)]. This website bears the following unauthorized health claim: "Drinking water with added fluoride in the proper amounts has been shown to be so effective that the ADA supports municipal water fluoridation and refers to this as the single most effective measure to prevent tooth decay." Health claims may not be made for food products, including bottled water, for which the label represents or purports that the food is for infants or toddlers less than two years of age, unless FDA has provided for such claim by regulation. 21 CFR 101.14(e)(5). [1]

Your product is misbranded within the meaning of section 403(q) of the Act [21 U.S.C. 343(q)] in that nutrition facts information is not in an appropriate format as defined in 21 CFR 101.9. The Nutrition Facts panel uses abbreviations for serving size and servings per container that are not in accordance with 21 CFR 101.9(j)(13)(ii)(B), which provides that the use of specific abbreviations to list nutrients is only for packages that have a total surface area available to bear labeling of 40 or less square inches. In addition, the correct heading on the Nutrition Facts panel for declaring the quantity of a nutrient is "% Daily Value" (not "Amount") in accordance with 21 CFR 101.9(d)(6).

This letter is not meant to be an all-inclusive review of your NURSERY® Purified Water product and its labeling. It is your

responsibility to ensure that all of your products are in compliance with the laws and regulations enforced by FDA. You should take prompt action to correct the violations described above and prevent their recurrence. Failure to promptly correct these violations may result in regulatory action without further notice, such as seizure and/or injunction.

Please respond in writing within fifteen (15) working days from your receipt of this letter. Your response should include each step that has been or will be taken to completely correct the labeling violations and to prevent the recurrence of similar violations, the time within which the correction will be completed, and any documentation necessary to show that the correction has been achieved. If applicable, please include a copy of your revised label. If corrective actions cannot be completed within fifteen (15) working days, state the reason for the delay and the time within which the corrections will be completed.

In addition, we have the following comments:

- The serving size of your NURSERY® Purified Water product is based on 8 fluid ounces. While FDA has not established a reference amount customarily consumed (RACC) for water by infants and toddlers, we recommend that you use the infant and toddler RACC for juices, which is 4 fl oz (120 mL) [21 CFR 101.12(b), Table I (Juices, all varieties)].
- Although minerals are added for taste, the statement of identity does not include this information (e.g., "purified water with minerals added for taste") [60 FR 57076 at 57080 and 57082, November 13, 1995].

If you need additional information or have questions concerning any products distributed through your website, please contact FDA. You may respond in writing to Felicia Binion Williams, Compliance Officer, Division of Enforcement, Center for Food Safety and Applied Nutrition, Food and Drug Administration, 5100 Paint Branch Parkway, College Park, MD 20740.

Sincerely,

/s/

Roberta F. Wagner

Director
Office of Compliance
Center for Food Safety
and Applied Nutrition

(b)(4)

1. FDA notes that the following claim regarding fluoridated water and reduced risk of dental caries or tooth decay may be made consistent with a health claim notification under section 403(r)(3)(C) of the Act [21 U.S.C. 343(r)(3)(C)]: "Drinking fluoridated water may reduce the risk of [dental caries or tooth decay]." [www.cfsan.fda.gov/~dms/flfluoro.html]. However, the prohibition under 21 CFR 101.14(e)(5) applies to these health claim notifications, and this notification explicitly excluded bottled water products that are specifically marketed for use by infants, and therefore your product is not eligible to bear this health claim. Furthermore, the language of the claim on your website differs significantly from the language in the claim in the notification.

DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Food and Drug Administration
Rockville MD 20857

FEB 21 1997

John V. Kelly
Assemblyman, 36th District
Bergen-Essex-Passaic Countries
371 Franklin Avenue, 2nd Floor
Nutely, New Jersey 07110

Dear Assemblyman Kelly:

This responds to your February 5, 1997, letter asking whether there have been any drug applications approved by the Food and Drug Administration for fluoride tablets for children or fluoride drops for infants since 1992.

At this time there are no approved new drug applications on file with the Agency for these products. If you have any further questions on this issue I can be contacted at (301) 594-0101.

Sincerely yours,

Frank R. Fazzari

Frank R. Fazzari
Prescription Drug Strategy Development & PDMA
Division of Prescription Drug Compliance and Surveillance
Office of Compliance
Center for Drug Evaluation and Research

DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Food and Drug Administration
Rockville MD 20857

DEC 21 2000

The Honorable Ken Calvert
Chairman
Subcommittee on Energy and Environment
Committee on Science
House of Representatives
Washington, D.C. 20515-6301

Dear Mr. Chairman:

Thank you for the letter of May 8, 2000, to Dr. Jane E. Henney, Commissioner of Food and Drugs, regarding the use of fluoride in drinking water and drug products. We apologize for the delay in responding to you.

We have restated each of your questions, followed by our response.

1. **If health claims are made for fluoride-containing products (e.g. that they reduce dental caries incidence or reduce pathology from osteoporosis), do such claims mandate that the fluoride-containing product be considered a drug, and thus subject the product to applicable regulatory controls?**

Fluoride, when used in the diagnosis, cure, mitigation, treatment, or prevention of disease in man or animal, is a drug that is subject to Food and Drug Administration (FDA) regulation. FDA published a final rule on October 6, 1995, for anticaries drug products for over-the-counter (OTC) human use (copy enclosed). This rule establishes the conditions under which OTC anticaries drug products are generally recognized as safe and effective and not misbranded. The rule has provisions for active ingredients, packaging conditions, labeling, and testing procedures that are required by manufacturers in order to market anticaries products. A new drug application (NDA) may be filed for a product containing fluoride that does not meet the provisions stated in the final rule. As you know, the Environmental Protection Agency regulates fluoride in the water supply.

2. Are there any New Drug Applications (NDA) on file, that have been approved, or that have been rejected, that involve a fluoride-containing product (including fluoride-containing vitamin products) intended for ingestion with the stated aim of reducing dental caries? If any such NDA's have been rejected, on what grounds were they rejected? If any such NDA have been approved, please provide the data on safety and efficacy that FDA found persuasive.

No NDAs have been approved or rejected for fluoride drugs meant for ingestion. Several NDAs have been approved for fluoride topical products such as dentifrices and gels. Fluoride products in the form of liquid and tablets meant for ingestion were in use prior to enactment of the Kefauver-Harris Amendments (Drug Amendments of 1962) to the Food, Drug, and Cosmetic Act in which efficacy became a requirement, in addition to safety, for drugs marketed in the United States (U.S.). Drugs in use prior to 1962 are being reviewed under a process known as the drug efficacy study implementation (DESI). The DESI review of fluoride-containing products has not been completed.

3. Does FDA consider dental fluorosis a sign of over exposure to fluoride?

Dental fluorosis is indicative of greater than optimal ingestion of fluoride. In 1988, the U.S. Surgeon General reported that dental fluorosis, while not a desirable condition, should be considered a cosmetic effect rather than an adverse health effect. Surgeon General M. Joycelyn Elders reaffirmed this position in 1994.

4. Does FDA have any action-level or other regulatory restriction or policy statement on fluoride exposure aimed at minimizing chronic toxicity in adults or children?

The monograph for OTC anticaries drug products sets acceptable concentrations for fluoride dentifrices, gels and rinses (all for topical use only). This monograph also describes the acceptable dosing regimens and labeling including warnings and directions for use. FDA's principal safety concern regarding fluoride in OTC drugs is the incidence of fluorosis in

children. Children under two years of age do not have control of their swallowing reflex and do not have the skills to expectorate toothpaste properly. Young children are most susceptible to mild fluorosis as a result of improper use and swallowing of a fluoride toothpaste. These concerns are addressed in the monograph by mandating maximum concentrations, labeling that specifies directions for use and age restrictions, and package size limits.

Thanks again for contacting us concerning this matter. If you have further questions, please let us know.

Sincerely,

Melinda K. Plaisier
Associate Commissioner
for Legislation

Enclosure
"Final Rule/Federal Register - October 6, 1995
Over-the-Counter Anticaries Drug Products"

Web site administrator's note:
To perform query to access this document
Enter: http://www.access.gpo.gov/su_docs/aces/aces140.html
Enter: checkmark for 1995 Volume 60
Enter: On: 10/06/95
Enter: Search terms: anticaries

Bill Osmunson DDS , MPH
bill@teachingsmiles.com 425.466.0100
2011 Not updated

WASHINGTON STATE BOARD OF HEALTH

RE: FLUORIDE'S EFFECTS ON THE ENDOCRINE SYSTEM, January 2024

OUTLINE:

SUMMARY: Fluoride is an endocrine disruptor.

Maximum fluoride intake goal <0.001 mg/kg/day.

I. EVALUATING FLUORIDE AS AN ENDOCRINE DISRUPTING TOXICANT.

Hundreds of research articles have reported adverse effects of excess fluoride exposure including but not limited to arthritis, bone, tooth, brain, cancer, cardiovascular, diabetes, thyroid, parathyroid, pancreas, pineal, adrenal, gonads, enteroendocrine, paraganglia, pituitary, placenta, endocrine, GI, kidney, and reproductive harm.

Fluoride has effects throughout the body. Fluoride should be evaluated at the biochemical, cellular, and organ levels as well as synergistic toxic effects with a margin of safety for race, age, nutritional deficiencies, ill health of those most vulnerable, total exposure and unknowns. To protect the public, we must use a margin of safety from the lowest observed adverse effect and a factor of 100. We do a disservice to humanity and science when we compartmentalize evidence without bringing the weight from all effects to the table for evaluation and judgment. In the end, judgment is required from a "global" perspective for all, not just the mean.

The NRC (2006) report to the EPA which labeled fluoride an "endocrine disruptor,"[1] as well as numerous studies,[2] reviews, and reasonable judgment.

The NRC (2006)[3] review members were tasked to determine "with absolute certainty" that research had demonstrated adverse effects---one member remembers the term, "bet the farm certainty". Such a high degree of certainty is not supported by Congress who requires the EPA to determine contaminate levels to be *"set at the level at which no known or anticipated adverse effects on the health of persons occur and which allows an adequate margin of safety."* The committee unanimously "bet the farm" that fluoride is an endocrine disruptor.

The endocrine system includes all of the glands of the body and the hormones produced by those glands, such as anterior and posterior pituitary, thyroid, parathyroid, adrenal, gonads, islets of pancreas, pineal, enteroendocrine, paraganglia and placenta. The glands are controlled directly by stimulation from the nervous system as well as by chemical receptors in the blood and hormones produced by interaction with other glands. By regulating the functions of organs in the body, these glands help to maintain

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[1] National Academies of Sciences, Engineering and Medicine 500 Fifth St. N.w. Washington, DC, 20001. Page 266 "Fluoride in Drinking Water: A Scientific Review of EPA's Standards."

[2] Such as Malin A, Till C, Exposure to fluoridated water and attention deficit hyperactivity disorder prevalence among children and adolescents in the United States: an ecological association. Environmental Health (2015) 14:17 and
Peckham et al, (2015) Centre for Health Services Studies, University of Kent, Canterbury, Kent, UK. J. Epidemiology Community Health do:10.1136/jech-2014-204971

[3] "Fluoride in Drinking Water: A Scientific Review of EPA's Standards."
http://www.nap.edu/catalog/11571/fluoride-in-drinking-water-a-scientific-review-of-epas-standards

the body's homeostasis, such as cellular metabolism, reproduction, sexual development, sugar and mineral homeostasis, heart rate, and digestion. Research has only begun to glimpse into fluoride's effects on these systems; however, we have enough evidence to confidently state fluoride is an endocrine toxicant, a disruptor. Current research supports the NRC (2006) conclusion and provides greater evidence to establish a least observable effect with margin of safety. The question is no longer whether fluoride is safe, the question is "like lead, is any dosage of fluoride is safe for everyone?"

The endocrine system is closely connected to the neurological system such as through neurosecretors which release neurotransmitters into the blood through extracellular fluids. We may consider three major classes of molecules that function as hormones in vertebrates: 1. water soluble peptide hormones such as epinephrine, 2. lipid soluble/fluid hormones with receptor on the nucleus of target cells which turns on transcription quickly such as testosterone, 3. local regulators/paracrine signaling which convey messages between neighboring cells such as cytokines (immune response). Numerous hormones such as ADH, FSH, LH, ACTH, growth hormones, pituitary hormones, pancreatic hormones, insulin IGF, hypo- and hyperthyroidism, insulin (diabetes), glucagon, adrenal glands, need to be considered individually, synergistically, and as they effect the entire human body. We must not leave the public at risk, waiting for the patients (public) to provide absolute proof of harm, such as prospective randomized controlled trials of lower IQ, before governments stop mass medication of fluoride without consent for a nonlethal and noncontagious disease prevented with good hygiene and diet.

We have a null probability of fluoride being safe for everyone at EPA's MCL, especially when in combination with synergistic toxicants, compromised endocrine systems, or various ages and stages of life and at concentrations greater than mother's milk which in most samples has no detectible fluoride (mean 0.004 ppm or about 0.001 mg/Kg/day) and the longest running fluoride research project known. Until we have robust research proving the level of fluoride in mother's milk is deficient, incomplete, or defective; mother's milk should be the normative model against which all other infant formulas should be compared, **<0.001 mg/Kg/day.** Most infants (80%-90%) receive some or all formula usually reconstituted with public water resulting in about 175 to 250 times more fluoride than mother's milk, mean of 0.004 ppm. (most samples not detectible)

Therefore, the evidence of mother's milk may not fit into a formula, rubric or matrix but the weight of evidence should be used for common sense judgment. Judgment, keeping in mind the insufficient evidence of benefit, lack of individual informed consent and weight of all evidence of risks for each individual, not just the mean or 90th

percentile. Fluoride is an endocrine disruptor and should be treated as a toxicant like lead.

Consider Nakamoto (2018)[4] "Fluoride Exposure in Early Life as the Possible Root Cause of Disease In Later Life."

Mechanism of action

Fluorine enters the body by ingestion, respiration and skin absorption. Exposed tissues are utilized by HF in neutralization reactions leaving the fluoride ion free to pass further into the body. The fluoride anion reacts with HCl in the stomach to form HF. HF is then absorbed by the GI tract and passes into the liver via the portal vein. Elemental F is one of the strongest oxidizers currently known. The anion is immune to the body's first line of defense of biotransformation, phase 1 metabolic reactions, which are generally oxidative reactions in the liver. HF passes into the blood stream and to all tissues. Calcium in all tissues reacts with HF to form an insoluble salt, calcium fluoride. Calcium fluoride is cleared by the body, leaching out some calcium which would be part of the bones, teeth, pineal gland, nerves, etc. The process results in increased density and brittleness, compressive strength of bones and teeth, with decreased tinsel strength.

"Normal" serum concentrations are vague. In part, because there is no "optimal" serum fluoride concentration, and no "optimal" tooth fluoride concentration. Teeth with and without dental caries have the same range of fluoride concentrations. The CDC suggests, *"Normal serum fluoride levels are <20 mcg/L (**0.02 ppm**) but varies substantially. . . ."*[5] We will see below, 0.02 ppm serum fluoride is not protective. Researchers have reported various serum fluoride concentrations in studies for their "controls." It is not unusual for studies which report harm to have controls assuming "normal" with fluoride serum concentrations higher than 0.02 ppm.

Taves ('66)	normal <0.013 ppm
Sowers	controls 0.05 ppm (4th quartile)
Sandhu	controls 0.042 ppm and tumors at 0.072 ppm (Xiang 0.064 ppm)
Zang	controls 0.04 ppm and 8 IQ loss 0.08 ppm
Rathe	controls 0.025 ppm and stones at 0.12 ppm
Hossney Mother's Milk	0.000 most samples - none detected

If controls had been <0.02 ppm, greater significance might have been reported.

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[4] Nakamoto T, Rawls HR. Fluoride Exposure in Early Life as the Possible Root Cause of Disease In Later Life. J Clin Pediatr Dent. 2018;42(5):325-330. doi: 10.17796/1053-4625-42.5.1. Epub 2018 May 15. PMID: 29763350.

[5] http://www.bt.cdc.gov/agent/sulfurylfluoride/casedef.asp

Keep in mind, birth control has efficacy at parts per billion. We report fluoride here in parts per million.

II. BIAS OF FLUORIDE EFFICACY

Bias sneaks into research and evaluations of research in several forms. Our nominations for cancer and neurologic harm provided a few studies on fluoride's lack of benefit and should be reviewed. A humble attitude should be taken, remembering "our knowledge is finite, our ignorance infinite."

Ben Goldacre suggests,[6] "Medicine shouldn't be about authority, and the most important question anyone can ask on any claim is simple: 'how do you know?'" Fluoridation of public water is a web of guesses, assumptions and beliefs. Healthcare is littered with the use of treatments that are based on habit, firmly held beliefs and policy rather than evidence. Several medical treatments and research studies were started in the 40's and 50's which lacked scientific rigor evaluating risks, such as artificial fluoridation, thalidomide, and the US Public Health Service Tuskegee experiments on syphilis,[7] Vioxx, Avandia, Herceptin, diethylstilbestrol, are further recent examples.

Another bias is the "natural" ebb and flow of diseases and natural resolution of disease. Dentists seldom see dental caries resolve on their own. If we see caries, we treat. Dentists tend to approach prevention with the same arbitrary mind set. However, prevention and good health are frustratingly less in our control and arbitrary than dental treatment, and less lucrative. Comparing developed countries finds caries have been reduced the same amount regardless of fluoridation. Fair tests, prospective RCT studies of efficacy need to be done rather than assumptions. OHAT must not assume fluoride ingestion mitigates dental caries. RCT studies are possible.

> "Our many errors show that the practice of causal inference. . . remains an art. Although to assist us, we have acquired analytic techniques, statistical methods and conventions, and logical criteria, ultimately the conclusions we reach are a matter of judgement."[8]

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[6] http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0050892/pdf/TOC.pdf "Testing Treatments Better Research For Better Healthcare, 2nd Ed. Imogen Evans et al. 2011.

[7] http://www.tuskegee.edu/about_us/centers_of_excellence/bioethics_center/about_the_usphs_syphilis_study.aspx

[8] Susser M. *Causal thinking in the health sciences,* Oxford: Oxford University Press, 1983. As quoted in "Testing Treatments Better Research For Better Healthcare, 2nd Ed.

The NRC (2006) review of fluoride in water used a “weight of evidence” approach. Without any prospective RCT studies, a “weight of evidence” approach is reasonable.

Patients of healthcare should be participants rather than recipients. Doctors and public health professionals are in error when they attempt to dispense health through chemistry under police powers. Professionals are more effective for good overall health when they dispense information for collaboration in better health. “Education, not Fluoridation.”

The assumption of ingested fluoride’s efficacy has biased public health policy and scientific evaluation. We have misled ourselves and need fair tests of the evidence. Studies funded by those with vested interests are four times more likely to have a positive result. Many desire miracle cures. The marketing claim of fluoride “preventing” caries is just marketing. If ingested fluoride has any benefit, the term mitigating, rather than “preventing” would be more appropriate.

The CDC funded (Caution: vested interest and potential bias) a 2015 Cochrane study[9] on the efficacy of fluoridation. The Cochrane study includes:

> “Although these results indicate that water fluoridation is effective at reducing levels of tooth decay in children’s baby and permanent teeth, the applicability of the results to current lifestyles is unclear because the majority of the studies were conducted before fluoride toothpastes and the other preventative measures were widely used in many communities around the world.”
>
> “There was insufficient information available to find out whether the introduction of a water fluoridation programme changed existing differences in tooth decay across socioeconomic groups.”
>
> “There was insufficient information available to understand the effect of stopping water fluoridation programmes on tooth decay.”
>
> “No studies met the review’s inclusion criteria that investigated the effectiveness of water fluoridation for preventing tooth decay in adults, rather than children.”

The Cochrane report should have used only RCT studies. But since there are none, the best available where are prior to fluoride toothpaste and other preventive measures. The lack of quality studies for the only mass medication should sound the alarm. Yes, they threw bones to everyone, supporting the funders of their study, the CDC, by saying fluoridation is “effective” and yet support most developed countries which do not

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[9] Iheozor-Ejiofor Z, Worthington HV, Walsh T, O’Malley L, Clarkson JE, Macey R, Alam R, Tugwell P, Welch V, Glenny A, Water fluoridation to prevent tooth decay, Cochrane Review, June 18, 2015

fluoridate by suggesting “applicability” is unclear. . . which scientifically means what? And, Cochrane used relative percentages rather than absolute percentage. In other words, a 25% relative percentage sounds bigger than a <1% absolute percentage. A decrease from two cavities to 1.5 cavities is a relative 25% decrease. Out of 128 possible cavities, a decrease of half a cavity is less than an absolute 1% decrease.

The Cochrane (2015) study is consistent with the FDA withdrawing approval of ingested fluoride supplements in 1975, for lack of evidence of efficacy.

For decades, calls for high quality research have been made and to date not one has been published. Proponents of fluoride ingestion have claimed RCT studies are not possible, a poor excuse. Some communities such as in Alaska have water trucked to them and these could be studied. The greatest obstacle for approval of an RCT study might be acceptance by a human studies ethics review board. And if a controlled study is unethical, the same act as policy is no more ethical. *“Absence of evidence is not evidence of absence or evidence of safety.”*

CDC: “Ingestion of fluoride is not likely to reduce tooth decay.”[10]

CDC: “. . . fluoride prevents dental caries predominately after eruption of the tooth into the mouth, and its actions primarily are topical for both adults and children…”[11]

“**Systemic Fluoride has theoretical benefit while the enamel is developing, up to age 6-8.**”[12]

It makes no sense to medicate everyone with artificially fluoridated water to theoretically benefit about 10% of the population while 41% of children have dental fluorosis, a biomarker of excess fluoride exposure, for a non contagious almost never lethal disease, without patient consent.

Dental caries is not the result of inadequate fluoride ingestion and no physiologic process requires fluoride. For those wishing to ingest fluoride, other sources of fluoride ingestion (such as toothpaste) are available.

PERSPECTIVE: The EPA’s proposed RfD will increase from 0.06 mg/kg/day to **0.08** mg/kg/day. In other words, the EPA is doing the opposite of the NRC recommendation. The NRC (2006 p. 222) reported: “Impaired glucose tolerance in humans has been reported in separate studies at fluoride intakes of **0.07**-0.4 mg/kg/day, . . . The primary mechanism appears to involve inhibition of insulin production.” Mother’s milk has mean dosage of **<0.001 mg/kg/day.**

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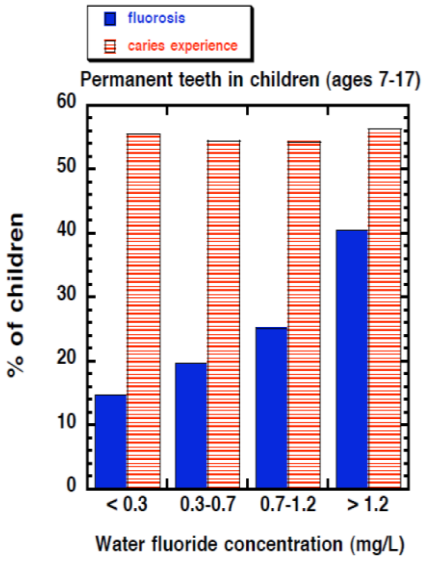
[10] (1999). Achievements in Public Health, 1900-1999: Fluoridation of Drinking Water to Prevent Dental Caries. MMWR, 48(41); 933-940, October 22

[11] IBID

[12] NRC 2006 & HHS HTSDR 2003 p 9

Vida & Kumar (2009) *"CONCLUSION: The results of this study suggest that teeth with fluorosis were more resistant to caries in U.S. schoolchildren than were teeth without fluorosis. Our results highlight the need for those considering policies regarding reduction of fluoride exposure to take into consideration the caries-preventive benefits associated with milder forms of enamel fluorosis."*

lida, H., and Kumar, J.V. 2009. The association between enamel fluorosis and dental caries in U.S. schoolchildren. JADA 140:855-862.



However, graphing lida and Kumar data demonstrates dental fluorosis does increase with more fluoride, but a discouragingly almost undetectable caries difference, well within the effects of confounding factors. Slightly more fluoride increases caries above the low fluoride. Risks increase, benefit is negligible.

Most studies evaluating the risks of fluoride are animal studies and use fluoride at higher concentrations than water fluoridation. Humans are significantly more sensitive to fluoride than rodents and an uncertainty factor of 100 is recommended. As a rough estimate, any study using 100 ppm fluoride or less on rodents raises concerns for humans (fluoridated water represents about half of human fluoride exposure).

A number of studies are published each year on fluoride's harm and studies provided here are not a definitive list. The reader should use judgment to put the weight of significance to each study and look the study up to read the full study. Abstracts often show bias.

Vandenberg et al. (2012)[13] included sodium fluoride in a list of endocrine disrupting chemicals (EDCs) with low-dose effects. They noted the EDC action of sodium fluoride as: "Inhibits insulin secretion, PTH, TH." The Vanderberg et al. paper was cited in a larger report, Science of Endocrine Disrupting Chemicals – 2012, co-published in January 2013 by the United Nations Environment Programme and the World Health Organization – see page 13

[13] Laura N. Vandenberg, Theo Colborn, Tyrone B. Hayes, Jerrold J. Heindel, David R. Jacobs, Jr., Duk-Hee Lee, Toshi Shioda, Ana M. Soto, Frederick S. vom Saal, Wade V. Welshons, R. Thomas Zoeller, and John Peterson Myers Hormones and Endocrine-Disrupting Chemicals: Low-Dose Effects and Nonmonotonic Dose Responses. Endocrine Reviews. First published ahead of print March 14, 2012 as doi:10.1210/er.2011-1050

III. NATIONAL RESEARCH COUNCIL 2006 "FLUORIDE IN DRINKING WATER: A SCIENTIFIC REVIEW OF EPA'S STANDARDS"

NRC (2006) report, in part, is included in sections here. Their review, although historic, is still the most definitive on the relationship between fluoride and the endocrine system. This section is quoted directly from the NRC (2006) report, starting page 214.

"OTHER ENDOCRINE ORGANS

> "The effects of fluoride exposure have been examined for several other endocrine organs, including the adrenals, the pancreas, and the pituitary (for details, see Appendix E, Tables E-16 and E-17). Effects observed in animals include changes in organ weight, morphological changes in tissues, increased mitotic activity, decreased concentrations of pituitary hormones, depressed glucose utilization, elevated serum glucose, and elevated insulin-like growth factor-1 (IGF-1). Effects reported in humans include "endocrine disturbances," impaired glucose tolerance, and elevated concentrations of pituitary hormones. Studies of the effects of fluoride on glucose metabolism and in diabetic animals are discussed below; information on other effects is extremely limited.

"Animal Studies (Diabetic Animals)

> "Two studies have examined the effects of fluoride exposure in diabetic rats. In the first study, Dunipace et al. (1996) compared male Zucker fatty diabetic rats and Zucker age-matched controls given drinking water with fluoride at 5, 15, or 50 mg/L. [These fluoride intakes were considered to be equivalent to intakes by humans of 1, 3, and 10 mg/L (Dunipace et al. 1996).] For the physiological, biochemical, and genetic variables that were monitored, no "measurable adverse effects" were noted. Statistically significant differences with respect to fluoride intake (as opposed to differences between normal and diabetic animals) were observed only for diabetic rats with fluoride at 50 mg/L. No endocrinological parameters (e.g., PTH) were measured. Dunipace et al. (1996) reported that fluoride intake, excretion, and balance were generally similar in this study and in a previous study with Sprague-Dawley rats but that there were "strain-specific differences in fluoride sensitivity"; these differences were not defined or explained. The Zucker fatty diabetic rat is considered to be an animal model for human Type II (noninsulin-dependent) diabetes mellitus, although the diabetic rats in this study did not experience renal insufficiency, and the study was terminated before an age that might be more comparable to ages associated with late-onset diabetes and diabetic complications in humans. The authors concluded that the diabetic rats "were not at increased risk of fluorosis," even though femoral fluoride concentrations (2,700-9,500 µg/g in ash for diabetic rats given fluoride at 15 or 50 mg/L versus 2,500-3,600 in normal rats given fluoride at 50 mg/L) were in the range associated with fluorosis in humans and exceeded concentrations of bone fluoride associated with decreased bone strength in rabbits (6,500-8,000 ppm in ash; Turner et al. 1997); no basis for their conclusion was given.

“In the second study, Boros et al. (1998) compared the effects of fluoride at 10 mg/L in drinking water for 3 weeks on young female rats (Charles River, Wistar), either normal (nondiabetic) or with streptozotocin-induced, untreated diabetes. An additional group of normal rats was given an amount of fluoride in drinking water corresponding to the fluoride intake by the diabetic rats (up to about 3 mg/day per rat). Both feed and water consumption increased significantly in the diabetic rats (with and without fluoridated water); water consumption was significantly higher in the diabetic rats on fluoridated water than in those on nonfluoridated water. Fasting blood glucose concentrations were increased significantly in both diabetic groups, but more so in the group on fluoridated water. Fluoride treatment of nondiabetic animals did not cause any significant alteration in blood glucose concentrations. Plasma fluoride was higher, and bone fluoride was lower, in diabetic than in nondiabetic animals given the same amount of fluoride, indicating lower deposition of fluoride into bone and lower renal clearance of fluoride in the diabetic animals. The increased kidney weight found in diabetic animals on nonfluoridated water was not seen in the fluoride-treated diabetic animals. Additional biochemical and hormonal parameters were not measured.

“In contrast to the Zucker fatty diabetic rats in the study by Dunipace et al. (1996), the streptozotocin-induced diabetic rats in this study (Boros et al., 1998) provide an animal model considered representative of Type I (insulin-dependent) diabetes mellitus in humans. In these rats, the general severity of the diabetes (blood glucose concentrations, kidney function, weight loss) was worse in animals given fluoride at 10 mg/L in their drinking water. In both types of diabetic rats, fluoride intake was very high because of the several-fold increase in water consumption, and corresponding plasma, soft tissue, and bone fluoride concentrations were elevated accordingly. Thus, any health effects related to plasma or bone fluoride concentrations, for example, would be expected to occur in animals or humans with uncontrolled (or inadequately controlled) diabetes at lower fluoride concentrations in drinking water than for nondiabetics, because of the elevated water intakes. In addition, the results reported by Boros et al. (1998) suggested that, for some situations (e.g., diabetes in which kidney function is compromised), the severity of the diabetes could be increased with increasing fluoride exposure.

“Animal Studies (Normal Animals)

“Turner et al. (1997) reported a 17% increase in serum glucose in female rabbits given fluoride in drinking water at 100 mg/L for 6 months. IGF-1 was also significantly increased (40%) in these rabbits, but other regulators of serum glucose, such as insulin, were not measured. The authors suggested that IGF-1 concentrations might have changed in response to changes in serum glucose concentrations. Dunipace et al. (1995, 1998) found no significant differences with chronic fluoride treatment in mean blood glucose concentrations in rats; specific data by treatment group were not reported, and parameters such as insulin and IGF-1 were not measured.

"Suketa et al. (1985) and Grucka-Mamczar et al. (2005) have reported increases in blood glucose concentrations following intraperitoneal injections of NaF; Suketa et al. (1985) attributed these increases to fluoride stimulation of adrenal function. Rigalli et al. (1990, 1992, 1995), in experiments with rats, reported decreases in insulin, increases in plasma glucose, and disturbance of glucose tolerance associated with increased plasma fluoride concentrations. The effect of high plasma fluoride (0.1-0.3 mg/L) appeared to be transient, and the decreased response to a glucose challenge occurred only when fluoride was administered before (as opposed to together with or immediately after) the glucose administration (Rigalli et al. 1990). In chronic exposures, effects on glucose metabolism occurred when plasma fluoride concentrations exceeded 0.1 mg/L (5 µmol/L) (Rigalli et al. 1992, 1995). The in vivo effect appeared to be one of inhibition of insulin secretion rather than one of insulin-receptor interaction (Rigalli et al. 1990). Insulin secretion (both basal and glucose-stimulated) by isolated islets of Langerhans in vitro was also inhibited as a function of fluoride concentrations (Rigalli et al. 1990, 1995). Rigalli et al. (1990) pointed out that recommended plasma fluoride concentrations for treatment of osteoporosis are similar to those shown to affect insulin secretion.

"Human Studies

"Jackson et al. (1994) reported no differences in mean fasting blood glucose concentrations between osteoporosis patients treated with fluoride and untreated controls, although 3 of 25 treated individuals had values outside the normal range (versus 1 of 38 controls). No significant differences were found between groups of older adults with different fluoride concentrations in drinking water in studies in China (Li et al. 1995; subjects described as "healthy" adults) and the United States (Jackson et al. 1997), and all mean values were within normal ranges. [In the study by Jackson et al. (1997), samples were nonfasting; in the study by Li et al. (1995), it is not clear whether samples were fasting or nonfasting.] Glucose tolerance tests were not conducted in these studies.

"Trivedi et al. (1993) reported impaired glucose tolerance in 40% of young adults with endemic fluorosis, with fasting serum glucose concentrations related to serum fluoride concentrations; the impaired glucose tolerance was reversed after 6 months of drinking water with "acceptable" fluoride concentrations (<1 mg/L). It is not clear whether individuals with elevated serum fluoride and impaired glucose tolerance had the highest fluoride intakes of the group with endemic fluorosis or a greater susceptibility than the others to the effects of fluoride. For all 25 endemic fluorosis patients examined, a significant positive correlation between serum fluoride and fasting serum immunoreactive insulin (IRI) was observed, along with a significant negative correlation between serum fluoride and fasting glucose/insulin ratio (Trivedi et al. 1993).

"The finding of increased IRI contrasts with findings of decreased insulin in humans after exposure to fluoride (Rigalli et al. 1990; de la Sota et al. 1997) and inhibition of insulin secretion by rats, both in vivo and in vitro (Rigalli et al. 1990, 1995). However, the assay for IRI used by Trivedi et al. (1993) could not distinguish between insulin

and proinsulin, and the authors suggested that the observed increases in both IRI and serum glucose indicate either biologically inactive insulin—perhaps elevated proinsulin—or insulin resistance. Inhibition of one of the prohormone convertases (the enzymes that convert proinsulin to insulin) would result in both elevated proinsulin secretion and increased blood glucose concentrations and would be consistent with the decreased insulin secretion reported by Rigalli et al. (1990, 1995) and de la Sota et al. (1997). Although Turner et al. (1997) suggested fluoride inhibition of insulin-receptor activity as a mechanism for increased blood glucose concentrations, Rigalli et al. (1990) found no difference in response to exogenous insulin in fluoride-treated versus control rats, consistent with no interference of fluoride with the insulin-receptor interaction.

“Discussion (Other Endocrine Function)

“More than one mechanism for diabetes or impaired glucose tolerance exists in humans, and a variety of responses to fluoride are in keeping with variability among strains of experimental animals and among the human population. The conclusion from the available studies is that sufficient fluoride exposure appears to bring about increases in blood glucose or impaired glucose tolerance in some individuals and to increase the severity of some types of diabetes. In general, impaired glucose metabolism appears to be associated with serum or plasma fluoride concentrations of about 0.1 mg/L or greater in both animals and humans (Rigalli et al. 1990, 1995; Trivedi et al. 1993; de al Sota et al. 1997). In addition, diabetic individuals will often have higher than normal water intake, and consequently, will have higher than normal fluoride intake for a given concentration of fluoride in drinking water. An estimated 16-20 million people in the U.S. have diabetes mellitus (Brownlee et al. 2002; Buse et al. 2002; American Diabetes Association 2004; Chapter 2); therefore, any role of fluoride exposure in the development of impaired glucose metabolism or diabetes is potentially significant.

“SUMMARY

“The major endocrine effects of fluoride exposures reported in humans include elevated TSH with altered concentrations of T3 and T4, increased calcitonin activity, increased PTH activity, secondary hyperparathyroidism, impaired glucose tolerance, and possible effects on timing of sexual maturity; similar effects have been reported in experimental animals. These effects are summarized in Tables 8-1 and 8-2, together with the approximate intakes or physiological fluoride concentrations that have been typically associated with them thus far. Table 8-2 shows that several of the effects are associated with average or typical fluoride intakes of 0.05-0.1 mg/kg/day (0.03 with iodine deficiency), others with intakes of 0.15 mg/kg/day or higher. A comparison with Chapter 2 (Tables 2-13, 2-14, and 2-15) will show that the 0.03-0.1 mg/kg/day range will be reached by persons with average exposures at fluoride concentrations of 1-4 mg/L in drinking water, especially the children. The highest intakes (> 0.1 mg/kg/d) will be reached by some individuals with high water intakes at 1 mg/L and by many or most individuals with high water intakes at 4 mg/L, as well as by young children with average exposures at 2 or 4 mg/L.

“Most of the studies cited in this chapter were designed to ascertain whether certain effects occurred (or in cases of skeletal fluorosis, to see what endocrine disturbances might be associated), not to determine the lowest exposures at which they do occur or could occur. Estimates of exposure listed in these tables and in Appendix E are, in most cases, estimates of average values for groups based on assumptions about body weight and water intake. Thus, individual responses could occur at lower or higher exposures than those listed. Although the comparisons are incomplete, similar effects are seen in humans at much lower fluoride intakes (or lower water fluoride concentrations) than in rats or mice, but at similar fluoride concentrations in blood and urine. This is in keeping with the different pharmacokinetic behavior of fluoride in rodents and in man (Chapter 3) and with the variability in intake, especially for humans.”

IV. THYROID, PARATHYROID, PANCREAS, PINEAL, ADRENAL, GONADS, ENTEROENDOCRINE, PARAGANGLIA, ANTERIOR AND POSTERIOR PITUITARY, AND PLACENTA.

NRC (2006) “In summary, evidence of several types indicates that fluoride affects normal endocrine function or response; the effects of the fluoride-induced changes vary in degree and kind in different individuals. **Fluoride is therefore an endocrine disruptor** in the broad sense of altering normal endocrine function or response. The mechanisms of action remain to be worked out and appear to include both direct and indirect mechanisms, for example, direct stimulation or inhibition of hormone secretion by interference with second messenger function, indirect stimulation or inhibition of hormone secretion by effects on things such as calcium balance, and inhibition of peripheral enzymes that are necessary for activation of the normal hormone.” (page 266). (National Research Council, 2006) (Emphasis supplied)

A. THYROID GLAND:

Metabolic active cells in the body require hormones produced by the thyroid gland, triiodothyronine (T3) and thyroxine (T4). Health consequences arise when the thyroid produces too much, or too little, of these hormones.

At relatively low doses fluoride is effective at reducing thyroid function in the hyperthyroid patients. Research confirms that (1) fluoride can exacerbate the anti-thyroid effects of iodine deficiency, (2) can cause goiter in some individuals, and (3) can alter thyroid hormone levels in a manner consistent with a general thyroid suppressant. Until the 1950s, doctors in Europe and South America prescribed fluoride for hyperthyroidism. (Merck Index 1968). Fluoride therapy did reduce thyroid activity in the treated patients. (McClaren 1969; Galletti 1958; May 1937). Clinical indications suggested 2 to 5 mg of sodium fluoride per day over several months was effective, (Galletti & Joyet 1958). Note: a person drinking 3 liters of fluoridated water at 0.7 ppm with NO other fluoride source, would receive a clinical dosage to reduce thyroid activity. A comparable proposed EPA safe dosage RfD of 0.08 mg/kg/day would exceed clinically used dosages. (0.08 mg/kg X 50 kg = 4 mg. For a 100 kg person, 0.08 mg/kg X 100 kg = 8 mg fluoride). Some ADD medications still contain fluoride.

Alterations in thyroid hormones, including reduced T3 and increased TSH, in populations exposed to elevated levels of fluoride in the workplace or in the water have been reported. (NRC 2006; Susheela 2005; Mikhailets 1996; Yao 1996; Bachinskii 1985; Yu 1985).

In **clinical hypothyroidism,** the thyroid gland fails to produce sufficient quantities of the hormones triiodothyronine (T3) and thyroxine (T4). Reduced T3 and T4 can contribute to fatigue, muscle/joint pain, depression, weight gain, menstrual disturbances, impaired

fertility, impaired memory, and inability to concentrate. When T3 and T4 levels begin to fall, the pituitary gland responds by increasing production of “Thyroid Stimulating Hormone” (TSH) as a means of getting the thyroid to produce more T3 and T4.

In **subclinical hypothyroidism,** TSH levels decrease but T3 and T4 hormones are in a normal range. Subclinical hypothyroidism in pregnant women results in reduced IQ in offspring, (Klein 2001; Haddow 1999), and a recent study in the Journal of the American Medical Association found that adults with subclinical hypothyroidism had a significantly higher rate of coronary heart disease. (Rodondi 2010).

Dental fluorosis is a poor indicator of fluoride’s effect on they thyroid gland.

Thyroid Hormone Levels Based on Severity of Dental Fluorosis (Hosur 2012).

In 2006, the NRC report on fluoride for the EPA suggested studies investigating fluoride’s impact on thyroid hormone levels have produced divergent findings, but are consistent with fluoride having an anti-thyroid effect under certain circumstances. Singh (2014 see Human Thyroid below) may in part explain the “divergent findings” because dental fluorosis is a poor indication of TSH levels (see Table 3 below). **77% with dental fluorosis and 67% without dental fluorosis had derangement in thyroid hormone levels.** Both groups had abnormal serum fluoride levels and delayed eruption. **Even Group 2 drinking 0.02 ppm-0.77 ppm fluoride in water had 50% of children with abnormal serum fluoride levels.** Note: USPHS new recommendation of 0.7 ppm, represents a 14% reduction of fluoride exposure and is not enough.

Table 3

Derangement in Thyroid hormone (FT_3 , FT_4 , TSH) levels and serum fluoride levels in children of different groups

Group	No. of cases with Derangement in Thyroid hormone (FT_3, FT_4, TSH) level	No. of children with abnormal serum fluoride level	No. of children with delayed eruption
Group1A (n = 30)	23 (77%)	29 (97%)	17 (57%)
Group1B (n = 30)	20 (67%)	30 (100%)	15 (50%)
Group 2 (n = 10)	1 (10%)	5 (50%)	0 (0%)

The most common thyroid effect associated with fluoride exposure appears to be an increase in TSH levels, with or without a corresponding effect on T3 or T4. (Susheela 2005). One of the most recent studies, for example, found a trend towards higher TSH in children based on the severity of their dental fluorosis, but without a significant effect on either T3 or T4. (Hosur 2012, see figure below). These and other findings indicate that fluoride can contribute to a subclinical, if not clinical, hypothyroid condition. It remains difficult to predict the toxic dose, however, as it appears to depend, in part, on the nutritional and health status of the individual, particularly the adequacy of iodine intake. (NRC 2006).

NRC (2006) page 218. "Thyroid Function

"Fluoride exposure in humans is associated with elevated TSH concentrations, increased goiter prevalence, and altered T4 and T3 concentrations; similar effects on T4 and T3 are reported in experimental animals, but TSH has not been measured in most studies. In animals, effects on thyroid function have been reported at fluoride doses of 3-6 mg/kg/day (some effects at 0.4-0.6 mg/kg/day) when iodine intake was adequate (Table 8-1); effects on thyroid function were more severe or occurred at lower doses when iodine intake was inadequate. In humans, effects on thyroid function were associated with fluoride exposures of 0.05-0.13 mg/kg/day when iodine intake was adequate and 0.01-0.03 mg/kg/day when iodine intake was inadequate (Table 8-2).

"Several sets of results are consistent with inhibition of deiodinase activity, but other mechanisms of action are also possible, and more than one might be operative in a given situation. In many cases, mean hormone concentrations for groups are within normal limits, but individuals may have clinically important situations. In particular, the inverse correlation between asymptomatic hypothyroidism in pregnant mothers and the IQ of the offspring (Klein et al. 2001) is a cause for concern. The recent decline in iodine intake in the United States (CDC 2002d; Larsen et al. 2002) could contribute to increased toxicity of fluoride for some individuals."

NRC (2006) Tables 8-1 and 8-2 are reproduced here.

TABLE 8-1 Summary of Major Observed Endocrine Effects of Fluoride in Experimental Animals, with Typical Associated Intakes and Physiological Fluoride Concentrations

End Point	Fluoride Intake, mg/kg/day	Fluoride in Serum or Plasma, mg/L	Fluoride in Urine, mg/L	Fluoride in Bone, ppm in ash	Key References
Altered thyroid function (altered T4 and T3 concentrations)	3-6 (lower with iodine deficiency)	NA[a]	≥ 6 (possibly ≥ 2-3)	≥2,400	Stolc and Podoba 1960; Bobek et al. 1976; Hillman et al. 1979; Guan et al. 1988; Zhao et al. 1998; Cinar and Selcuk 2005
Altered calcitonin activity	2	NA	NA	3,200-3,500[b]	Rantanen et al. 1972
Altered melatonin production; altered timing of sexual maturity	3.7	NA	NA	2,800	Luke 1997
Inhibited parathyroid function	5.4	NA	NA	NA	Rosenquist et al. 1983
Increased serum glucose; increased severity of diabetes	7-10.5	0.1-0.7[c,d]	NA	>1,000	Rigalli et al. 1990, 1992, 1995; Turner et al. 1997; Boros et al. 1998
Increased parathyroid hormone concentrations, secondary hyperparathyroidism	9-10	≥ 0.2[c]	NA	2,700-3,200	Faccini and Care 1965; Chavassieux et al. 1991

[a]Not available.
[b]ppm.
[c]Serum.
[d]Plasma.

The NRC (2006) listed several limitations of the endocrine studies. More current research has included some of these limitations. One of the limitations is the interdependence of endocrine systems. The NRC (2006) p 223. "In addition, the different endocrine organs do not function entirely separately: thyroid effects (especially elevated TSH) may be associated with parathyroid effects (Stoffer et al. 1982; Paloyan Walker et al. 1997), and glucose metabolism may be affected by thyroid or parathyroid status (e.g., McCarty and Thomas 2003; Procopio and Borretta 2003; Cettour-Rose et

al. 2005). Adverse effects in individuals might occur when hormone concentrations are still in the normal ranges for a population but are low or high for that individual (Brucker-Davis et al. 2001; Belchetz and Hammond 2003). Some investigators suggest that endocrine-disrupting chemicals could be associated with nonmonotonic dose- response curves (e.g., U-shaped or inverted-U-shaped curves resulting from the superimposition of multiple dose-response curves) and that a threshold for effects cannot be assumed (Bigsby et al. 1999; Brucker-Davis et al. 2001)."

TABLE 8-2 Summary of Major Observed Endocrine Effects of Fluoride in Humans, with Typical Associated Intakes and Physiological Fluoride Concentrations

End Point	Fluoride Intake, mg/kg/day[a]	Fluoride in Serum or Plasma, mg/L	Fluoride in Urine, mg/L	Key References
Altered thyroid function (altered T4 and/or T3 concentrations)	0.05-0.1 (0.03 with iodine deficiency)	≥0.25[a]	2.4	Bachinskii et al. 1985; Lin et al. 1991; Yang et al. 1994; Michael et al. 1996; Susheela et al. 2005
Elevated TSH concentrations	0.05-0.1 (0.03 with iodine deficiency)	≥0.25[a]	≥2	Bachinskii et al. 1985; Lin et al. 1991; Yang et al. 1994; Susheela et al. 2005
Elevated calcitonin concentrations	0.06-0.87	0.11-0.26[b]	2.2-18.5 mg/day	Teotia et al. 1978
Goiter prevalence ≥ 20%	0.07-0.13 (≥ 0.01 with iodine deficiency)	NA[c]	NA	Day and Powell-Jackson 1972; Desai et al. 1993; Jooste et al. 1999
Impaired glucose tolerance in some individuals	0.07-0.4	0.08[a] 0.1-0.3[b]	2-8	Rigalli et al. 1990, 1995; Trivedi et al. 1993; de la Sota 1997
Increased parathyroid hormone concentrations, secondary hyperparathyroidism, in some individuals	0.15-0.87	0.14-0.45[b]	3-18.5 mg/day	Juncos and Donadio 1972; Teotia and Teotia 1973; Larsen et al. 1978; Teotia et al. 1978; Duursma et al. 1987; Dandona et al. 1988; Stamp et al. 1988, 1990; Pettifor et al. 1989; Srivastava et al. 1989; Dure-Smith et al. 1996; Gupta et al. 2001

[a]Serum.
[b]Plasma.
[c]Not available.

Peckham (2015) "We found that higher levels of fluoride in drinking water provide a useful contribution for predicting prevalence of hypothyroidism. We found that practices located in the West Midlands (a wholly fluoridated area) are nearly twice as likely to report high hypothyroidism prevalence in comparison to Greater Manchester (non-fluoridated area)."

Zhang (2015)[14] (Note: although this study focused on decrease in IQ with fluoride, thyroid hormone levels were also measured.) ". . . The children's IQ, fluoride contents in drinking water (W-F), serum (S-F), and urine (U-F); serum thyroid hormone levels, COMT Val158Met polymorphism, and plasma proteomic profiling were determined. . . . In conclusion, fluoride exposure was adversely associated with children's intelligence, whereas the COMT polymorphism may increase the susceptibility to the deficits in IQ due to fluoride exposure. Moreover, the proteomic analysis can provide certain basis for identifying the early biological markers of fluorosis among children."

[14] Zhang S, Zhang X, Liu H, Qu W, Guan Z, Zeng Q, Jiang C, Gao H, Zhang C, Lei R, Xia T, Wang Z, Yang L, Chen Y, Wu X, Cui Y, Yu L, Wang A. Modifying effect of COMT gene polymorphism and a predictive role for proteomics analysis in children's intelligence in endemic fluorosis area in Tianjin, China. Toxicol Sci. 2015 Apr;144(2):238-45. doi: 10.1093/toxsci/kfu311. Epub 2015 Jan 1.

A critical study to consider is Singh (2014) which raised serious concerns that **dental fluorosis is a poor indication of excess total fluoride exposure.** Both those with and without dental fluorosis had thyroid derangement and high serum fluoride concentrations.

Singh (2014)[15] "The study was undertaken to determine serum/urinary fluoride status and comparison of free T4, free T3 and thyroid stimulating hormone levels of 8 to 15 years old children with and without dental fluorosis living in an endemic and non-endemic fluorosis area. . . A significant relationship of water fluoride to urine and serum fluoride concentration was seen. The serum fluoride concentration also had significant relationship with thyroid hormone (FT3/FT4) and TSH concentrations. The testing of drinking water and body fluids for fluoride content, along with FT3, FT4, and TSH in children with dental fluorosis is desirable for recognizing underlying thyroid derangements and its impact on fluorosis. . . . Conclusion: The results of this study question the validity of the fluoridation of drinking water, milk, fruit juices, and salt by public health authorities and also the step taken to prevent ill effects of excess fluorine and iodine deficiencies in endemic fluorosis areas. The children with dental fluorosis living in endemic fluorosis areas may not have a frank thyroid disease due to excessive fluorine consumption but they do show thyroid disease leading to many health effect hence they require special care and attention."

And further, Singh (2014), "Group 1 included 60 male and female school children, which were equally divided into two subgroups: Group 1A (children with dental fluorosis) and Group 1B (children without dental fluorosis). Group 2 included 10 children from Sardarpura colony of Udaipur city, a non endemic area, which was taken as a control for the study samples."

Tables 1, 2, 3, and 6 of Singh (2014) are reproduced here.

Table 1: Comparing fluoride Group 1 A (dental fluorosis) and 1B (no fluorosis), with control Group 2 is consistent with other studies when urine and serum fluoride concentrations are compared with water fluoride concentrations, provided significant other sources such as fluoridated toothpastes are not in use.

[15] Singh N[1], Verma KG[2], Verma P[3], Sidhu GK[4], Sachdeva S[3]. A comparative study of fluoride ingestion levels, serum thyroid hormone & TSH level derangements, dental fluorosis status among school children from endemic and non-endemic fluorosis areas. Springerplus. 2014 Jan 3;3:7. doi: 10.1186/2193-1801-3-7. eCollection 2014.

Table 1

Levels of fluoride naturally ingested from drinking water and body fluids in different sample groups

Parameters	Group 1A	Group 1B	Group 2	Total
Water fluoride (WF)	1.6–5.1 ppm	1.6–5.5 ppm	0.98–1 ppm	0.98–5.5 ppm
Urine fluoride (UF)	0.24–8.9 ppm	0.4–7.79 ppm	0.19–1.01 ppm	0.24–8.9 ppm
Serum fluoride (SF)	0.02–0. 77 ppm	0.03–0.75 ppm	0.02–0.09 ppm	0.02–0.77 ppm

NOTE: The absence of dental fluorosis does not indicate lower or safe fluoride urine or serum concentrations.
NOTE: All three groups had some individuals with low serum and urine fluoride concentrations. The significant difference is those with high serum and urine fluoride concentrations.
And remember, endemic fluoride is usually CaF which is estimated at 800 times less toxic than NaF or HSF used for artificial fluoridation.

Table 2

Levels of thyroid hormones in all sample groups

Parameters	Group 1A	Group 1B	Group 2	Total
Free T_3 (FT_3)	1.1–4.39 pg/ml	1.2–4.57 pg/ml	1.90–4.13 pg/ml	1.1–4.57 pg/ml
Free T_4 (FT_4)	0.94–1.98 ng/dL	0.8–1.7 ng/dL	0.87–1.67 ng/dL	0.8–1.98 ng/dL
TSH	1.41–8.46 µIU/m	1.92–10.99 µIU/m	0.96–3.54 µIU/m	0.96–10.99 µIU/m

Table 6

Correlation analysis between fluoride content in body fluids and their effect FT3, FT4, TSH within Group 1

Parameters	Spearman rho analysis	FT_3	FT_4	TSH	WF	UF	SF
FT_3	'r'	1	-0.169	-0.252	-0.711	-0.388	-0.400
	p-value	-	0.196	0.052*	0.000**	0.002**	0.002**
FT_4	'r'	-0.169	1	-0.079	0.196	0.119	0.119
	p-value	0.196	-	0.547	0.134	0.365	0.366
TSH	'r'	-0.252	-0.079	1	0.151	0.079	0.552
	p-value	0.052*	0.547	-	0.250	0.550	0.000**
WF	'r'	-0.711	0.196	0.151	1	0.690	0.529
	p-value	0.000**	0.134	0.250	-	0.000**	0.000**
UF	'r'	-0.388	0.119	0.079	0.690	1	0.525
	p-value	0.002**	0.365	0.550	0.000**	-	0.000**
SF	'r'	-0.400	0.119	0.552	0.529	0.525	1
	p-value	0.002**	0.366	0.000**	0.000**	0.000**	-

*Correlation is significant at 0.05 levels (2 tailed), **Correlation is highly significant below 0.01 levels correlation coefficient(r).

Table 3 should be carefully considered and we graphed their Table 3 below. **Even with fluoride serum levels between 0.02 ppm and 0.09 ppm (1 ppm fluoride in water), 10% had derangement of the thyroid.** Remember, endemic fluoride is not as toxic as sodium fluoride or HFS, and second, rural villagers often use less fluoride toothpaste, dental and medical products or fluoride pesticides.

The CDC's recommendation of normal fluoride serum concentrations <0.02 ppm may not be protective and provides no margin of safety. A 0.7 ppm artificial fluoridation will not reduce serum fluoride concentrations to within CDC recommendations.

Table 3

Derangement in Thyroid hormone (FT_3, FT_4, TSH) levels and serum fluoride levels in children of different groups

Group	No. of cases with Derangement in Thyroid hormone (FT_3, FT_4, TSH) level	No. of children with abnormal serum fluoride level	No. of children with delayed eruption
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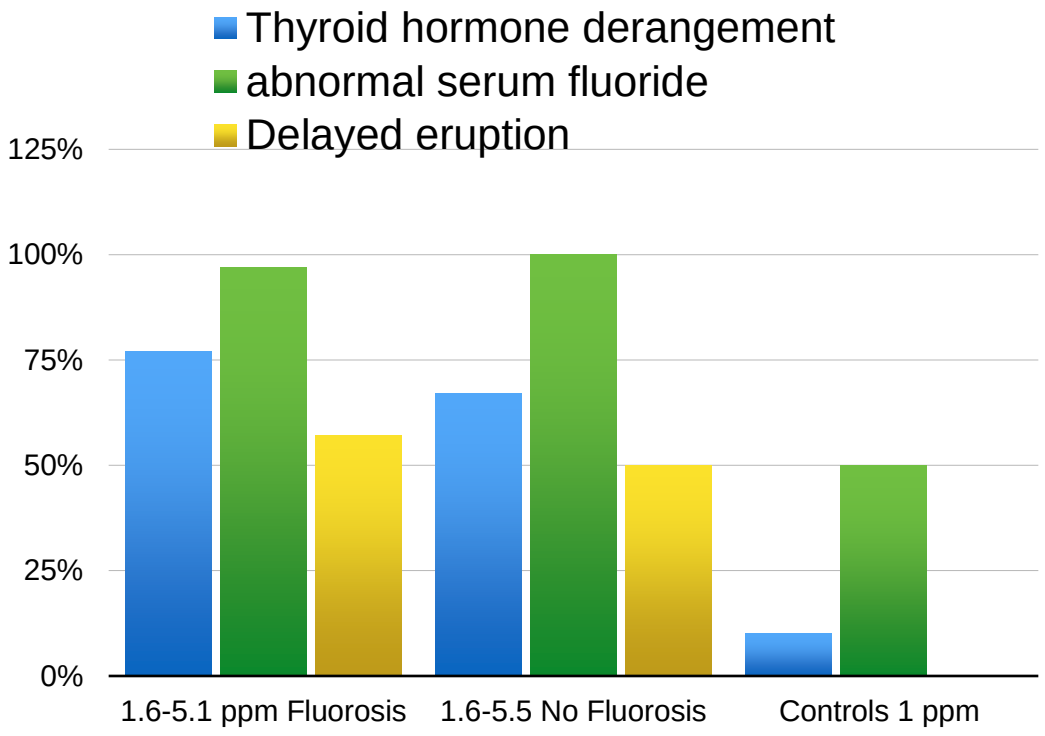
Group 2 (n = 10)	1 (10%)	5 (50%)	0 (0%)



Liu (2014)[16] "In many regions, excessive fluoride and excessive iodide coexist in groundwater, which may lead to biphasic hazards to human thyroid. To explore fluoride-induced thyroid cytotoxicity and the mechanism underlying the effects of excessive iodide on fluoride-induced cytotoxicity, a thyroid cell line (Nthy-ori 3-1) was exposed to excessive fluoride and/or excessive iodide. Cell viability, lactate dehydrogenase (LDH) leakage, reactive oxygen species (ROS) formation, apoptosis, and the expression levels of inositol-requiring enzyme 1 (IRE1) pathway-related molecules were detected. Fluoride and/or iodide decreased cell viability and increased LDH leakage and apoptosis. ROS, the expression levels of glucose-regulated protein 78 (GRP78), IRE1, C/EBP homologous protein (CHOP), and spliced X-box-binding protein-1 (sXBP-1) were enhanced by fluoride or the combination of the two elements. Collectively, excessive fluoride and excessive iodide have detrimental influences on human thyroid cells. Furthermore, an antagonistic interaction between fluoride and excessive iodide exists, and cytotoxicity may be related to IRE1 pathway-induced apoptosis."

Kutlucan (2013)[17] "AIM: To compare the urine iodine, fluoride, and to measure thyroid volumes in 10-15-year-old children using ultrasonography, a gold standard in evaluating thyroid volume. . . . After puberty, echobody index in subjects with fluorosis was markedly high. Based on our results, we thought that fluorosis increases thyroid volume in children with fluorosis after puberty."

TSH is considered a "'precise and specific barometer' of thyroid status in most situations" (NRC 2006) The relationship between fluoride and elevated TSH has been found even where T3 and T4 levels remain normal, suggesting that fluoride could contribute to subclinical hypothyroidism, which is a condition of "mild thyroid failure" marked by increased TSH and normal T3/T4.

Subclinical hypothyroidism is now considered a "clinically important disorder that has adverse clinical consequences." (Gencer 2012). Several studies have found that subclinical hypothyroidism in pregnant woman was a risk factor for reduced IQ in the offspring. (Klein 2001; Haddow 1999). Although most of the more than 40 human studies evaluating fluoride and IQ did not measure TSH, those that did so reported that children with high fluoride exposures had elevated TSH levels. (Wang 2001; Yao 1996; Lin 1991). Lin reported that elevated TSH correlated with reduced IQ. TSH levels could be one of the contributing factors towards the reduced IQ reported in the studies to date.

In 2010, a study in the Journal of the American Medical Association found that adults with subclinical hypothyroidism had a significantly higher incidence of, and mortality from, coronary heart disease. (Rodondi 2010). Whether this could help explain the relationship between elevated fluoride and cardiovascular disease remains to be

[16] Liu H, Zeng Q, Cui Y, Yu L, Zhao L, Hou C, Zhang S, Zhang L, Fu G, Liu Y, Jiang C, Chen X, Wang A. The effects and underlying mechanism of excessive iodide on excessive fluoride-induced thyroid cytotoxicity. Environ Toxicol Pharmacol. 2014 Jul;38(1):332-40. doi: 10.1016/j.etap.2014.06.008. Epub 2014 Jun 27.

[17] Kutlucan A[1], Kale Koroglu B, Numan Tamer M, Aydin Y, Baltaci D, Akdogan M, Ozturk M, Vural H, Ermis F. The investigation of effects of fluorosis on thyroid volume in school-age children. Med Glas (Zenica). 2013 Feb;10(1):93-8.

determined. As reported below, one recent study (Karademir 2011) did find a relationship between fluoride exposure, thyroid levels, and cardiovascular indices, although TSH levels were not found to be elevated.

Banjo (2013) "The study investigated the role of Spirulina platensis in reversing sodium fluoride-induced thyroid, neurodevelopment and oxidative alterations in offspring of pregnant rats. . . . Fluoride-induced alterations in thyroid hormones, behaviour and increased oxidative stress. Spirulina augmented the displacement of fluoride, facilitated antioxidant formation, improved behaviour and protected Purkinje cells. Supplementing Spirulina during pregnancy could reduce the risk of fluoride toxicity in offspring."[18]

Karademir (2011)[19] "In this study we examined the deleterious effect of fluorosis on cardiovascular system including detailed ECG with dispersion analysis, echocardiography, and HRV with Holter analysis in children. We found statistically significant low T4 levels, hypocalcemia and hyponatremia, increased QT and QTc interval in children with dental fluorosis. Our results show that fluorosis might increase risk of arrhythmia indirectly, due to its hypocalcemic, hypernatremic, and hypothyroidism effects."

Ba (2009)[20] "The concentration of serum TSH of children from high fluoride and iodine area and high iodine area was higher than that of children from high fluoride area and control area. Conclusion: High fluoride and iodine increase the prevalence of goiter. High iodine increases the concentration of FT4. Fluoride can increase the concentration of FT4 under high iodine condition."

Ruiz-Pagan (2006)[21] "This study was designed to evaluate adverse health effects in adolescents from chronic exposure to various water fluoride concentrations in three communities located in Northern Mexico: Ciudad Juarez, Samalayuca, and Villa Ahumada. In these communities the fluoride concentration in water averages 0.3, 1.0, and 5.3 mg/L, respectively. The residents of Villa Ahumada have been exposed to excessive levels of fluoride in drinking water since their birth. . . . In Villa Ahumada, a significant inverse relationship was found between urine fluoride levels and stature; this association suggests that fluoride exposure may affect the teeth but also the growth of adolescents. Serum samples of these individuals showed

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[18] Banji D et al (2013) Investigation on the role of Spirulina platensis in ameliorating behavioural changes, thyroid dysfunction and oxidative stress in offspring of pregnant rats exposed to fluoride. 2013 Sep 1;140(1-2):321-31. doi: 10.1016/j.foodchem.2013.02.076. Epub 2013 Feb 28.

[19] Karademir S, et al. (2011). Effects of fluorosis on QT dispersion, heart rate variability and echocardiographic parameters in children. Anadolu Kardiyol Derg 11(2):150-55.

[20] Ba Y, et al. (2009). Effect of different fluoride and iodine concentration in drinking water on children's dental fluorosis and thyroid function. Chinese Journal of Public Health 25(8):942-43.

[21] Ruiz-Payan A. (2006). Chronic effects of fluoride on growth, blood chemistry and thyroid hormones in adolescents residing in three communities in Northern Mexico. *ETD Collection for University of Texas, El Paso.* Paper AAI3214004.
http://digitalcommons.utep.edu/dissertations/AAI3214004

elevated levels of alkaline phosphatase (ALP), potassium, magnesium, calcium, and phosphate, and decreased levels of thyroid hormone T3 and uric acid. These findings show that chronic exposure to high levels of fluoride have a definitive impact on the prevalence and severity of dental fluorosis, decreased stature, and decreased [] thyroid hormone secretion."

Susheela (2005)[22] "Although it has long been suggested that dental fluorosis is associated with IDD and thyroid dysfunction,7-9,14 this study, to our knowledge, is the first to investigate dental fluorosis in relation to TSH and the thyroid hormones FT4 and FT3, the latter now confirmed to be the biologically active thyroid hormone. As evident from the data in Table 5, deviations in thyroid hormone levels in the 49 affected children of the sample group fall into five distinct categories, which are discussed below. It is also evident that even in some of the children in the two control groups consuming "safe" water (<1.0 ppm F–), fluoride levels in their blood and urine are above current upper limits, indicating other sources of fluoride ingestion, such as from foods and beverages, dental products, drugs, air, or salt. In those children disturbances in thyroid hormone ratios are observed as well. . . . Some of the conclusions and recommendations we draw from this study are:
• Children with dental fluorosis living in endemic fluorosis areas and IDD (iodine deficiency disorder) may have thyroid derangements that require special care and attention.
• The primary cause of IDD may not always be iodine deficiency, but it might be induced by fluoride poisoning.
• Testing of drinking water and body fluids for fluoride content, along with FT3, FT4, and TSH—even in children without dental fluorosis—is desirable for recognizing thyroid derangements.
• Prevention and control of fluorosis and IDD require an integrated approach for diagnosis and patient management, contrary to prevailing practices.
• The results of this study question the validity of the fluoridation of drinking water, milk, fruit juices, and salt by public authorities."

Social (2005)[23] "In the current investigation 46.9% of the children in the [high fluoride] group have elevated TSH and normal FT4 and FT3 levels, while a similar derangement is also observed in 18.2% of the children in [the lower fluoride group]. This is our first category and is usually the first indication of thyroid dysfunction, termed sub-clinical hypothyroidism."

Cigar (2005)[24] "In this study, the serum levels of thyroxine (T4), triiodothyronine (T3), and protein-bound iodine (PBI) in the control cows were in the normal range of

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[22] AK Susheela, M Bhatnagar, K.Vig, NK Mondald, EXCESS FLUORIDE INGESTION AND THYROID HORMONE DERANGEMENTS IN CHILDREN LIVING IN DELHI, INDIA. Fluoride 2005;38(2):151–161 Research report 151

[23] Susheela AK, et al. (2005). Excess fluoride ingestion and thyroid hormone derangements in children living in New Delhi, India. Fluoride 38(2):98-108.

[24] Cinar A, Selcuk M. (2005). Effects of chronic fluorosis on thyroxine, triiodothyronine, and protein-bound iodine in cows. Fluoride 38(1):65-68.

healthy cows, but they were significantly lower ($p<0.05$) in the fluorotic cows. These findings are consistent with the results of research with sheep, calves, cattle, and rats. . . . On the other hand, Choubisa reported that none of a group of fluorotic domestic animals exhibited any apparent evidence of hypothyroidism, stunted growth, [or] low milk production . . . . In our view, the reason for decreased levels of T4, T3, and PBI in our cows with chronic fluorosis might be due to: 1) inhibition of the absorption of the iodine and some amino acids (e.g., tyrosine) in the gastrointestinal tract, 2) insufficient synthesis and secretion of thyroglobulin and oxidized iodides from the thyroid glands, 3) low levels of bioavailable iodine in the Tendurek Mountain region."

"Wang (2001)[25] In conclusion, high iodine and high fluorine in the drinking water have, to some extent, effects on children's intelligence and thyroid function."[26]Wang (2001) "TSH value was obviously higher than the control point, indicating that, under high iodine and high fluorine condition, T3 and T4 secreted by the thyroid are in the normal range, while TSH value secreted by the pituitary clearly increased. This is probably because high iodine and high fluorine suppress the synthesis and secretion of the thyroid peroxidase and thyroid hormones . . . . The body accelerates the Hypothalamic TSH secretion by negative feedback regulation, thus increasing the secretion of TSH, stimulating the composition of T3 and T4 of the thyroid. As a result, the TSH in the peripheral blood circulation is high while T3 and T4 are not clearly reduced."

Liu (2001) "Objective: To investigate the effects of fluoride on thyroid structure in chicks.. . . Conclusions Fluoride can seriously damage thyroid structure . During the earlier stage, fluoride can induce thyroid atrophia, however, during the later stage, it can induce thyroid enlargement which is nodular and colloid goiter."[27]

Wan (1999)[28] [Objective: To study the significant test of diagnosing endemic fluorosis. Methods Twenty one routine and biochemical marks of blood and urine from 600 cases of the patients with different degree endemic fluorosis were determined and analysed. Results . . . The average of T3 and T4 were lower than the reference value, particularly in those with moderate and severe stages of the disease. Conclusions The RBC, Hb, serum calcium,phosphorus, AKP, urinary calcium, globulin, T3 and T4 were signifiant diagnostic indicators of endemic fluorosis.]

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[25] Wang X, et al. (2001). Effects of high iodine and high fluorine on children's intelligence and thyroid function. *Chinese Journal of Endemiology* 20(4):288-90.

[26] Wang X, et al. (2001). Effects of high iodine and high fluorine on children's intelligence and thyroid function. Chinese Journal of Endemiology 20(4):288-90.

[27] Liu GY, et al. (2001). Effects of fluoride on thyroid structure in chicks. Chinese Journal of Endemiology.

[28] Wan G, et al. (2001). Determination and analysis on multimark of test of patients with endemic fluorosis. Chinese Journal of Endemiology 20(2):137-39.

Xiaoli (1999)[29] [In a group of 8-12 year old children living in an endemic fluorosis area in China, TSH levels were significantly elevated, while T4 levels were significantly decreased and T3 levels significantly increased.]

Yao (1996)[30] "The TSH level is a sensitive index which both reflects the state of the body's thyroid function, and screens the level of iodine (lack thereof) in a population. TSH is also a sensitive indicator in terms of making timely discoveries of people suffering from poor thyroid function or below-average intelligence. The results from this test show that TSH values of children with dental fluorosis from the two endemic areas is at a remarkably higher level than those from the non-endemic area. Children from the endemic areas were also found to have a lower level of intelligence than the non-endemic group. The heavier the level/concentration of fluoride found in the region, the more significant the difference in the results."

Mikhail's (1996)[31] "Conclusions: 1. Abnormalities in the thyroid function characterized by a decreased iodine absorption function of the thyroid, a low level T3 syndrome, and a slight increase of the TSH level are observed in cases of chronic fluorine intoxication in the industrial workers. 2. The observed changes progressed with the increase of the time of exposure to fluorides and a more advanced disease stage. 3. The highest frequency of occurrence of the low level T3 syndrome was observed in workers with chronic fluoride intoxication including TPP (toxic liver damage). 4. The lowered iodine absorption function of the thyroid and/or the low level T3 syndrome can serve as diagnostic signs of chronic fluorine intoxication. 5. The decrease in the T3 level most probably occurs due to the disrupted conversion of T4 to T3 at the cell- target level. The disruption of conversion may be caused by fluorine affecting the enzyme system of deiodination as well as the toxic liver damage it causes."

Shufen (1996)[32] "The levels of serum T3, T4 and TSH were analyzed in children with fluoride-aluminum combined toxicosis in the Shuicheng area of Guizhou as compared with the children without fluoride-aluminum combined toxicosis. The results showed that serum T4 content decreased in the children with fluoride aluminum combined toxicosis (103.9±15.9 nmol/L vs 150.67±16.5 nmol/L, p 0 01), but no obvious differences of serum T3 and TSH were found among total three groups. It suggests that the disorder of the thyroid function should be considered when treating the children with fluoride aluminum combined toxicosis."

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[29] Xiaoli L, et al. (1999). The detection of children's T3, T4 and TSH contents in endemic fluorosis areas. Endemic Disease Bulletin 14(1):16-17.

[30] Yao Y, et. al. (1996). Analysis on TSH and intelligence level of children with dental Fluorosis in a high fluoride area. Literature and Information on Preventive Medicine 2(1):26-27

[31] Mikhailets ND, et al. (1996). Functional state of thyroid under extended exposure to fluorides. Probl Endokrinol 42:6-9.

[32] Shufen J, et al. (1996). The change of thyroid functlon from children with fluoride aluminum combined toxicosis in Shuicheng area of Guizhou. Journal of Guiyang Medical College.

Michael (1996)[33] "While levels of thyroid stimulating hormone (TSH) and triiodothyronine (T3) did not vary, a significant increase in the thyroxine (T4) levels suggested alteration in thyroid function."

Yang (1994)[34] "An excess of fluoride and a lack of iodine in the same environment has been shown to have a marked effect on child intellectual development, causing a more significant intellectual deficit than lack of iodine alone. In our study the study group of children from the high fluoride-high iodine village area had an average IQ of 76.67±7.75, which was somewhat lower than the control (IQ 81.67 ±11.9), although the difference is not statistically significant (P > 0.05). However, as seen in Table 2, the percentage of children in the low range (16.67%) is higher in the endemic group than in the control group (10.0%), suggesting that a high iodine-high fluoride environment also has a definite negative influence on child intellectual ability."

Xu (1994)[35] "The number of children whose level of intelligence is lower is significantly increased in regions of high fluoride/iodine, regions of high fluoride only, regions of high fluoride/low iodine, against their respective comparative groups."

Lin (1991)[36] "Area A (high fluoride, low iodine) differed from area B (normal fluoride, low iodine) by having lower mean IQ, higher TSH, slightly higher 1311 uptake, and higher urinary iodine. . . . The significant ditferences in IQ among these regions suggests that fluoride can exacerbate central nervous lesions and somatic developmental disturbance caused by iodine deficiency. . . . [W]e found that 69% of the children with mental retardation had elevated TSH levels. IQ and TSH were negatively correlated. Many investigators regard an elevated TSH in the presence of normal T4 and T3 levels as evidence for hypothyroidism that is subclinical but that can still affect the development of brain and cerebral function to some degree."

Liu (1988)[37] "Endemic fluorosis is a systemic disease. We investigated the serum free fluoride, thyroid hormones and TSH concentrations in 37 cases. Significantly lowered serum T4 . . . and increased TSH were found in patients. Patients'serum T3 concentrations were not significantly different from the controls. Significant negative correlations were found between serum free fluoride concentrations and T3 concentrations or T3/T4 ratios. We propose that fluoride intoxication might decrease thyroid function and suggest the method to prevent and treat this condition."

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[33] Michael M, et al. (1996). Investigations of soft tissue function in fluorotic individuals of North Gujurat. Fluoride 29(2):63-71.

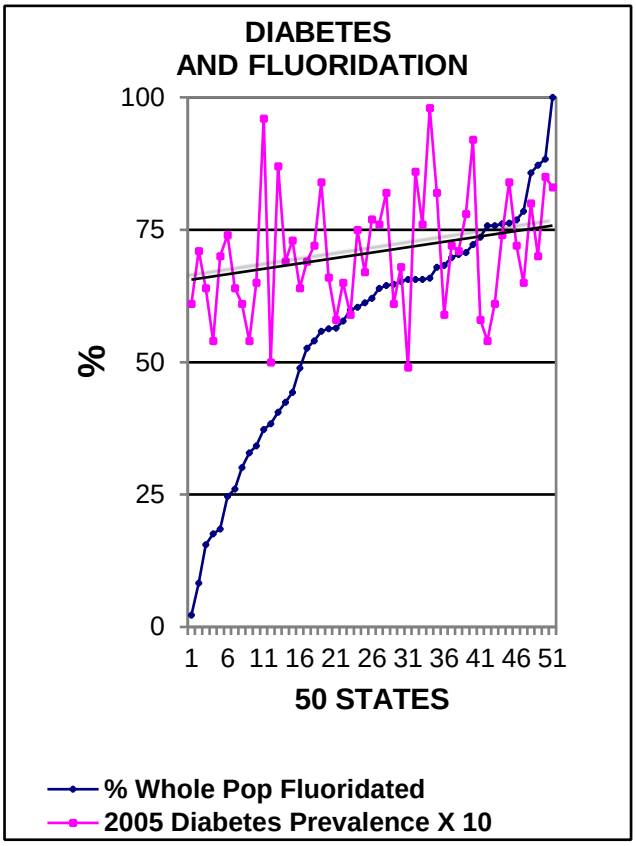
[34] Yang Y, et al. (1994). The effects of high levels of fluoride and iodine on intellectual ability and the metabolism of fluoride and iodine. Chinese Journal of Epidemiology 15(4):296-98 (republished in Fluoride 2008; 41:336-339).

[35] Xu Y, et al. (1994). The effect of fluorine on the level of intelligence in children. *Endemic Disease Bulletin* 9(2):83-84.

[36] Lin F; et al (1991). The relationship of a low-iodine and high-fluoride environment to subclinical cretinism in Xinjiang. *Endemic Disease Bulletin* 6(2):62-67 (republished in *Iodine Deficiency Disorder Newsletter* Vol. 7(3):24-25).

[37] Liu Z, et al. (1988). An investigation on the serum thyroid hormones and fluoride concentrations in patients with endemic fluorosis. Chinese Journal of Endemiology 7(4):216-18. [Article in Chinese with English summary]

Bachinskii (1985)[38] "The ingestion of drinking water with high concentrations of fluoride (122 +/- 5 micromoles per liter) leads, in healthy people, to stress of the functional status of the pituitary-thyroid system, as evidenced by a reduction in the concentration of T3, an increase in the production (by the hypothalamus) of TSH in the serum, and a more avid uptake of I131 by the thyroid tissue. This permits us to classify the excessive accumulation of fluorine in the body as a risk factor providing a basis for the development of thyroid dysfunction."



Yu (1985)[39] "A study on the serum T4, T3 and TSH levels was performed in 27 patients with chronic skeletal fluorosis and the data obtained were compared with those of 20 health persons. The results showed that serum T4 in the patients was lower than in the controls and TSH was higher, while serum T3 showed no significant difference. There was no goiter found in the patients. These data indicate that fluorine may reduce serum T4 by interfering [with] thyroid function.The increase of TSH secretion is the consequence stimulated by a feedback mechanism but no proliferation and enlargement of the thyroid gland resulted . . . ."

Graphing the 50 US states ranked on the percentage of the whole population fluoridated and plotting their respective rate of diabetes (X10)[40] provides this graph, perhaps a 10% increase in diabetes. Remember, fluoridated water represents only about half of fluoride exposure.

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[38] Bachinskii PP et al. 1985. Action of the body fluorine of healthy persons and thyroidopathy patients on the function of hypophyseal-thyroid the system. Probl Endokrinol (Mosk) 31(6):25-9. [Article in Russian, translated into English]

[39] Yu Y. (1985). Study on serum T4, T3, and TSH levels in patients with chronic skeletal fluorosis. Chinese Journal of Endemiology 4(3):242-43.

[40] Note: In order to view the data on one graph, the percentage of fluoridated in each state is correct but the percentage of diabetes is increased by 10 fold. In other words, 75 is actually 7.5% for diabetes and 75% for fluoridation. Source of data: http://apps.nccd.cdc.gov/nohss/FluoridationV.asp
http://www.unitedhealthfoundation.com/shr2005/components/obesity.html
http://pubs.usgs.gov/circ/2004/circ1268/htdocs/table05.html

Treatment

ANIMAL TREATMENT Sarkar (2014) Resveratrol (3,4,5-trihydroxystilbene), a polyphenol and well-known natural antioxidant has been evaluated for its protective effect against fluoride-induced metabolic dysfunctions in rat thyroid gland. . .Resveratrol supplementation in fluoride-exposed animals appreciably prevented metabolic toxicity caused by fluoride and restored both functional status and ultra-structural organization of the thyroid gland towards normalcy. This study first establishes the therapeutic efficacy of resveratrol as a natural antioxidant in thyroprotection against toxic insult caused by fluoride."[41]

B. PARATHYROID GLAND

Wang (2015)[42] "Parathyroid hormone (PTH), PTH-related peptide (PTHrP), and calcium-sensing receptor (CaSR) play important roles in maintaining calcium homeostasis. Here, we study the effect of fluoride on expression of PTH, PTHrP, and CaSR both in vitro and in vivo. MC3T3-E1 cells and Sprague-Dawley rats were treated with different concentrations of fluoride. Then, the free calcium ion concentration in cell culture supernatant and serum were measured by biochemical analyzer. The expression of PTH, PTHrP, and CaSR was analyzed by qRT-PCR and Western blot. We found that the low dose of fluoride increased ionized calcium (i[Ca(2+)]) and the high dose of fluoride decreased i[Ca(2+)] in cell culture supernatant. The low dose of fluoride inhibited the PTH and PTHrP expression in MC3T3-E1 cells. The high dose of fluoride improved the PTHrP expression in MC3T3-E1 cells. Interestingly, we found that NaF decreased serum i[Ca(2+)] in rats.

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[41] Sarkar C[1], Pal S. Ameliorative effect of resveratrol against fluoride-induced alteration of thyroid function in male wistar rats. Biol Trace Elem Res. 2014 Dec;162(1-3):278-87. doi: 10.1007/s12011-014-0108-3. Epub 2014 Aug 28.

[42]Wang Y1, Duan XQ, Zhao ZT, Zhang XY, Wang H, Liu DW, Li GS, Jing L., Fluoride Affects Calcium Homeostasis by Regulating Parathyroid Hormone, PTH-Related Peptide, and Calcium-Sensing Receptor Expression. Biol Trace Elem Res. 2015 Jun;165(2):159-66. doi: 10.1007/s12011-015-0245-3. Epub 2015 Feb 3.

Fluoride increased CaSR expression at both messenger RNA (mRNA) and protein levels in MC3T3-E1 cells and rats. The expression of PTHrP protein was inhibited by fluoride in rats fed regular diet and was increased by fluoride in rats fed low-calcium diet. Fluoride also increased the expression of PTH, NF-kappaB ligand (RANKL), and osteoprotegerin (OPG) in rats. The ratio of RANKL/OPG in rats fed low-calcium food in presence or absence of fluoride was significantly increased. These results indicated that fluoride might be able to affect calcium homeostasis by regulating PTH, PTHrP, and CaSR."

Shashi (2013)[43] **Abstract:** The present study assessed the effect of fluoride on parathyroid function in 860 patients (mean age 32.50±10.50) affected with skeletal fluorosis, selected randomly from endemic fluorotic areas of district Bathinda, Punjab, India. The fluoride content in water sources was found to vary from 0.68-15.78 mg/L in study areas. Hence, the study areas were categorized as five different groups Control (0.68- 1.00 mg/L), A-I (1.01-4.00 mg/L), A-II (4.01-8.00 mg/L), A-III (8.01-12.00 mg/L) and A-IV (12.01-16.00 mg/L). An age and sex matched group of 140 control subjects without skeletal fluorosis were also included. The functional activity of the parathyroid was measured by radio immuno assay of parathyroid hormone (PTH). The biochemical estimations were made for serum and urinary fluoride, serum calcium, phosphorus, calcitonin and alkaline phosphatase (ALKP). The results revealed that level of serum and urinary fluoride was significantly ($p<0.001$) higher in fluorotic patients in comparison to control. The serum PTH, calcitonin and activity of ALKP was significantly ($P<0.001$) elevated in fluorotic patients. Significant ($P<0.05$) hypocalcaemia was observed in study group A-I and A-II and elevation in group A-IV. However, the alterations in calcium level in group A-III was statistically non significant. Hyperphosphatemia ($P<0.001$) was also observed in patients of fluorosis. Pearson's bivariate correlation showed positive correlation between water F vs serum F ($r= 0.98$, $P<0.001$), serum F vs PTH ($r= 0.97$, $P<0.007$), serum F vs calcitonin ($r=0.80$, $P<0.01$) and serum F vs ALKP ($r=0.93$, $P<0.02$). Negative correlation was noted between serum and urinary concentration of fluoride. When the serum fluoride concentration was increased the corresponding urinary fluoride excretion declined along with the advancing age. It may be concluded that high fluoride ingestion has a definite relation with increased calcitonin concentration, which may be the major cause of hypocalcemia in fluorotic patients, which may further leads to the increased parathyroid function i.e raised PTH levels in the serum to maintain serum calcium levels and may have a role in toxic manifestations of clinical and skeletal fluorosis."

Puranik (2013)[44] "Objective: This study investigated fluoride's effects on iPTH secretion

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[43] *A Shashi and Swati Singla.* **Parathyroid Function in Osteofluorosis,** World Journal of Medical Sciences 8 (1): 67-73, 2013 ISSN 1817-3055 © IDOSI Publications, 2013**,** DOI: 10.5829/idosi.wjms.2013.8.1.72168 http://www.idosi.org/wjms/8(1)13/11.pdf

[44] *Puranik, Chaitanya Prakash,* Ph.D., **Effect of Fluoride on Parathyroid Hormone Secretion,** Dissertation. **THE UNIVERSITY OF NORTH CAROLINA AT CHAPEL HILL**, 2013, 129 pages; 3606754

and its underlying mechanism. . . . Conclusion: Fluoride modulates iPTH secretion in vitro and in vivo. However, Fluoride's action on the parathyroid gland is not mediated through CASR. While fluoride's effects, in vitro, were equivalent between the two mouse strains, early strain-dependent effect on iPTH secretion was observed in vivo. Difference in fluoride-mediated gene expression in C3H and B6 suggests an underlying difference in physiologic handling of fluoride by the two strains."

Peng (2013)[45] "Chronic exposure to combined fluoride and arsenic continues to be a major public health problem worldwide, affecting thousands of people. In recent years, more and more researchers began to focus on the interaction between the fluorine and the arsenic. In this study, the selected investigation site was located in China. The study group was selected from people living in fluoride-arsenic polluted areas due to burning coal. The total number of participants was 196; including the fluoride-arsenic anomaly group (130) and the fluoride-arsenic normal group (63). By observing the changes in gene and protein expression of PTH/PKA/AP1 signaling pathway, the results show that fluoride can increase the expression levels of PTH, PKA, and AP1, but arsenic can only affect the expression of AP1; fluoride and arsenic have an interaction on the expression of AP1. Further study found that fluoride and arsenic can affect the mRNA expression level of c-fos gene (AP1 family members), and have an interaction on the expression of c-fos, but not c-jun. The results indicate that PTH/PKA/AP1 signaling pathway may play an important role in bone toxicity of fluoride. Arsenic can affect the expression of c-fos, thereby affecting the expression of transcription factor AP1, indirectly involved in fluoride-induced bone toxicity."

Gutowska (2013)[46] "Chronic long-term exposure to high levels of fluoride leads to fluorosis, manifested by skeletal fluorosis and damage to internal organs, including kidneys, liver, parathyroid glands, and brain. Excess fluoride can also cause DNA damage, trigger apoptosis, and change cell cycle. The effect of fluoride may be exacerbated by lead (Pb), a potent inhibitor of many enzymes and a factor causing apoptosis, still present in the environment in excessive amounts. Therefore, in this study, we investigated the effects of sodium fluoride (NaF) and/or lead acetate (PbAc) on development of apoptosis, cell vitality, and proliferation in the liver cell line HepG2. We examined hepatocytes from the liver cell line HepG2, incubated for 48 h with NaF, PbAc, and their mixture (NaF + PbAc), and used for measuring apoptosis, index of proliferation, and vitality of cells. Incubation of the hepatocytes with NaF or PbAc increased apoptosis, more when fluoride and Pb were used simultaneously. Vitality of the cells depended on the compound used and its concentration. Proliferation slightly increased and then decreased in a high fluoride environment; it

[45]Zeng QB1, Xu YY1, Yu X2, Yang J2, Hong F3, Zhang AH1. Arsenic may be involved in fluoride-induced bone toxicity through PTH/PKA/AP1 signaling pathway. Environ Toxicol Pharmacol. 2014 Jan;37(1):228-33. doi: 10.1016/j.etap.2013.11.027. Epub 2013 Dec 7.

[46] Gutowska I1, Baranowska-Bosiacka I, Siwiec E, Szczuko M, Kolasa A, Kondarewicz A, Rybicka M, Dunaj-Stanczyk M, Wiernicki I, Chlubek D, Stachowska E. Lead enhances fluoride influence on apoptosis processes in liver cell line HepG2. Toxicol Ind Health. 2013 Nov 5. [Epub ahead of print]

decreased significantly after addition of Pb in a dose-dependent manner. When used together, fluoride inhibited the decreasing effect of Pb on cell proliferation."

Wen (2012)[47] The aim of this study was to explore the association of parathyroid hormone (PTH) gene Bst BI polymorphism, calciotropic hormone levels, and dental fluorosis of children. A case-control study was conducted in two counties (Kaifeng and Tongxu) in Henan Province, China in 2005-2006. Two hundred and twenty-five children were recruited and divided into three groups including dental fluorosis group (DFG), non-dental fluorosis group (NDFG) from high fluoride areas, and control group (CG). Urine fluoride content was determined using fluoride ion selective electrode; PTH Bst BI were genotyped using PCR-RFLP; osteocalcin (OC) and calcitonin (CT) levels in serum were detected using radioimmunoassay. Genotype distributions were BB 85.3% (58/68), Bb 14.7% (10/68) for DFG; BB 77.6% (52/67), Bb 22.4% (15/67) for NDFG; and BB 73.3% (66/90), Bb 27.7% (24/90) for CG. No significant difference of Bst BI genotypes was observed among three groups ($P > 0.05$). Serum OC and urine fluoride of children were both significantly higher in DFG and NDFG than in CG ($P < 0.05$, respectively), while a similar situation was not observed between DFG and NDFG in high fluoride areas ($P > 0.05$). Serum OC level of children with BB genotype was significantly higher compared to those with Bb genotype in high fluoride areas ($P < 0.05$). However, no significant difference of serum CT or calcium (Ca) was observed. In conclusion, there is no correlation between dental fluorosis and PTH Bst BI polymorphism. Serum OC might be a more sensitive biomarker for detecting early stages of dental fluorosis, and further studies are needed.

The parathyroid gland produces parathyroid hormone (PTH). PTH regulates the amount of calcium in our bones and blood supply. When the calcium level in blood starts to fall, PTH triggers the breakdown of bone tissue as a means of transferring the body's stored supply of calcium into the blood supply. When the parathyroid produces too much PTH a condition known as hyperparathyroidism develops. Hyperparathyroidism has been found to occur as a secondary effect of the fluoride-induced bone disease skeletal fluorosis, and may help to explain some of the bone effects encountered in fluorosis.

When calcium is removed from the bones (osteoclastic activity) the fluoride in the bones increases blood fluoride concentrations.

Gupta et al. (2001)[48] and Suketa (2002) show again that in cases of fluorosis there is hyperparathyroidism, as seen in elevated parathyroid hormone (PTH) levels.

Acevedo (1996)[49] Chardin (1998)[50] When thyroid and parathyroid glands are removed in subjects, same mineral effects occur as can be observed in dental fluorosis patients.

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[47] Wen S1, Li A, Cui L, Huang Q, Chen H, Guo X, Luo Y, Hao Q, Hou J, Ba Y., The relationship of PTH Bst BI polymorphism, calciotropic hormone levels, and dental fluorosis of children in China., Biol Trace Elem Res. 2012 Jun;147(1-3):84-90. doi: 10.1007/s12011-011-9313-5. Epub 2012 Jan 5.

[48] Gupta SK, Khan TI, Gupta RC, Gupta AB, Gupta KC, Jain P, Gupta A - "Compensatory hyperparathyroidism following high fluoride ingestion - a clinico - biochemical correlation" Indian Pediatr 38(2):139-46 (2001)

Stamp (1990)[51]

> "1. To determine the relationships between parathyroid hormone activity and long-term sodium fluoride therapy in osteoporosis . . . .
> 2. Cross-sectional data showed a fourfold mean increase in biologically active parathyroid hormone on fluoride treatment . . . .
> 3. Fluoride-treated patients were then analysed in two groups according to the level of biologically active parathyroid hormone. . . .
> 4. Results show that long-term fluoride and calcium therapy increase biologically active parathyroid hormone in osteoporosis and that excessive parathyroid hormone activity may account for certain features of the refractory state."

Chen (1988)[52] "Fluoride ion (F-) alone or in conjunction with aluminum (Al3+) has been shown to stimulate the activity of guanine nucleotide-binding proteins (G proteins) in cell membrane preparations from a variety of cell types and in intact hepatic cells. Several studies have indicated that G proteins are involved in the regulation of parathyroid hormone (PTH) secretion. Intracellular second messengers which modulate PTH secretion (e.g., cAMP) have also been found to be regulated by G proteins. We have, therefore, employed F- as a probe to investigate the possible role of G proteins in the modulation of PTH release and the intracellular second messengers that have been implicated in the control of PTH secretion. F- produces a dose-dependent inhibition of PTH release with a maximal inhibitory effect (67%) at 5 mM. F- exerts its inhibitory effect within 5 min and the degree of suppression of PTH secretion gradually increases over 1 hr. F- (5 mM) inhibits PTH secretion at 0.5 mM Ca2+ to the level observed with 2 mM Ca2+ alone; moreover, the effects of F- and high Ca2+ are not additive. . . . We conclude that F- is a potent inhibitor of PTH secretion."

Mertz (1987)[53] "Fluorine is known to bind calcium in the body, causing ionic calcium to decrease; this, in turn, causes secondary hyperparathyroidism."

However, more recent investigations have revealed that a new mechanism of action: hyperparathyroidism is caused by chronically elevated TSH levels. (Fluoride is **the** TSH clone]. Elevated TSH levels are usually seen in hypothyroidism, and therefore explain

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[49] Acevedo AC, Chardin H, Staub JF, Septier D, Goldberg M - "Morphological study of amelogenesis in the rat lower incisor after thyro-parathyroidectomy, parathyroidectomy and thyroidectomy." Cell Tissue Res 283(1):151-7 (1996)

[50] Chardin H, Acevedo AC, Risnes S - "Scanning electron microscopy and energy-dispersive X-ray analysis of defects in mature rat incisor enamel after thyroparathyroidectomy." Arch Oral Biol 43(4):317-27 (1998)

[51] Stamp TC1, Saphier PW, Loveridge N, Kelsey CR, Goldstein AJ, Katakity M, Jenkins MV, Rose GA. Fluoride therapy and parathyroid hormone activity in osteoporosis. Clin Sci (Lond). 1990 Sep;79(3):233-8.

[52] Chen CJ1, Anast CS, Brown EM. Effects of fluoride on parathyroid hormone secretion and intracellular second messengers in bovine parathyroid cells. J Bone Miner Res. 1988 Jun;3(3):279-88.

[53] [Trace Elements in Human and Animal Nutrition - Fifth Edition, Edited by Walter Mertz, U. S. Dept. of Agriculture, Agricultural Research Service, Beltsville Human Nutrition Research Center, Beltsville, Maryland, p. 375 (1987)

why hyperparathyroidism is so closely associated with hypothyroidism (Paloyan et al,1997).[54]

Hyperparathyroidism is ten times more frequent in thyroid patients than expected in a general medical population and is especially prevalent in patients with **goiter** (Stoffer, 1982).

Roy (1962) “These experiments may be interpreted to show that the effect of NaF is to reduce the solubility of the apatite complex and thus to lower the basic level of equilibrium of calcium between fluid and solid phases. To compensate for this decreased level, the glands of the intact animals are required to increase secretion with an ultimate increase in osteoclast proliferation.”[55]

[54] Paloyan Walker R, Kazuko E, Gopalsami C, Bassali J, Lawrence AM, Paloyan E - "Hyperparathyroidism associated with a chronic hypothyroid state" Laryngoscope 107(7):903-9 (1997)

[55] Roy V. Talmage, S.B. Doty The effect of sodium fluoride on parathyroid function in the rat as studied by peritoneal lavage General and Comparative Endocrinology Volume 2, Issue 5, October 1962, Pages 473–479

C. PANCREAS:

The pancreas produces a hormone called insulin which regulates the uptake of glucose from the bloodstream. Fluoride increases the levels of glucose in the blood. Vinals provides a review and background of the mechanism which fluoride acts on the insulin receptors and is moved to the top of the list of studies to provide a foundation.

Vinals (1993)[56] "Fluoride is a nucleophilic reagent which has been reported to inhibit a variety of different enzymes such as esterases, asymmetrical hydrolases and phosphatases. In this report, we demonstrate that fluoride inhibits tyrosine kinase activity of insulin receptors partially purified from rat skeletal muscle and human placenta. Fluoride inhibited in a similar dose-dependent manner both β-subunit autophosphorylation and tyrosine kinase activity for exogenous substrates. This inhibitory effect of fluoride was not due to the formation of complexes with aluminium and took place in the absence of modifications of insulin-binding properties of the insulin receptor. Fluoride did not compete with the binding site for ATP or Mn2+. Fluoride also inhibited the autophosphorylation and tyrosinekinase activity of receptors for insulin-like growth factor I from human placenta. Addition of fluoride to the pre-phosphorylated insulin receptor produced a slow (time range of minutes) inhibition of receptor kinase activity. Furthermore, fluoride inhibited tyrosine kinase activity in the absence of changes in the phosphorylation of pre-phosphorylated insulin receptors, and the sensitivity to fluoride was similar to the sensitivity of the unphosphorylated insulin receptor. The effect of fluoride on tyrosine kinase activity was markedly decreased when insulin receptors were pre-incubated with the copolymer of glutamate/tyrosine. Prior exposure of receptors to free tyrosine or phosphotyrosine also prevented inhibitory effect of fluoride. However, the protective effect of erosion or phosphotyrosine was maximal at low concentrations, suggesting the interaction of these compounds with the receptor itself rather than with fluoride. These data suggest: (i) that fluoride interacts directly and slowly with the insulin receptor, which causes inhibition of its phosphotransferase activity; (ii) that the binding site of fluoride is not structurally modified by receptor phosphorylation; and (iii) based on the fact that fluoride inhibits phosphotransferase activity in the absence of alterations in the binding of ATP, Mn2+ or insulin, we speculate that fluoride binding might affect the transfer of phosphate from ATP to the tyrosine residues of the β-subunite of the insulin receptor and to the tyrosine residues of exogenous substrates.
"The insulin receptor is a disulphide-linked herotetrameric membrane glycoprotein consisting of two alph (M 135000) and two transmembrane beta (M 95000) subunits (Massague et al., 1981); Massage and Czech, 1982; Ullrich et al, 1985; Ebina et al., 1985). The alpha subunits are entirely extracellular and participate in insulin binding, whereas the beta-subunits contain extracellular , transmembrane and intracellular domains. . . . The tyrosine kinase activity of the insulin receptor appears to be essential for certain cellular responses to insulin. Thus anti-insulin-receptor antibodies, which inhibit the kinase activity of the insulin receptors, also block the ability of cells to

[56] VINALS F, TESTAR X, PALACIN M and ZORZANO A. Inhibitory effect of fluoride on insulin receptor autophosphorylation and tyrosinekinase activity, "Biochem.J.(1993)291,615-622(PrintedinGreatBritain) 615

respond to insulin (Morgan et al., 1986; Morgan and Roth, 1987). In addition, the microinjection of insulin receptors in *Xenopus* oocytes causes an increase in the phosphorylation of ribosomal S6 subunit, which is further increased by prior receptor activation, due to insulin-receptor autophosphorylation (Maller et al., 1986). Studies with receptors mutated at the ATP-binding site (Chou et al., 1987; Ebina et al., 1987; McClain et al., 1987) or at tyrosine residues 1162 and 1163 (Ellis et al., 1986; Decant et al., 1988) have also led to the conclusion that that tyrosine phosphotransferase function of the insulin receptor is an absolute requirement for the hormone to activate the receptor signaling function in cells.

"Based on the pivotal role of insulin-receptor kinase activity on insulin action, the catalytic properties of the insulin-receptor kinase require thorough characterization. In studies initially designed to investigate the interaction between regulatory G-proteins and insulin receptors, we substantiated a potent inhibitory effect of fluoride on insulin-receptor kinase activity. On the basis of this finding and the fact that the use of fluoride , a potent nucleophilic reagent (Edwards and Pearson, 1962), has yielded useful information on the kinetics of a variety of enzymes (Layne and Najjar, 1975; Bunick and Kashket, 1982; Nilsson and Branden, 1982), we have characterized the inhibitory effect of fluoride on insulin-receptor autophosphorylation and receptor kinase for exogenous substrates."

(A few references primarily in author alphabetical order are provided here. I have not read each article and only a few quotes which were handy, are included here.)

Adebayo 2012[57] "We conclude that fluoride exerts biochemical effect on **lipid peroxidation** and antioxidant enzymes of both PU and well-fed rats. This effect varied widely between the liver and the pancreas but it seems that the liver is more sensitive to the toxic assault of fluoride than the pancreas especially in PU rats."

Agalakova (2012)[58] "The molecular mechanisms underlying fluoride toxicity are different by nature. Fluoride is able to stimulate G-proteins with subsequent activation of downstream signal transduction pathways such as PKA-, PKC-, PI3-kinase-, Ca2+-, and MAPK-dependent systems. G-protein-independent routes include tyrosine phosphorylation and protein phosphatase inhibition. Along with other toxic effects, fluoride was shown to induce oxidative stress leading to excessive generation of ROS, lipid peroxidation, decrease in the GSH/GSSH ratio, and alterations in activities of antioxidant enzymes, as well as to inhibit glycolysis thus causing the depletion of cellular ATP and disturbances in cellular metabolism. Fluoride triggers the disruption of mitochondria outer membrane and release of cytochrome c into cytosol, what activates caspases-9 and -3 (intrinsic) apoptotic

[57] Olusegun Lateef Adebayo and Gbenga Adebola Adenuga, 2012. Biochemical Changes in the Liver and the Pancreas of Well-fed and Protein Undernourished Rats Following Fluoride Administration. *Asian Journal of Applied Sciences, 5: 215-223.*

[58] Natalia Ivanovna Agalakova and Gennadii Petrovich Gusev. Molecular Mechanisms of Cytotoxicity and Apoptosis Induced by Inorganic Fluoride, ISRN Cell Biology, Volume 2012 (2012), Article ID 403835, 16 pages http://dx.doi.org/10.5402/2012/403835

pathway. Extrinsic (death receptor) Fas/FasL-caspase-8 and -3 pathway was also described to be implicated in fluoride-induced apoptosis. Fluoride decreases the ratio of antiapoptotic/proapoptotic Bcl-2 family proteins and upregulates the expression of p53 protein. Finally, fluoride changes the expression profile of apoptosis-related genes and causes endoplasmic reticulum stress leading to inhibition of protein synthesis.

Banu P et al. Toxicity of fluoride to diabetic rats. Fluoride 1997 30(1) 43-50.

Birkner E, et al. Influence of sodium fluoride and caffeine on the concentration of fluoride ions, glucose, and urea in blood serum and activity of protein metabolism enzymes in rat liver. Bill Trace Elem Res. 2006 112(2) 169-74.

Boros I et al. Fluoride intake, distribution, and bone content in diabetic rats consuming fluoridated drinking water. Fluoride 1998 31(1) 33-42.

Bolgul BS et al. Evaluation of caries risk factors and effects of fluoride-releasing adhesive material in children with insulin-dependent diabetes mellitus (IDDM): Initial first-year results. Act Odontological Scandinavia, 2004 62(5) 289-292.

Chehoud KA, Chiba FY, Sassaki Kt, et al. Effects of fluoride intake on insulin sensitivity and insulin signal transduction. Fluoride. October-December 2008 41(4) 270-275.

Chiba FY, Garbin CAS, Sumida DH. Effect of fluoride intake on carbohydrate metabolism, glucose tolerance, and insulin signaling. Fluoride July-September 2012 45(3 Pt 2) 239-241.

Chiba FY, Colombo NH, Shirakashi DJ, Gomes WD, Moimaz SAS, Garbin CAS, Silva CA, Sumida DH. Insulin signal decrease in muscle but not in the liver of castrated male rats from chronic exposure to fluoride. Fluoride January-March 2010. 43(1)25-30.

Chlubek D et al. Activity of pancreatic anti oxidative enzymes and malondialdehyde concentrations in rats with hyperglycemia caused by fluoride intoxication. J. Trace Elem. Med. Bill. 2003 17 57-60.

Chlulnek D, et al. Activity of Pancreatic antioxidative enzymes and malondialdehyde concentrations in rats with hyperglycemia caused by fluoride intoxication. Journal of trace elements in medicine and biology. 2003, vol 17(1)57-60.

Chuba FY, Columbo NH et al. NaF treatment increases TNF-a and resistin concentrations and reduces insulin signal in rats. Journal of Fluorine Chemistry 2012 136 3-7.

Eliud (2009)[59] "Chronic exposure to high fluoride (F^-) may lead to local tissue disturbances, known as fluorosis. F^- is an oxidizing agent and a well-known reversible enzymatic inhibitor that interferes with the enzyme activity of at least 80 proteins. The goals of the current study were to evaluate whether F^- exposure affected the oral glucose tolerance test (OGTT) in C57BL6 mice; and to determine the mechanisms at work in glucose homeostasis at the cellular level, in mouse pancreatic β-cells (βTC-6) exposed to F^-…. Exposure to high levels of F^- in drinking water may decrease insulin mRNA and its secretion from β-cells, and might therefore affect the OGTT."

Garcia-Montalvo EA, Reyes-Perez H, Del Razo LM. Fluoride exposure impairs glucose tolerance via decreased insulin expression and oxidative stress. Toxicology September 19 2009 263(2-3) 75-83.

Greenberg LW, Nelsen CE, Kramer N. Nephrogenic diabetes insipidus with fluorosis. Pediatrics 1974 54 320-322.

GutowaskI, Baranowska-Bosiack I et al. Changes in the concentration of fluoride in the serum and bones of female rats with strptozotocin induced diabetes. Fluoride 2009. January-March 42(1) 9-16.

Gruck-Mamczar E, et al. Activities of some enzymes and concentration of ammonia in serum of rats with fluoride hyperglycemia. Ann Acad Med Stetin. 2004 50 Suppl 1 36-41.

Hattori Y, Matsuda N, Sato A, Watanuki S, Tomioka H, Kawasaki H, Kanno M. Predominant contribution of the G protein-mediated mechanism to NaF-induced vascular contractions in diabetic rats: association with an increased level of G(qalpha) expression. J Pharmacy Exp there. 2000 292(2) 761-8.

Hu (2012)[60] "Studies on the role of insulin and insulin receptor (InsR) in the process of skeletal fluorosis, especially in osteogenic function, are rare. We evaluated the effect of increasing F^- doses on the marker of bone formation, serum insulin level and pancreatic secretion changes in vivo and mRNA expression of InsR and osteocalcin (OCN) in vitro. . . .To sum up, there existed a close relationship between insulin secretion and fluoride treatment. The insulin signal pathway might be involved in the underlying occurrence or development of skeletal fluorosis."

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[59] Eliud A. García-Montalvo, Hugo Reyes-Pérez, Luz M. Del Razo, Fluoride exposure impairs glucose tolerance via decreased insulin expression and oxidative stress. Toxicology September 2009, 263(2-3) 75-83.

[60] Hu CY1, Ren LQ, Li XN, Wu N, Li GS, Liu QY, Xu H. Effect of fluoride on insulin level of rats and insulin receptor expression in the MC3T3-E1 cells. Biol Trace Elem Res. 2012 Dec;150(1-3):297-305. doi: 10.1007/s12011-012-9482-x. Epub 2012 Aug 8.

Irmak (2014)[61] "The incidence of type 1 diabetes (T1D) has increased substantially in Finland, but the exact trigger for the onset of T1D is still unknown. We know that use of amoxicillin and anti-cariogenic fluoride tablets is a common practice for children in Finland. It seems that beta-cell destruction is initiated by modification of the proinsulin by combined effects of fluoride (F2) and amoxicillin. Amoxicillin especially when used together with clavulanic acid results in an acid environment around the beta-cells that promotes the conversion of F2 to hydrogen fluoride (HF). Unlike F2, HF can diffuse easily into the beta-cell cytosol. Because the cytosol has a neutral pH, virtually all HF reverts to F2 in the cytosol and F2 cannot easily diffuse out of the cell. Exposure to excess F2 promotes proinsulin covalent dimerization and simultaneously hyperexpression of MHC Class I molecules. Proinsulin dimers then migrate to the cell membrane with MHC class I molecules, accumulate at the beta-cell membrane and produces a powerful immunogenic stimulus for the cytotoxic T-cells. Production of cytotoxic cytokines from the infiltrating T-cells initiates the destruction of beta-cells. In Finnish children, this might be helped along by a higher beta-cell activity and by a reactive thymus-dependent immune system induced by higher levels of thyroid hormones and calcitonin respectively. After repeated similar attacks, more and more effector T-cells are raised and more and more beta-cells are destroyed, and clinical diabetes occurs."

Lima Leite A, (2014) "Administration of high doses of fluoride (F) can alter glucose homeostasis and lead to insulin resistance (IR)."

Lobo JG, Leite AL, Pereira HA, Fernandes MS, Peres-Buzalaf C, Sumida DH, Rigalli A, Buzalaf MA. Low-Level Fluoride Exposure Increases Insulin Sensitivity in Experimental Diabetes. J Dent Res. 2015 Jul;94(7):990-7. doi: 10.1177/0022034515581186. Epub 2015 Apr 10.

Lombarte, Mercedes Fina, Brenda L Lupo, Maela Buzalaf et al. Physical exercise ameliorates the toxic effect of fluoride on the insulin-glucose system. Journal of Endocrinology. 2013. 218 (1) 99-103.

Lupo M, Buzalaf MA, Rigalli A, Effect of fluoridated water on plasma insulin levels and glucose homeostasis in rats with renal deficiency. Biological Trace Element Research. 2001. 140 198-207.

Menoyo I, Puche RC Rigalli A. Fluoride-induced resistance to insulin in the rat. Fluoride 2008 41 260-269.

MenoyoI Rigalli A, Puche RC. Effect of fluoride on the secretion of insulin in the rat. Arzneimittel Forschung (Drg Res) 2005 55(5) 455-60.

[61] M. Kemal Irmak, Ilknur Senver Ozcelik, Abdullah Kaya. Fluoride toxicity and new-onset diabetes in Finland: a hypothesis. J Exp Integr Med. 2014; 4(1): 3-8
doi: 10.5455/jeim.011113.hp.007

Michaud DS. Epidemiology of pancreatic cancer. Minerva Chir, 2004 59(2)99-111.

Mohammed AHS, Ata S, Dawood EM. Influence of the different does of sodium fluoride on the rabbit exocrine pancreas. Hist-pathological study. Technical Instutitue/Kufa. www.iasj.net/iasj?func=fulltext&aId=39462

National Health and Medical Research Council (NHMRC) 2007. A systematic review of the efficacy and safety of fluoridation Part B: EXCLUDED STUDIES.

NRC (2006) page 214. "OTHER ENDOCRINE ORGANS "The effects of fluoride exposure have been examined for several other endocrine organs, including the adrenals, the pancreas, and the pituitary (for details, see Appendix E, Tables E-16 and E-17). Effects observed in animals include changes in organ weight, morphological changes in tissues, increased mitotic activity, decreased concentrations of pituitary hormones, depressed glucose utilization, elevated serum glucose, and elevated insulin-like growth factor-1 (IGF-1). Effects reported in humans include "endocrine disturbances," impaired glucose tolerance, and elevated concentrations of pituitary hormones. Studies of the effects of fluoride on glucose metabolism and in diabetic animals are discussed below; information on other effects is extremely limited.

Pan (2015)[62] "Two-dimensional gel electrophoresis (2-DE) was used to detect fluoride-induced alterations in the proteome of the rat hippocampus. Male Sprague-Dawley rats (n=30) were subjected to treatments three weeks after weaning. Animals of the first group were injected intraperitoneally (i.p.) with aqueous NaF (20 mg/kg/body weight/day), the second group, injected with physiological saline, served as the control. After 30 days, the body weight of the fluoride-treated rats was lower than that of the control, and F- levels in serum were higher than in the control. The hippocampus was subjected to proteomic analysis, and the fluoride-treated group was found to contain 19 up-regulated and eight down-regulated proteins. The proteins, identified by mass-spectroscopic analysis of their fragments obtained after digestion, were found to be involved in amino acid biosynthesis, the insulin signaling pathway and various other crucial functions. Our results also provide useful information on the mechanism of the reduction of the learning ability and memory induced by F."

Pujary UR, Rao P, Mohanthy S, Krishna R, Reedy D. Correlation between serum fluoride and hyperglycemia in endemic fluorosis area. Indian Journal of Clinical Biochemistry. December 2007 22(Suppl) 383.

Prystupa, J. Fluorine—A current literature review. An NRC and ATSDR based review of safety standards for exposure to fluorine and fluorides. Toxicology Mechanisms and Methods. 2011. 21(2) 103-170.

[62] Pan Y, Lü P, Yin L, Chen K, He Y., Z Effect of fluoride on the proteomic profile of the hippocampus in rats. Naturforsch C. 2015 Jun 13. pii: /j/znc.ahead-of-print/znc-2014-4158/znc-2014-4158.xml. doi: 10.1515/znc-2014-4158. [Epub ahead of print]

Rashid K1, Sinha K, Sil PC. An update on oxidative stress-mediated organ pathophysiology. Food Chem Toxicol. 2013 Dec;62:584-600. doi: 10.1016

Rasmussen DD, Boldt BM, Wilkinson CW, Yellon SM, Matsumoto AM. Daily melatonin administration at middle age suppresses male rat visceral fat, plasma leptin, and plasma insulin to youthful levels. Endocrinology 1999. 140, 1009-1012.

Rigalli A, et al. Comparative study of the effect of sodium fluoride and sodium monofluorophosphate on glucose homeostasis in the rat. Drug Res 1995. 45(3) 289-92.

Rigalli A, Ballina JC Puche RC. Bone mass increase and glucose tolerance in rats chronically treated with sodium fluoride. Bone and Mineral. 1992. 16, 101-108.

Rigalli A. Inhibitory effect of fluoride on the secretion of insulin. Calico. Tissue Int. 1990. 46, 333-338.

Saber (2000)[63] "Influence of fluoride on exocrine pancreas cells was examined morphologically with traditional and prolonged osmium fixation techniques. . . . These findings indicate that fluoride disrupts the export of zymogens from the rER, resulting in formation of intracisternal granules and autophagosomes, and that the osmiophilic saccules participate in sequestration of cytoplasmic organelles in forming autophagosomes."

Shahed AR, et al. Effect of F on rat serum insulin levels in vivo. Journal of Dental Research. 1986. 65 756.

Tokar VI, Zyryanova VV, Shcherbakov SV. Chronic Fluorides Impact on Pancreas Islet Cells in Workers. Gigiena i Santitariia. November-December 1992, 42-44.

Trivedi N, Mithal A, Gupta SK, Godbole MM, Reversible impairment of glucose tolerance in patients with endemic fluorosis. Diabetologia, 1993 (36) 826-828.

Vandenberg LN, Colborn T, Hayes TB, Heindel JJ, Jacobs DR, Lee DH, Shioda T Soto AM, vom Saal FS, Welshons WV, Zoeller RT, Myers JP. Hormones and endocrine-disrupting chemicals: Low dose effects and nonmonotonic dose responses. Endocrine Reviews, 2012 33(3) 378-455.

Wang Z, Yang X, Yang S, Ren G, Ferreri M, Su Y, Chen L, Han B. Sodium fluoride suppress proliferation and induce apoptosis through decreased insulin-like growth factor-I expression and oxidative stress in primary cultured mouse osteoblast. Archives of Toxicology, November 2011 85(11) 1407-17

[63] Saburou Matsuo, Hiroshi Nakagawa, Ken-ichi Kiyomiya, Masaru Kurebe Fluoride-induced ultrastructural changes in exocrine pancreas cells of rats: fluoride disrupts the export of zymogens from the rough endoplasmic reticulum (rER) Archives of ToxicologyFebruary 2000, Volume 73, Issue 12, pp 611-617

Whitford, GM, Allman DW, Shahed AR. Topical fluorides: effects on physiologic and biochemical processes. J Dent Res. 1987 66(5) 1072-8.

Xie, Yong-ping, Ge Xiang-jin et al, Clinical Study of Effect of High Fluoride on the Function of the Pancreas Islet B Cells, Chinese Journal of Endemiology. 2000, 19(2) 84-85.

D. PINEAL GLAND:

In the seventeenth century, Descartes called the pineal gland the seat of the soul, the connection between the intellect and the body.[64] The pineal gland is about the size of a grain of rice (5mm X 8 mm) the only unpaired midline brain structure. It is located just below the brain in the quadrigeminal cistern and part of the epithalamus. It produces the hormone melatonin which regulates the body's circadian rhythm as well as the onset of puberty (See: Schlesinger ER, Overton DE, Chase HC, Cantwell KT (1956). Newburgh-Kingston caries-fluorine study X111. Pediatric findings after ten years. J Amer Dent Assoc 52: 296-306).

The NRC (2006) review of the literature to that date should be carefully considered and is quoted here.

"Pineal Gland Calcification

> "The pineal gland is a calcifying tissue; in humans, calcified concretions can be found at any age, although the likelihood increases with age (Vígh et al. 1998; Akano and Bickler 2003) and may be associated with menopause (Sandyk et al. 1992). The occurrence of pineal calcifications varies among different populations and nations (Vígh et al. 1998), possibly in association with the degree of industrialization (Akano and Bickler 2003), rates of breast cancer (Cohen et al. 1978), and high circannual light intensity near the equator (Vígh et al. 1998). Osteoporosis might be associated with fewer concretions (Vígh et al. 1998).
>
> "Melatonin secretion is well correlated with the amount of uncalcified pineal tissue (Kunz et al. 1999) but not with the size of pineal calcification (Vígh et al. 1998; Kunz et al. 1999). An increase in calcification of the pineal gland in humans probably represents a decrease in the number of functioning pinealocytes and a corresponding decrease in the individual's ability to produce melatonin (Kunz et al. 1999). The degree of calcification, relative to the size of an individual's pineal gland, has been suggested as a marker of the individual's decreased capability to produce melatonin (Kunz et al. 1999).
>
> "As with other calcifying tissues, the pineal gland can accumulate fluoride (Luke 1997, 2001). Fluoride has been shown to be present in the pineal glands of older people (14-875 mg of fluoride per kg of gland in persons aged 72-100 years), with the fluoride concentrations being positively related to the calcium concentrations in the pineal gland, but not to the bone fluoride, suggesting that pineal fluoride is not necessarily a function of cumulative fluoride exposure of the individual (Luke 1997, 2001). Fluoride has not been measured in the pineal glands of children or young adults, nor has there been any investigation of the relationship between pineal fluoride concentrations and either recent or cumulative fluoride intakes.

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[64] Descartes and the Pineal Gland (Stanford Encyclopedia of Philosophy)
Descartes R. "The Passions of the Soul" excerpted from "Philosophy of the Mind," Chalmers, D. New York: Oxford University Press, Inc.; 2002. ISBN 978-0-19-514581-6

“In Vitro Studies

“Few studies have examined the effects of fluoride on pineal function. NaF (2.5-20 mM, or fluoride at 47.5-380 mg/L) produces markedly increased adenylyl cyclase activity (up to four times control activity) of rat pineal homogenates in vitro (Weiss 1969a,b), as it does in other tissues (Weiss 1969a); ATPase activity in the homogenates was inhibited by up to 50% (Weiss 1969a). Potassium fluoride (7-10 mM, or fluoride at 133-190 mg/L) has been used experimentally to increase adenylyl cyclase activity in rat pineal glands in vitro (Zatz 1977, 1979).

“Animal Studies

“Details of the effect of fluoride on pineal function are presented in Appendix E, Table E- 15. Luke (1997) examined melatonin production as a function of age and time of day in Mongolian gerbils (Meriones unguiculatus). On an absolute basis, melatonin production by the low-fluoride group was constant at ages 7-28 weeks, with no difference between males and females. Relative to body weight, melatonin output declined progressively with age until adulthood (by 11.5 weeks in females and 16 weeks in males). In contrast, prepubescent gerbils fed the high-fluoride diet had significantly lower pineal melatonin production than prepubescent gerbils fed the low-fluoride diet. Relative to body weight, the normal higher rate of melatonin production in sexually immature gerbils did not occur.

“Sexual maturation in females occurred earlier in the high-fluoride animals (Luke 1997); males had increases in melatonin production relative to body weight between 11.5 and 16 weeks (when a decrease normally would occur), and testicular weight at 16 weeks (but not at 9 or 28 weeks) was significantly lower in high-fluoride than in low-fluoride animals. The circadian rhythm of melatonin production was altered in the high-fluoride animals at 11.5 weeks but not at 16 weeks. In high-fluoride females at 11.5 weeks, the nocturnal peak (relative to body weight) occurred earlier than in the low-fluoride animals; also, the peak value was lower (but not significantly lower) in the high-fluoride animals. In males, a substantial reduction ($P < 0.00001$) in the nocturnal peak (relative to body weight) was observed in the high-fluoride animals.

“Human Studies

“Although no studies are available that specifically address the effect of fluoride exposure on pineal function or melatonin production in humans, two studies have examined the age of onset of menstruation (age of menarche) in girls in fluoridated areas (Schlesinger et al. 1956; Farkas et al. 1983; for details, see Appendix E, Table

E-15) ; the earlier study was discussed by Luke (1997) as part of the basis for her research. No comparable information on sexual maturation in boys is available.”

12

Both Schlesinger et al. (1956) and Farkas et al. (1983) referred to tables of the distribution of ages at the time of first menstruation, but, in fact, both studies provided only frequencies by age (presumably at the time of study, in either 1-year or 0.5-year increments) of girls having achieved menarche by the stated age. Farkas et al. (1983) specifically indicated use of the probit method for ascertainment of the median age at menarche; the data provided by Schlesinger et al. (1956) appear to correspond to that method, but they do not specifically mention it. The probit (or status quo) method appears to be routinely used to estimate the median (or other percentiles of) age at menarche, sometimes in conjunction with an estimated mean age at menarche based on recall data (e.g., Wu et al. 2002; Anderson et al. 2003; Chumlea et al. 2003; Padez and Rocha 2003). According to Grumbach and Styne (2002), “The method of ascertainment of the age of menarche is of importance. Contemporaneous recordings are performed with the probit method of asking, ‘yes’ or ‘no,’ are you menstruating? These may be incorrect because of social pressures of the culture and socioeconomic group considered. Recalled ages of menarche are used in other studies and considered to be accurate within 1 year (in 90% of cases) during the teenage years and in older women, too.”

“In girls examined approximately 10 years after the onset of fluoridation (1.2 mg/L, in 1945) in Newburgh, New York, the average age at menarche was 12 years, versus 12 years 5 months among girls in unfluoridated Kingston (Schlesinger et al. 1956). The authors stated that this difference was not statistically significant. Note that those girls who reached menarche during the time period of the study had not been exposed to fluoride over their entire lives, and some had been exposed perhaps for only a few years before menarche (they would have been 8-9 years old at the time fluoridation was started). Those girls in Newburgh who had been exposed to fluoridated water since birth (or before birth) had not yet reached menarche by the time of the study.

“A later study in Hungary (Farkas et al. 1983) reported no difference in the menarcheal age of girls in a town with “optimal” fluoride concentration (1.09 mg/L in Kunszentmárton, median menarcheal age 12.779 years) and a similar control town (0.17 mg/L in Kiskunmajsa; median menarcheal age 12.79 years). This study shows postmenarcheal girls present at younger ages in the higher fluoride town than in the low-fluoride town, although the reported median ages were the same (Farkas et al. 1983).

“Discussion (Pineal Function)

“Whether fluoride exposure causes decreased nocturnal melatonin production or altered circadian rhythm of melatonin production in humans has not been investigated. As described above, fluoride is likely to cause decreased melatonin production and to have other effects on normal pineal function, which in turn could contribute to a variety of effects in humans. Actual effects in any individual depend on age, sex, and probably other factors, although at present the mechanisms are not fully understood.”

Luke (2001)[65] "By old age, the pineal gland has readily accumulated F and its F/Ca ratio is higher than bone. . . The pineal gland is a mineralizing tissue. . . The concretions are composed of hydroxyapatitie (HA). . . calcium is distributed throughout the pinealocytes: in the mitochondria, golgi apparatus, cytoplasm, and nucleus. Fluoride does not accumulate in the brain. Of all tissues, brain has the lowest fluoride concentrations. It is generally agreed that the blood-brain barrier restricts the passage of fluoride into the central nervous system. The human pineal gland is outside the blood-brain barrier. . . . pinealocytes have free access to fluoride in the bloodstream. This fact, coupled with the presence of HA, suggest that the pineal gland may sequester fluoride from the bloodstream." See Luke's graph below.

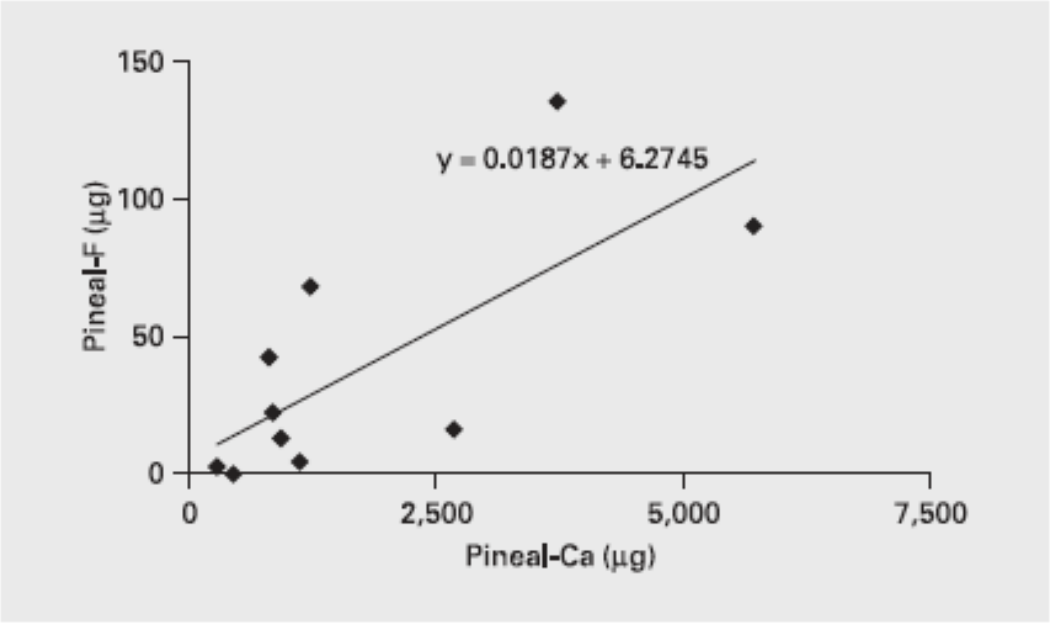

Fig. 1. The relationship between the calcium and fluoride contents of ten aged human pineal glands.

The pineal gland is bathed in cerebrospinal fluid but is not isolated by the blood brain barrier and is second only to the kidneys in blood profusion. (After the blood brain barrier is formed, the barrier mitigates fluoride transmission, but not for the pineal gland who's blood source is outside the blood brain barrier.) Innervation is sympathetic, parasympathetic, from the otic ganglia and trigeminal ganglion with nerve fibers containing the neuropeptide PACAP.

The pineal gland consists mainly of two types of pinealocytes, like photoreceptors, and

[65] Luke, J., Fluoride deposition in the human Pineal Gland. Caries Research | 2001; 35(2):125-128 | School of Biological Sciences, University of Surrey, Guildford, UK.

decline by way of apoptosis as the age of the organism increases.[66] High concentrations of fluoride and other toxins cause apoptosis. Type 1 cells are high in mitochondria and convert the amino acid tryptophan to serotonin then N-acetyl-serotonin and then to melatonin. Type 2 contain vacuoles, melatonin and are thought to act like endocrine and neuronal cells.[67]

Pinealocytes contain synaptic ribbons in children and adults but not human fetuses. Synaptic ribbons are important in neurotransmitter release.[68]

One of the difficulties in studying the pineal gland is the significant difference between rodents and higher vertebrates with rodent pineal gland lacking pineal gland neurons.

Although the effects of high concentrations of fluoride remain poorly understood, animal experiments have found that high doses of fluoride had a reduced melatonin production and an earlier onset of puberty.

The abundant melatonin levels in children are believed to inhibit sexual development which maybe a mechanism for early puberty with increased fluoride exposure.

> "Studies on rodents suggest that the pineal gland may influence the actions of recreational drugs, such as cocaine,[69] and antidepressants, such as fluoxetine (Prozac),[70] and its hormone melatonin can protect against neurodegeneration."[71]
>
> It is only a matter of time before researchers more clearly elucidate whether fluoride's effect is a contributing or causative factor for calcification and apoptosis of the pineal gland and the resulting decrease in melatonin production, early puberty and insomnia.

Kalisinska (2014) "Fluoride concentration in the pineal gland was significantly greater than in the bone and the brain of the duck."[72]

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[66] Polyakova, V. O., N. S. Linkova, and S. A. Pichugin (2011). "Changes in Apoptosis and Cell Proliferation in Human Pineal Gland during Aging". *Bulletin of Experimental Biology and Medicine* **150** (4): 468–70. doi:10.1007/s10517-011-1170-x. PMID 22268045.

[67] Khavinson, V. Kh, N. S. Linkova, I. M. Kvetnoy, T. V. Kvetnaia, V. O. Polyakova, and H. W. Korf (2012). "Molecular Cellular Mechanisms of Peptide Regulation of Melatonin Synthesis in Pinealocyte Culture". *Bulletin of Experimental Biology and Medicine* **153** (2): 255–58. doi:10.1007/s10517-012-1689-5.

[68] Spiwoks-Becker, I., C. Maus, S. Dieck, A. Fejtová, L. Engel, T. Wolloscheck, U. Wolfrum, L. Vollrath, and R. Spessert (2008). "Active Zone Proteins Are Dynamically Associated with Synaptic Ribbons in Rat Pinealocytes". *Cell and Tissue Research* **333** (2): 185–95. doi:10.1007/s00441-008-0627-3. PMC 2757586. PMID 18523806.

[69] Uz T, Akhisaroglu M, Ahmed R, Manev H (2003). "The pineal gland is critical for circadian Period1 expression in the striatum and for circadian cocaine sensitization in mice". *Neuropsychopharmacology* **28** (12): 2117–23. doi:10.1038/sj.npp.1300254. PMID 12865893

[70] Uz T, Dimitrijevic N, Akhisaroglu M, Imbesi M, Kurtuncu M, Manev H (2004). "The pineal gland and anxiogenic-like action of fluoxetine in mice". *Neuroreport* **15** (4): 691–4. doi:10.1097/00001756-200403220-00023. PMID 15094477.

[71] Manev H, Uz T, Kharlamov A, Joo J (1996). "Increased brain damage after stroke or excitotoxic seizures in melatonin-deficient rats". *FASEB J* **10** (13): 1546–51. PMID 8940301.

[72] Kalisinska E1, Bosiacka-Baranowska I, Lanocha N, Kosik-Bogacka D, Krolaczyk K, Wilk A, Kavetska K, Budis H, Gutowska I, Chlubek D. Fluoride concentrations in the pineal gland, brain and bone of goosander (Mergus

merganser) and its prey in Odra River estuary in Poland. Environ Geochem Health. 2014 Dec;36(6):1063-77. doi: 10.1007/s10653-014-9615-6. Epub 2014 Apr 18.

E. Adrenal Gland

Schetinina 1997)[73] The activity of carboxypeptidase (CP) H, the enzyme taking part in neuropeptide formation, and activity of recently described phenylmethylsulfonyl fluoride (PMSF)--inhibiting CP in males and females of white mongrel rats were studied. Minor differences between the CPH activities in brain regions were found in hippocampus. PMSF-inhibited activity of carboxypeptidase was significantly higher in females than in males in pituitary gland, adrenal gland, olfactory bulbus, optic and auditory bills, cerebellum, hippocampus, striatum, cerebral hemispheres and spleen. The CPH activity was 5-fold higher in ovaries than in testicles. PMSF-inhibited CP activity in testicles was 3.7-fold lower than in ovaries. Possible participation of basic CP in determination of sexual differences of some neuropeptide level and protein catabolism is studied.

Juska (1995)[74] A mathematical model relating the activity of adenylate cyclase (AC) with concentrations of stimulators, equilibrium dissociation constants, specific activity and efficacies of AC depending on the states of its binding sites has been developed and used for analysis of the data on activation of AC of bovine adrenal cortex plasma membranes presented in (De Foresta et al. (1987) FEBS Lett. 216, 107-112). Equilibrium dissociation constants. chi h and chi l, corresponding to high- and low-affinity forskolin-binding sites were estimated to be 0.37 and 17 microM: these constants characterize forskolin's potency more adequately than does ED50, the concentration eliciting half-asymptotic activity of AC. Corticotropin does not affect the affinity of AC for forskolin whereas fluoride increases this affinity, thus augmenting forskolin's potency. . . .”

Cannon (1994)[75] “Guanine nucleotide binding proteins (G proteins) act as signal transducers between membrane receptors and ion channels. In the present study, the whole-cell arrangement of the patch clamp technique was used to examine the effect of G proteins on K+ channels in cultured bovine adrenal chromaffin cells . . . . Treatment of the chromaffin cells with fluoride decreased nicotine-evoked secretion of catecholamines in a concentration-dependent manner. . . .”

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73 Shchetinina NVVernigora ANGengin MTAuthor information [Basic carboxypeptidase activity in rats of both sexes]. [Article in Russian] Ukr Biokhim Zh (1978). 1997 May-Jun;69(3):115-8.

74 Juska A, de Foresta B .Analysis of effects of corticotropin, forskolin and fluoride on activity of adenylate cyclase of bovine adrenal cortex. Biochim Biophys Acta. 1995 Jun 14;1236(2):289-98

75 Cannon SD[1], Wilson SP, Walsh KB. A G protein-activated K+ current in bovine adrenal chromaffin cells: possible regulatory role in exocytosis. Mol Pharmacol. 1994 Jan;45(1):109-16

Vitale (1993)[76] **“**The use of non-hydrolyzable analogues of GTP in permeabilized secretory cells suggests that guanine nucleotide-binding regulatory proteins (G proteins) may be involved in regulated exocytosis. . . . These results suggest that the secretory machinery in chromaffin cells can be blocked by activating a G(o) protein. Consistent with this finding, two other known activators of heterotrimeric G proteins, aluminum fluoride and benzalkonium chloride, inhibited calcium-evoked catecholamine secretion in streptolysin O-permeabilized chromaffin cells. We conclude that an inhibitory G(o) protein, possibly located on the membrane of secretory granules, is involved in the final stages of exocytosis in chromaffin cells.”

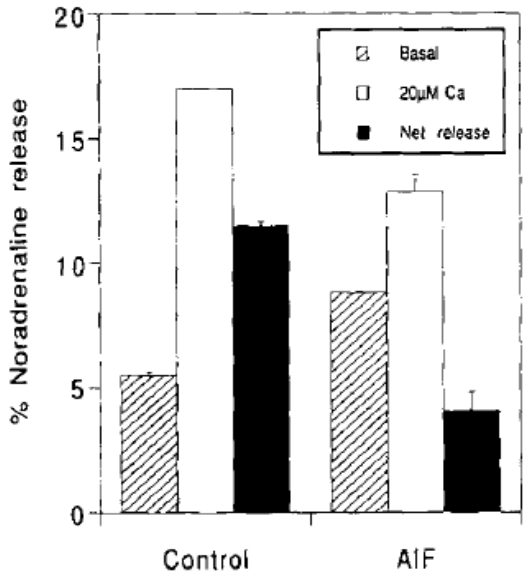

FIG. 6. **Effect of AlF_4^- on secretion from SLO-permeabilized chromaffin cells.** SLO-permeabilized cells were preincubated for 10 min in calcium-free KG medium in the presence (*AlF*) or absence (*Control*) of 20 mM NaF and 50 μM $AlCl_3$. Cells were then stimulated for 10 min with KG medium containing 20 μM free calcium (*open columns*). The basal release was estimated in calcium-free KG medium (*scratched columns*) and subtracted to obtain the net noradrenaline release (*closed columns*). AlF_4^- inhibited calcium-dependent noradrenaline release in chromaffin cells.

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76 Vitale N[1], Mukai H, Rouot B, Thiersé D, Aunis D, Bader MF. J Biol Chem. Exocytosis in chromaffin cells. Possible involvement of the heterotrimeric GTP-binding protein G(o).
1993 Jul 15;268(20):14715-23.

Ito (1991)[77] We have reported recently that prostaglandin E2 (PGE2) stimulated phosphoinositide metabolism in bovine adrenal chromaffin cells and that PGE2 and ouabain, an inhibitor of Na+, K(+)-ATPase, synergistically induced a gradual secretion of catecholamines from the cells. Here we examined the involvement of a GTP-binding protein(s) in PGE receptor-induced responses by using NaF. In the presence of Ca2+ in the medium, NaF stimulated the formation of all three inositol phosphates, i.e., inositol monophosphate, bisphosphate, and trisphosphate, linearly over 30 min in a dose-dependent manner (15-30 mM). This effect on phosphoinositide metabolism was accompanied by an increase in cytosolic free Ca2+. NaF also induced catecholamine release from chromaffin cells, and the dependency of stimulation of the release on NaF concentration was well correlated with those of NaF-enhanced inositol phosphate formation and increase in cytosolic free Ca2+. Although the effect of NaF on PGE2-induced catecholamine release in the presence of ouabain was additive at concentrations below 20 mM, there was no additive effect at 25 mM NaF. Furthermore, the time course of catecholamine release stimulated by 20 mM NaF in the presence of ouabain was quite similar to that by 1 microM PGE2, and both stimulations were markedly inhibited by amiloride, with half-maximal inhibition at 10 microM. Pretreatment of the cells with pertussis toxin did not prevent, but rather enhanced, PGE2-induced catecholamine release over the range of concentrations examined. These results demonstrate that NaF mimics the effect of PGE2 on catecholamine release from chromaffin cells and suggest that PGE2-evoked catecholamine release may be mediated by the stimulation of phosphoinositide metabolism through a putative GTP-binding protein insensitive to pertussis toxin.

De Foresta (1987)[78] “The diterpene forskolin maximally stimulated bovine adrenal cortex adenylate cyclase activity 9-fold with a concentration producing half-maximum effect (ED50) of about 4 microM. The effects of forskolin and the fully active corticotropin fragment ACTH (I 24) were additive over nearly the whole range of concentration of both effectors, indicating separate and independent mechanisms of action. By contrast, 10 mM NaF blocked forskolin action in the nanomolar range of the diterpene concentration, while it allowed a partial stimulation by forskolin in the micromolar range. NaF thus reveals a heterogeneity of forskolin action in the adrenal cortex plasma membranes. Moreover, our data suggest that ACTH and NaF activation effects, both mediated by the stimulatory regulatory protein Gs, proceed through different mechanisms.”

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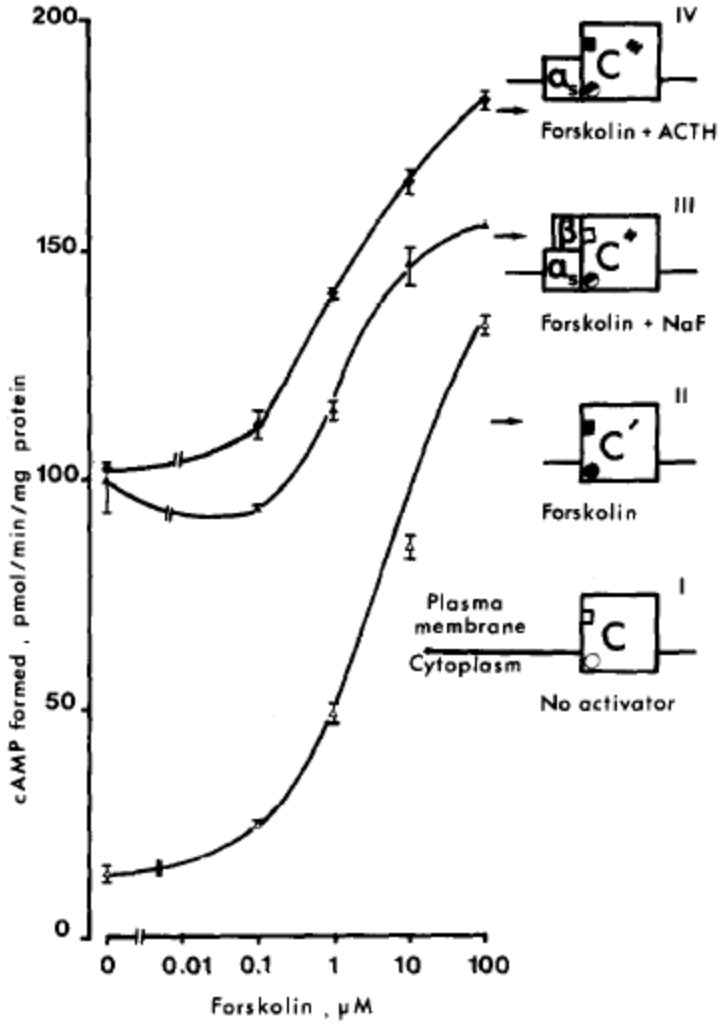
77 Ito S[1], Negishi M, Mochizuki-Oda N, Yokohama H, Hayaishi O., Sodium fluoride mimics the effect of prostaglandin E2 on catecholamine release from bovine adrenal chromaffin cells. J Neurochem. 1991 Jan;56(1):44-51.

78 de Foresta B, Rogard M, Gallay J., Adenylate cyclase of bovine adrenal cortex plasma membranes. Divergence between corticotropin and fluoride combined effects with forskolin. FEBS Lett. 1987 May 25;216(1):107-12.



Suketa (1985)[79] "Changes in adrenal function as a possible mechanism for elevated serum glucose by a single large dose of fluoride."

Wolff (1970)[80] "Chlorpromazine (3 x 10(-4)M) prevents the stimulation of adenyl cyclase activity in thyroid membranes produced by thyrotropin and prostaglandin, ACTH stimulation of adenyl cyclase in adrenal tissue, and glucagon- and epinephrine-stimulation of adenyl cyclase activity in liver. Baseline activity is unaffected. Parathyroid hormone stimulation of kidney preparations was not inhibited under these conditions. At chlorpromazine concentrations >3 x 10(-4)M F(-)-stimulated cyclase activity of thyroid and adrenal tissue was increased. Other phenothiazines, trifluoperazine, and prochlorperazine, have similar effects on thyrotropin and F(-)-

[79] Suketa Y, Asao Y, Kanamoto Y, et al. "Changes in adrenal function as a possible mechanism for elevated serum glucose by a single large dose of fluoride." To Appl Pharm. 1985. 80 199-205.

[80] Wolff J, Jones ABInhibition of hormone-sensitive adenyl cyclase by phenothiazines. Proc Natl Acad Sci U S A. 1970 Feb;65(2):454-9

stimulated cyclase activity of thyroid. Na(+)- K(+)-dependent ATPase of thyroid is also inhibited by chlorpromazine. Since thymol causes a similar dissociation of hormone- and F(-)-stimulated adenyl cyclase, it is concluded that the surface properties of these agents best account for their effects on adenyl cyclase.”

F. GONADS

Ovaries: The first study is by Yin (2015), and a significant portion is presented here because it illustrates the risks better and confirms earlier studies with depth.

Yin (2015)[81] "Reproductive toxicity has been an exciting topic of research in reproductive biology in recent years. Soluble fluoride salts are toxic at high concentrations; their reproductive toxicity was assessed in this study by administering different fluoride salt concentrations to mice. Continuous feeding for five weeks resulted in damage to the histological architecture of ovaries. The expression of genes, including Dazl, Stra8, Nobox, Sohlh1, and ZP3 gene, associated with oocyte formation were much lower in the experimental group as compared with the control group. The number of in vitro fertilization of mature oocytes were also much lower in the experimental group as compared with control. Moreover, the fertility of female mice, as assessed by mating with normal male mice, was also lower in experimental compared with control groups. The expression of the oocyte-specific genes: Bmp15, Gdf9, H1oo, and ZP2, which are involved in oocyte growth and the induction of the acrosome reaction, decreased with the fluoride administration. DNA methylation and histone acetylation (H3K18ac and H3K9ac) are indispensable for germline development and genomic imprinting in mammals, and fluoride administration resulted in reduced levels of H3K9ac and H3K18ac in the experimental group as compared with the control group, as detected by immunostaining. Our results indicate that the administration of high concentrations of fluoride to female mice significantly reduced the number of mature oocytes and hampered their development and fertilization. Thus, this study lays a foundation for future studies on fluoride-induced reproductive disorders in women.

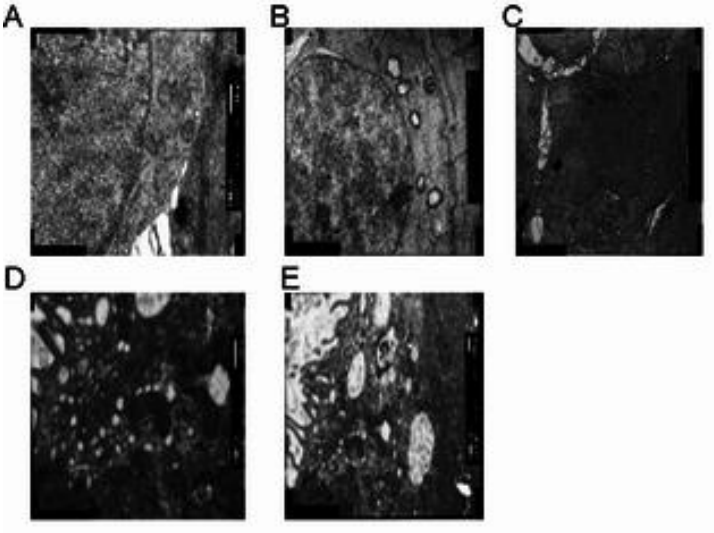


Fig 1. Effect of fluoride administration on ultrastructural features of ovary. (A-E): Ovaries were removed from female mice and ultrathin sections were cut. The histological architecture of ovaries from the control group (A, administered 0 mg/L NaF) and experimental (B-E; administered 50, 100, 150, and 200 mg/L NaF, respectively) groups was examined by transmission electron microscopy.

[81] Yin S[1], Song C[1], Wu H[1], Chen X[1], Zhang Y[1]. Adverse Effects of High Concentrations of Fluoride on Characteristics of the Ovary and Mature Oocyte of Mouse. PLoS One. 2015 Jun 8;10(6):e0129594. doi: 10.1371/journal.pone.0129594. eCollection 2015.

Effect of fluoride administration on expression of germline-specific genes in the ovary

RNA was isolated from ovaries of mice from the control and experimental groups, and the expression of potential germline-specific genes, particularly Dazl, Stra8, Sohlh1, Nobox, and Zp3, was analysed by RT-PCR. As observed in Fig 2A–2E, the expression of these genes was lower in the experimental groups (administered 50, 100, 150, or 200mg/L NaF) compared with the control group (administered 0mg/L NaF). Increase in fluoride concentration resulted in the decreased expressions of these genes, particularly Nobox, which was rarely detected in the experimental groups (Fig 2D).

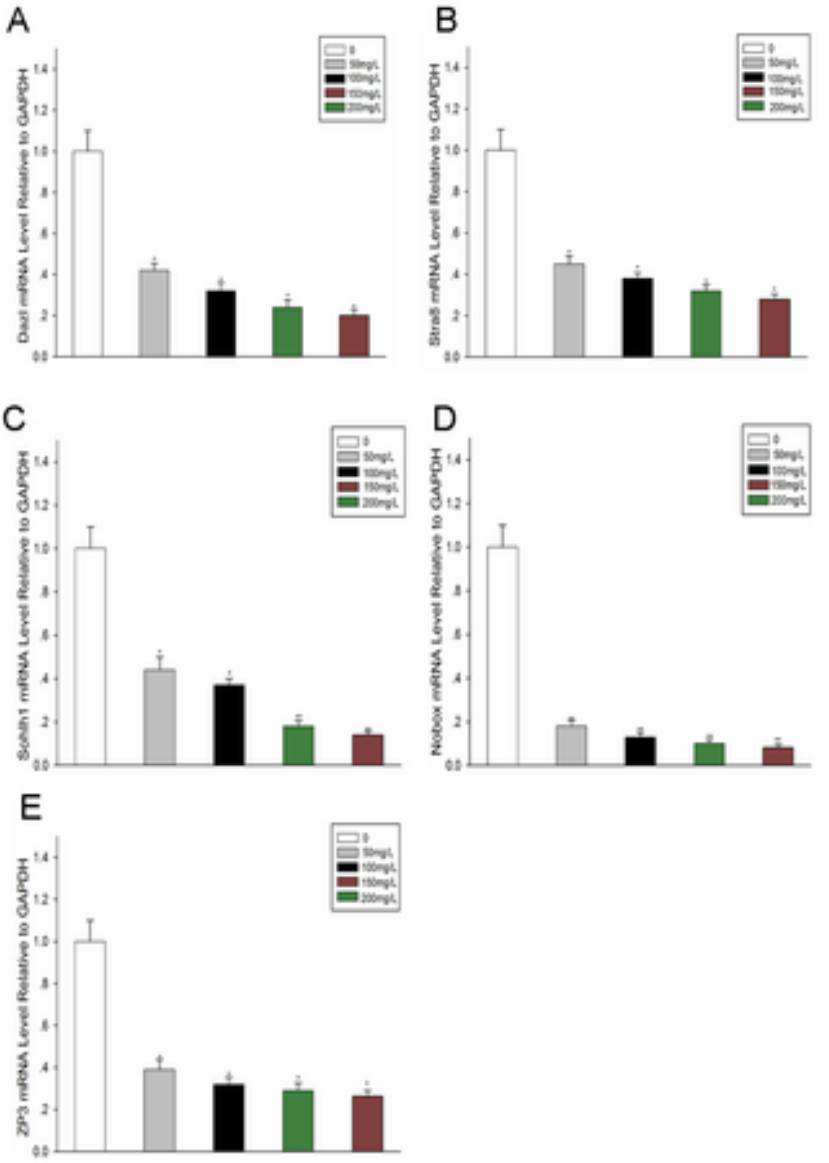


Fig 2. Effect of fluoride administration on expression of germline-specific genes in the ovary.

(A-E): mRNA was harvested from ovaries of mice from the control and experimental groups. qPCR was performed for assessing the relative expression levels of germline-specific genes (A: Dazl, B: Stra8, C: Sohlh1, D: Nobox, and E: Zp3) in the ovary. All data are presented as the mean ± SD and are derived from three independent experiments. *P<0.05; **P<0.01.

Effect of fluoride administration on the formation and in vitro/in vivo fertilization of mature oocytes

The effect of high concentrations of fluoride on the formation and in vitro fertilization of mature oocytes was investigated; furthermore, the fertility of female mice exposed to fluorides was examined by mating with normal male mice. Superovulation was achieved by the administration of 10 IU pregnant mare serum gonadotropin and 10 IU human chorionic gonadotropin before mating or harvesting of mature oocytes from the oviduct ampullae, as detailed in Materials and Methods. Fig 3A shows that the number of mature oocytes per ovary was significantly lower in the experimental groups (administered 50, 100, 150, or 200 mg/L NaF) compared with the control group (administered 0 mg/L NaF). This result is also reflected in the lower fertility of fluoride-administered female mice, as assessed by mating with normal male mice (Fig 3B), and in the lower efficiency of in vitro fertilization for the experimental groups compared with the control group (Fig 3C).

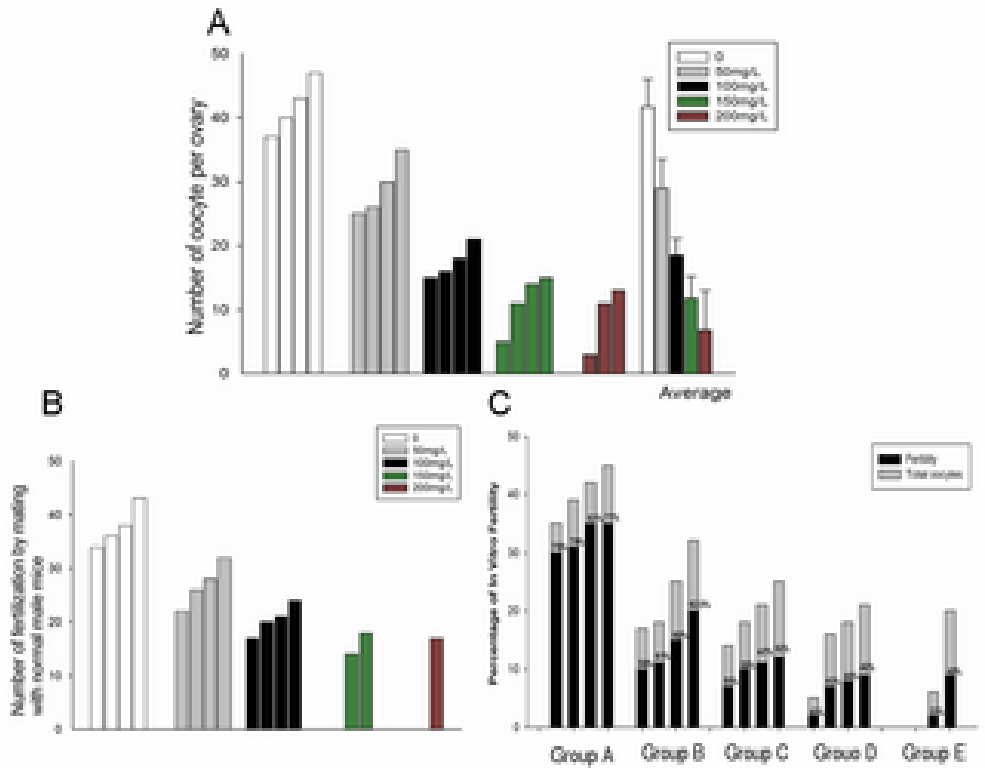


Fig 3. Effect of fluoride administration on formation and in vitro/in vivo fertilization of mature oocytes.

Mature oocytes were released from oviduct ampullae of superovulated mice ~14–16 h following the administration of human chorionic gonadotropin, and the number of the mature oocytes in the ovaries (A) and the efficiency of in vitro fertilization (C)were estimated. Mice from the control and experimental groups were mated with normal male mice following the administration of human chorionic gonadotropin for detecting the in vivo fertilization efficiency (B). (Data are presented as mean ± SD, with four mice (n = 4) per group).

Effect of fluoride administration on the expression of oocyte-specific genes

The results mentioned above indicate that the number and fertilization of mature oocytes are affected by high concentrations of fluoride. Therefore, the expression of oocyte-specific genes was evaluated by RT-PCR following the direct synthesis of cDNA from mature oocytes, as detailed in Materials and Methods. Four oocyte-specific genes, Bmp15, Gdf-9, Zp2, and H1oo, were focused on in this study because of their crucial functions. Expression of all these genes was found to be lower in the experimental groups compared with the control group, with negative association observed between the expression of these genes and fluoride concentration (Fig 4A–4D).

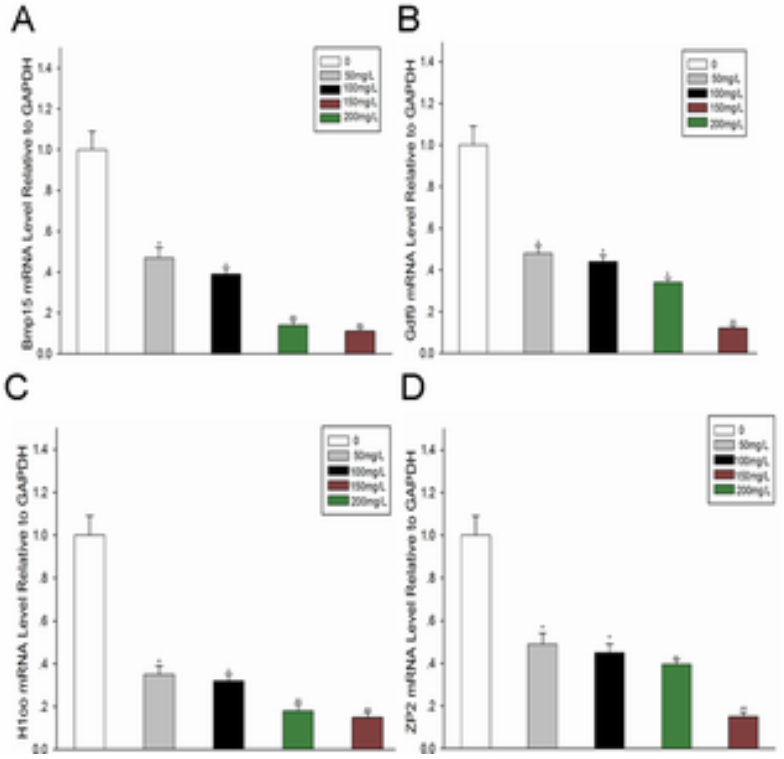


Fig 4. Effect of fluoride administration on the expression of oocyte-specific genes.

(A-D): mRNA was harvested from oocytes of mice from the control and experimental groups. RT-PCR was performed for assessing the relative expression levels of oocyte-specific genes (A: Bmp15, B: Gdf9, C: H1oo, and D: Zp2) in the oocytes. All data are presented as the mean ± SD and are derived from three independent experiments. *P<0.05; **P<0.01.

Effect of fluoride administration on DNA methylation and histone acetylation in mature oocytes

Immunostaining was performed to assess the effect of fluoride administration on global DNA methylation and histone acetylation (notably, H3K18ac and H3K9ac) in mature oocytes. As seen in Fig 5A, significant differences were not observed in 5-methylcytosine levels between the experimental (administered various fluoride

concentrations) and control groups. In contrast, lower levels of H3K18ac and of the H3K9ac were observed in the experimental groups (Fig 5B and 5C). (Not included)

Fig 5. Effect of fluoride administration on DNA methylation and histone acetylation in mature oocytes.
Mature oocytes were released from the oviduct ampullae of superovulated mice ~14–16 h following the administration of human chorionic gonadotropin. Immunofluorescence was performed for the detection of levels of 5-methylcytosine (A), H3K9ac (B), and H3K18ac (C). Each sample was stained with anti-5-methylcytosine (green), anti-H3K9ac (green), or anti- H3K18ac (green) antibodies and counterstained with DAPI (blue) to allow DNA visualization. Samples were visualized at (original magnification × 200) for exposure time of 200 ms (anti-5-methylcytosine, anti-H3K9ac and anti-H3K18ac).

Discussion

Fluorides are well recognized as pollutants, with a great deal of research focused on the environmental hazard that they cause [22, 23]. While the effects of fluoride exposure on fertility are known, its exact effect on the production of mature oocytes in mammalian ovaries remains to be investigated. The objective of this study is to explicitly assess the adverse effects of high concentrations of fluoride on the characteristics of mouse ovary and mature oocyte.

The consumption of large quantities of fluoride administration resulted in obvious damage to the histological architecture of mouse ovaries, as reported previously [14, 24]. Further, the effect of fluoride administration on the expression of germline-specific genes was investigated. Previous studies have reported the association between expression of particular ovary-specific genes and oocyte formation. Dazl, expressed during embryonic development in the female gonads of mice well before the onset of meiosis, functions in the first phase of gametogenesis during the differentiation, proliferation and maintenance of primordial germ cells and their substitutes [25]; Stra8 is required for meiotic progression in the mouse ovary, previous studies demonstrated that meiosis is a sex-specific event where germ cells undergo cellular differentiation to form oocytes or spermatozoa, with abnormal gene expression during meiosis leading to aberrant gamete formation, which is often a major cause of infertility in both males and females [26]; Nobox deficiency has been shown to disrupt early folliculogenesis and expression of oocyte-specific genes [27]; Sohlh1 is a transcription factors of the bHLH family and is specifically expressed in germ cells; it plays a role in oocyte differention, in female, such that Sohlh1 ablation causes oocyte loss in the neonatal ovary [28]; Zp3 plays an important role in the development of mouse zona pellucida, which is critical for fertilization [29]. This study revealed that the expression of these genes was much lower in the experimental groups compare with the control group and showed an inverse association with the concentration of fluoride adminstratered. The changes in histological architecture and expression of germline-specific genes in the ovary are likely to affect the formation and fertilization of mature oocytes. The effect of high

concentrations of fluoride on the formation of mature oocytes was investigated by inducing superovulation followed by collection of mature oocytes; moreover, in vitro fertilization and in vivo fertilization following mating with normal male mice were also assessed. The results obtained showed that increase in fluoride concentration resulted in lower yield of mature oocytes as well as lower efficiency of in vivo and in vitro fertilization in the experimental groups compared with the control group, which is in agreement with the observed expression of germline-specific genes, as detailed above.

The expression of the following oocyte-specific genes was also assessed following fluoride administration: Bmp15, which is involved in oocyte maturation and follicular development; Gdf-9, which regulates the oocyte growth and function of oocytes as well as growth and differention of granulose cell; zp2, which mediates species-specific sperm binding, induces acrosome reaction, and prevents post fertilization polyspermy; and H1oo, whose expression is restricted to the growing/maturing oocyte and to the zygote [30]. The expression of these oocyte-specific genes was decreased upon fluoride administration, which is expected to disrupt the normal maturation of oocyte.

The important role played by histone acetylation and DNA methylation in oogenesis is widely accepted. Previous studies have shown that occurrence of 5-methylcytosine in mammals genomes is crucial for normal mammalian development, while histone acetylation is associated with a transcriptionally active state and allows access of transfactor to DNA sequence. Abnormal epigenetic modification is expected to be detrimental to offspring as a consequence of DNA damage [31]. Therefore, the levels of global DNA methylation, and the active histone marks H3K9ac and H3K18ac were assessed in mature oocytes following the administration of fluoride to mice. The results revealed the absence of significant differences in the level of 5-methylcytosine between the experimental and control groups. However, the levels of H3K9ac and H3K18ac were lower in the experimental compared with the control groups and decreased with increase in fluoride concentration. Such abnormal epigenetic modification is likely to be particularly detrimental to offspring.

Behavioral differences were also observed in mice belonging to various experimental groups. Mice belonging to the experimental group D (administered 150 mg/L NaF) were observed to be thinner compared with the other groups, while the mice of group E (administered 200 mg/L NaF) consumed a much greater quantity of water; moreover, the mice of groups C, D, and E (administered 100, 150, and 200 mg/L NaF, respectively) displayed a tendency to closely approach one another. This is attributable to the neurotoxicity and behavioral changes caused upon fluoride consumption in animals [32, 33].

Taken together, this study suggests that the administration of high concentrations of fluoride to female mice not only results in ovarian damage but also significantly reduces the number and the fertilization potential of mature oocytes by reducing the expression of genes that play an important role in the normal development and maturation of oocytes. The results obtained in this study could thus be employed for statistical analysis of the association between exposure to high concentrations of fluoride and reproductive disorders in women.”

Geng (2014)[82] The toxicity of sodium fluoride (NaF) to female fertility is currently recognized; however, the mechanisms are unclear. Previously, we reported a reduction in successful pregnancy rates, ovarian atrophy and dysfunction following exposure to NaF. The purpose of this study was to elucidate the underlying molecular mechanisms. Female Sprague-Dawley rats (10 rats/group) received 100 or 200mg/L NaF in their drinking water for 6 months or were assigned to an untreated control group. Apoptotic indices and oxidative stress indicators in blood and ovarian tissue were analyzed following sacrifice. The results confirmed the NaF-induced ovarian apoptosis, with concomitant activation of oxidative stress. Further investigations in ovarian granular cells showed that exposure to NaF activated extracellular regulated protein kinase (ERK) and c-Jun NH2 kinase (JNK), disrupting the ERK and JNK signaling pathways, while p38 and PI3K remained unchanged. These data demonstrated that oxidative stress may play a key role in NaF-induced ovarian dysfunction by activating the apoptotic ERK and JNK signaling pathways.

Zhou (Feb 2013)[83] "The aim of this study was to investigate the effects of sodium fluoride (NaF) on female reproductive function and examine the morphology of the ovaries and uteri of rats exposed to NaF. . . . These results suggest that female reproductive function is inhibited by NaF and that exposure to NaF causes ovarian and uterine structural damage. NaF may thus significantly reduce the fertility of female rats."

Zhou (Sept 2013),[84] "Recognition of the harmful effects of sodium fluoride (NaF) on human reproduction is increasing, especially as it relates to female reproduction. However, the mechanism by which NaF interferes with female reproduction is unclear. The aims of the present study were to investigate the effects of fluoride exposure on female fertility and to elucidate the mechanisms underlying these effects. . . . These results suggest that the reproductive hormone reduction and the abnormalities of related receptor proteins expression are important factors underlying the decreased fertility observed in female rats that have been exposed to NaF."

Johanna (2013)[85] The effects of oral administration of sodium fluoride (NaF) and/or arsenic trioxide (As(2)O(3)) (5 mg and 0.5 mg/kg body weight, respectively) for 30 days were investigated on free radical induced toxicity in the mouse ovary. The

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[82] Geng Y[1], Qiu Y[2], Liu X[3], Chen X[4], Ding Y[5], Liu S[6], Zhao Y[7], Gao R[8], Wang Y[9], He J[10]. Sodium fluoride activates ERK and JNK via induction of oxidative stress to promote apoptosis and impairs ovarian function in rats. J Hazard Mater 2014 May 15;272:75-82. doi: 10.1016/j.jhazmat.2014.03.011. Epub 2014 Mar 18.

[83] Zhou Y[1], Zhang H, He J, Chen X, Ding Y, Wang Y, Liu X. Effects of sodium fluoride on reproductive function in female rats. Food Chem Toxicol. 2013 Jun;56:297-303. doi: 10.1016/j.fct.2013.02.026. Epub 2013 Feb 28.

[84] Zhou Y[1], Qiu Y, He J, Chen X, Ding Y, Wang Y, Liu X. The toxicity mechanism of sodium fluoride on fertility in female rats. Food Chem Toxicol. 2013 Dec;62:566-72. doi: 10.1016/j.fct.2013.09.023. Epub 2013 Sep 23.

[85] Jhala DD[1], Chinoy NJ, Rao MV. Mitigating effects of some antidotes on fluoride and arsenic induced free radical toxicity in mice ovary. Food Chem Toxicol. 2008 Mar;46(3):1138-42. doi: 10.1016/j.fct.2007.11.009. Epub 2007 Nov 23.

reversibility of the induced effects after withdrawal of NaF+As(2)O(3) treatment and by administration of antioxidant vitamins (C, E) and calcium alone as well as in combination were also studied. The combined treatment of NaF and As(2)O(3) impaired significantly ($p<0.001$) the production of free radical scavengers such as glutathione and ascorbic acid as well as antioxidant enzymes, namely, glutathione peroxidase (GSH-Px), superoxide dismutase (SOD) and catalase (Cat), thereby increasing ovarian lipid peroxides (LPO) which might have rendered the ovary susceptible to injury. The withdrawal of the combined (NaF and As(2)O(3) for 30 days) treatment caused partial recovery in the ovary, which was more pronounced ($p<0.001$) by treatment with vitamin C, calcium, or vitamin E alone and in combination. Hence the induced toxicity was transient and reversible.

Hou (2013) "To explore the influence of water fluoride exposure on reproductive hormones in female. Cross-sectional study was conducted in seven villages of a county in Henan province by using simple random sampling including high fluoride area, defluoridation project area and control area on April, 2011 based on the preliminary study results of fluoride concentration in drinking water. Women who were born and growth or lived in the village at least 5 years and aged 18-48 years old were recruited using cluster sampling. They were divided into high fluoride group (HFG, 116 subjects), defluoridation project group (DFPG, 132 subjects) and control group (CG, 227 subjects) in accordance with the above areas. All subjects accepted questionnaire and physical checkup. . . Fluoride exposure may influence reproductive hormones in female, especially in ovulatory and luteal phase of menstrual cycle."[86]

The Oxford Journals (2006)[87] is many pages in length and a good source for review of the ovary and developing follicle. In part, they report:

"Ovarian follicle development is a complex process that begins with the establishment of what is thought to be a finite pool of primordial follicles and culminates in either the atretic degradation of the follicle or the release of a mature oocyte for fertilization. This review highlights the many advances made in understanding these events using transgenic mouse models. Specifically, this review describes the ovarian phenotypes of mice with genetic mutations that affect ovarian differentiation, primordial follicle formation, follicular growth, atresia, ovulation and corpus luteum (CL) formation. In addition, this review describes the phenotypes of mice with mutations in a variety of genes, which affect the hormones that regulate folliculogenesis. Because studies using transgenic animals have revealed a variety of reproductive abnormalities that resemble many reproductive disorders in women, it is likely that studies using transgenic mouse models will impact our understanding of ovarian function and fertility in women."

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[86] Hou JX1, Yang YJ, Gong B, Li SH, Ding Z, Wen SB, Li SQ, Cheng XM, Cui LX, Ba Y. [The influence of high fluoride exposure in drinking water on endocrine hormone in female]. [Article in Chinese] Zhonghua Yu Fang Yi Xue Za Zhi. 2013 Feb;47(2):142-6.

[87] Ovarian follicle development and transgenic mouse models, Hum. Reprod. Update Oxford Journals (September/October 2006) 12 (5): 537-555. doi: 10.1093/humupd/dml022 First published online: May 25, 2006 Update (September/October 2006) 12 (5): 537-555. doi: 10.1093/humupd/dml022

Stan (1994)[88] “A review of fluoride toxicity showed decreased fertility in most animal species studied. The current study was to see whether fluoride would also affect human birth rates. A U.S. database of drinking water systems was used to identify index counties with water systems reporting fluoride levels of at least 3 ppm. These and adjacent counties were grouped in 30 regions spread over 9 states. For each county, two conceptionally different exposure measures were defined, and the annual total fertility rate (TFR) for women in the age range 10–49 yr was calculated for the period 1970–1988. For each region separately, the annual TFR was regressed on the fluoride measure and sociodemographic covariables. Most regions showed an association of decreasing TFR with increasing fluoride levels. Meta-ana/ysis of the region-specific results confirmed that the combined result was a negative TFR/fluoride association with a consensus combined p value of .0002-.0004, depending on the analytical scenario. There is no evidence that this outcome resulted from selection bias, inaccurate data, or improper analytical methods. However, the study is one that used population means rather than data on individual women. Whether or not the fluoride effect on the fertility rate found at the county level also applies to individual women remains to be investigated.”

[88] Stan C. Freni., Exposure to high fluoride concentrations in drinking water is associated with decreased birth rates. Journal of Toxicology and Environmental Health, 1994, Volume 42, Issue 1, pages 109-121

TESTES:

Han (2015)[89] "Numerous studies have shown that fluoride exposure adversely affected the male reproductive function, while the molecular mechanism is not clear. The present study was to investigate the effects of fluoride exposure (60days) on the expressions of reproductive related genes, serum sex hormone levels and structures of the hypothalamus-pituitary-testicular axis (HPTA), which plays a vital role in regulating the spermatogenesis in male mice. In this study, 48 male mice were administrated with 0, 25, 50, and 100mg/L NaF through drinking water. Results showed that the malformation ratio of sperm was significantly increased ($P<0.05$). At transcriptional level, the expression levels of follicle-stimulating hormone receptor (FSHR), luteinizing hormone receptor (LHR), inhibin alpha (INHα), inhibin beta-B (INHβB), and sex hormone binding globulin (SHBG) mRNA in testis were significantly decreased ($P<0.05$). Moreover, histological lesions in testis and ultrastructural alterations in hypothalamus, pituitary and testis were obvious. However, the same fluoride exposure did not lead to significant changes of related mRNA expressions in hypothalamus and pituitary ($P>0.05$). Also, there were no marked changes in serum hormones. Taken together, we conclude that the mechanism of HPTA dysfunction is mainly elucidated through affecting testes, and its effect on hypothalamus and pituitary was secondary at exposure for 60days."

Hamza (2015)[90] "Sodium fluoride (NaF) intoxication is associated with oxidative stress and altered antioxidant defense mechanism. The present study was carried out to evaluate the potential protective role of blackberry and quercetin (Q) against NaF-induced oxidative stress and histological changes in liver, kidney, testis and brain tissues of rats. . . .RESULTS AND CONCLUSIONS: NaF caused an elevation in lipid peroxidation level paralleled with significant decline in glutathione peroxidase, glutathione reductase, glutathione S-transferase, superoxide dismutase and catalase activities as well as the total antioxidant activity in liver, kidney, testes and brain. Some histopathological changes were detected in all tested tissues of the NaF treated group. Q and BBJ had successfully maintained normal histological architecture and mitigated the induction of oxidative stress caused by NaF. Q effectively reduced the elevation in thiobarbituric acid reactive substances level and restored the activities of antioxidant enzymes in liver, kidney, testis and brain. Less histopathological changes were observed in Q+NaF and BBJ+NaF treated groups. As a result, BBJ and Q significantly reduced NaF-induced oxidative and histological changes in rats. In the combination of BBJ and Q against NaF toxicity, the effects were more severe than from separate exposure, thus indicating that these flavonoids exhibited synergistic effects on all antioxidant and histological parameters."

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[89] Han H1, Sun Z1, Luo G2, Wang C3, Wei R1, Wang J4., Fluoride exposure changed the structure and the expressions of reproductive related genes in the hypothalamus-pituitary-testicular axis of male mice. Chemosphere. 2015 Sep;135:297-303.

[90] Hamza RZ, El-Shenawy NS, Ismail HA. Protective effects of blackberry and quercetin on sodium fluoride-induced oxidative stress and histological changes in the hepatic, renal, testis and brain tissue of male rat. J Basic Clin Physiol Pharmacol. 2015 May;26(3):237-51.

Song (2014)[91] '"The biological effects of fluoride on human health are often extensive, either beneficial or detrimental. Among the various effects of fluoride exposure in different organs, the reproductive tract is particularly susceptible to disruption by fluoride at a sufficient concentration. It has attracted much attention to the effect of sodium fluoride on male fertility, gestational female, and offspring. Herein, we applied a widespread natural compound sodium fluoride (NaF) and investigated the effects of acute NaF exposure on Leydig cells, including their proliferation, apoptosis, and signal pathway changes. Our results demonstrated that high dosage of NaF could inhibit cell proliferation by stress-induced apoptosis, which was confirmed by cellular and molecular evidences. We found that fluoride exposure affected the expression levels of stress response factors, signal transduction components, and apoptosis-related proteins, including caspase-3/caspase-9, B-cell lymphoma 2 (Bcl-2), and Bax. This study suggests that the complex effects of fluoride on Leydig cells are closely related to its dosage."

Geng (2014) "The toxicity of sodium fluoride (NaF) to female fertility is currently recognized; however, the mechanisms are unclear. Previously, we reported a reduction in successful pregnancy rates, ovarian atrophy and dysfunction following exposure to NaF. The purpose of this study was to elucidate the underlying molecular mechanisms. . . The results confirmed the NaF-induced ovarian apoptosis, with concomitant activation of oxidative stress. Further investigations in ovarian granular cells showed that exposure to NaF activated extracellular regulated protein kinase (ERK) and c-Jun NH2 kinase (JNK), disrupting the ERK and JNK signaling pathways, while p38 and PI3K remained unchanged. These data demonstrated that oxidative stress may play a key role in NaF-induced ovarian dysfunction by activating the apoptotic ERK and JNK signaling pathways."[92]

Wang (2014) "Sodium fluoride (NaF) has been found to interfere with the reproductive system of animals. However, the cellular mechanisms underlying the reproductive toxicity of fluoride are unclear. The present study aims to define a possible mechanism of NaF-induced reproductive toxicity with respect to mineral, oxidative stress and c-Fos expression and the role of aluminum (Al) in intervening the toxic effect of NaF on rat testes. . . The present study suggested that NaF could decrease the contents of Ca, Fe and Mg and enhance oxidative stress leading to c-Fos overexpression, and some deleterious effects were more prominent at lower NaF intake. Furthermore, Al within the research concentration could minimize reproductive toxicity caused by fluoride."[93]

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[91] Song Gh[1], Wang RL, Chen ZY, Zhang B, Wang HL, Liu ML, Gao JP, Yan XY. Toxic effects of sodium fluoride on cell proliferation and apoptosis of Leydig cells from young mice. J Physiol Biochem. 2014 Sep;70(3):761-8. doi: 10.1007/s13105-014-0344-1. Epub 2014 Jul 30.

[92] Geng Y1, Qiu Y2, Liu X3, Chen X4, Ding Y5, Liu S6, Zhao Y7, Gao R8, Wang Y9, He J10. Sodium fluoride activates ERK and JNK via induction of oxidative stress to promote apoptosis and impairs ovarian function in rats. J Hazard Mater. 2014 May 15;272:75-82. doi: 10.1016/j.jhazmat.2014.03.011. Epub 2014 Mar 18.

[93] Wang J1, Zhang H, Xu F, Xu F, Zhang K, Zhang Y. The antagonism of aluminum against fluoride-induced oxidative stress and c-Fos overexpression in rat testes. Toxicol Mech Methods. 2014 Feb;24(2):136-41. doi: 10.3109/15376516.2013.869779. Epub 2013 Dec 16.

Yang (2014)[94] “Investigated the effects of N-acetylcysteine (NAC) on endoplasmic reticulum stress of sertoli cells induced by sodium fluoride (NaF). METHODS: Rat sertoli cells were exposed to various concentration of (0, 6, 12, 24 μg/ml) sodium fluoride with or without 2 mmol/L NAC for 24 hours. The cell viability was evaluated using trypan blue exclusion test. Intracellular reactive oxygen species (ROS) was measured using the fluorescent probe DCFH-DA. Western blot was used to test the expression of GRP78, PERK and CHOP. RESULTS: It was found that treatment with NAC (2 mmol/L) restored the reduced cell viability and excessive oxidative stress (P < 0.01). Moreover, fluoride exposure upregulated the expression of GRP7 8, PERK and CHOP protein (P <0. 01). NAC was also found to suppress the levels of GRP78, PERK and CHOP expression in NaF-treated cells (p<0.01). CONCLUSION: Endoplasmic reticulum stress signaling pathways were activated by ROS, and NAC attenuate endoplasmic reticulum stress through inhibiting the levels of ROS in NaF-treated sertoli cells.”

Zhang (2013)[95] “Long-term excessive fluoride intake is known to be toxic and can damage a variety of organs and tissues in the human body. However, the molecular mechanisms underlying fluoride-induced male reproductive toxicity are not well understood. In this study, we used a rat model to simulate the situations of human exposure and aimed to evaluate the roles of endoplasmic reticulum (ER) stress and inflammatory response in fluoride-induced testicular injury. Sprague-Dawley rats were administered with sodium fluoride (NaF) at 25, 50 and 100mg/L via drinking water from pre-pregnancy to gestation, birth and finally to post-puberty. And then the testes of male offspring were studied at 8weeks of age. Our results demonstrated that fluoride treatment increased MDA accumulation, decreased SOD activity, and enhanced germ cell apoptosis. In addition, fluoride elevated mRNA and protein levels of glucose-regulated protein 78 (GRP78), inositol requiring ER-to-nucleus signal kinase 1 (IRE1), and C/EBP homologous protein (CHOP), indicating activation of ER stress signaling. Furthermore, fluoride also induced testicular inflammation, as manifested by gene up-regulation of tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2), in a nuclear factor-κB (NF-κB)-dependent manner. These were associated with marked histopathological lesions including injury of spermatogonia, decrease of spermatocytes and absence of elongated spermatids, as well as severe ultrastructural abnormalities in testes. Taken together, our results provide compelling evidence that ER stress and inflammation would be novel and significant mechanisms responsible for fluoride-induced disturbance of spermatogenesis and germ cell loss in addition to oxidative stress.”

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[94]Yang Y, Huang H, Feng D, Liu W, Cheng X, Ba Y, Cui L.[Effects. of N-acetylcysteine on fluoride-induced endoplasmic reticulum stress in sertoli cells]. [Article in Chinese] Wei Sheng Yan Jiu. 2014 Sep;43(5):805-8, 813.

[95]Zhang S[1], Jiang C, Liu H, Guan Z, Zeng Q, Zhang C, Lei R, Xia T, Gao H, Yang L, Chen Y, Wu X, Zhang X, Cui Y, Yu L, Wang Z, Wang A. Fluoride-elicited developmental testicular toxicity in rats: roles of endoplasmic reticulum stress and inflammatory response. Toxicol Appl Pharmacol. 2013 Sep 1;271(2):206-15. doi: 10.1016/j.taap.2013.04.033. Epub 2013 May 22.

Deng (2013) “To discuss the significance of calcineurin (CaN) and nuclear factor of active T cells 1 (NFATc1) in the damage mechanism of the testis of rats with chronic fluorosis. . . The changes in the signaling pathway of expression of CaN may be involved in the injury mechanism of testis tissues of rats with chronic fluorosis.”[96]

Dimcevici (2013) “It has been revealed that excessive fluoride intake on long-term is associated with toxic effects and can damage a variety of organs and tissues in the human body, including the male reproductive system. . . The results indicate that natrium fluoride administered in different doses, even at homeopathic dose or at allopathic-homeopathic dose, determined vacuolar dystrophy of epididymal epithelial cells, vacuolar dystrophy of linear seminal cells and necrosis.”[97]

Xiao (2011)[98] “The rat fluorosis models were successfully established. The fluoride content in testis was significantly increased in all the fluorosis groups(P<0.01). Testicular structures were damaged in all of fluoride groups. The TNOS, iNOS activities, and MDA content of each fluoride group were significantly higher than that of the control group on day 120 and 180 (P<0.05 or 0.01). The TNOS, iNOS activities, and MDA content significantly increased in a dose dependent manner (P<0.05 or 0.01). The SOD activities significantly decreased in all the fluoride groups (P<0.05 or 0.01). **CONCLUSIONS:** Endemic fluoride poisoning caused by coal burning can cause disorders in the oxidative system and antioxidative system in rat testis. The oxidative stress may play an important role in the fluorides induced reproductive toxicity in male rats.

Hao (2010)[99] **OBJECTIVE:** To study of endocrine disturbing effect of fluoride on human hypothalamus-hypophysis-testis axis hormones. **METHODS:** Sunying County, Kaifeng City was selected as polluted district which the fluoride in drinking water was 3.89 mg/L, and Shenlilou county was selected as control district which the fluoride was less than 1.0 mg/L. 150 individual lived there more than 5 years were selected randomly. And investigated by medical examination, then blood and urine sample were collected, and the serum level of gonadotropin-releasing hormone (GnRH), luteinizing hormone (LH), testosterone (T) and estradiol (E2) were measured by RIA method, and the urine level of fluoride were measured. Other than that, the concentration of fluoride in the water, food, soil and air were detected by the

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[96] Deng CN1, Yu YN2, Xie Y1, Zhao LN1. [Expression of calcineurin and nuclear factor of activated T cells 1 in testis of rats with chronic fluorosis]. [Article in Chinese] Zhonghua Yu Fang Yi Xue Za Zhi. 2013 Dec;47(12):1142-7.

[97] Dimcevici Poesina N1, Bălălău C, Bârcă M, Ion I, Baconi D, Baston C, Băran Poesina V. Testicular histopathological changes following sodium fluoride administration in mice. Rom J Morphol Embryol. 2013;54(4):1019-24.

[98] Xiao YH[1], Sun F, Li CB, Shi JQ, Gu J, Xie C, Guan ZZ, Yu YN.[Effect of endemic fluoride poisoning caused by coal burning on the oxidative stress in rat testis]. Zhongguo Yi Xue Ke Xue Yuan Xue Bao. 2011 Aug;33(4):357-61. doi: 10.3881/j.issn.1000-503X.2011.04.002 [Article in Chinese]

[99] Hao P[1], Ma X, Cheng X, Ba Y, Zhu J, Cui L.[Effect of fluoride on human hypothalamus-hypophysis-testis axis hormones]. Wei Sheng Yan Jiu. 2010 Jan;39(1):53-5. [Article in Chinese]

standard methods. **RESULTS:** The concentrations of fluoride in the water, food and soil of the fluoride polluted district were significantly higher than those of control district ($P < 0.05$), and the concentration fluoride in the air of two district were not found. There was no significant difference of serum level of GnRH between fluoride polluted district and control district ($P > 0.05$). The serum level of LH in men of fluoride polluted district was significantly higher than that of control group ($P < 0.05$), and the serum level of T in men of fluoride polluted district was significantly less than that of control group ($P < 0.05$). There was no significant difference of serum level of LH between fluoride polluted district and control district ($P > 0.05$), and the serum level of T in women of fluoride polluted district was significantly higher than that of control group ($P < 0.05$). There was no significant difference of serum level of E2 between fluoride polluted district and control district ($P > 0.05$). **CONCLUSION:** Fluoride could effect hormone levels of each layer of the hypothalamus-hypophysis-testis axis, and show the reproductive endocrine disturbing effects. The reproductive endocrine disturbing effects of male maybe more severe than those of female.

Ma (2008)[100] **OBJECTIVE:** To study the endocrine disturbing effect of fluorin on Hypothalamus-Hypophysis-Testis axis in male rats. **METHODS:** A total of 36 Wister male rats weighting 60-70 g were randomly divided into group I (high fluoride group of F-100 mg/l), group II (low fluoride group of F- 30 mg/l), group III (control group with pure water), with 12 rats in each group. Fluoride was administered with drinking water for 8 weeks. Then the level of procreation hormone in serum was detected by RIA method. And the spermatozoa quality was analysized. **RESULTS:** There was difference between group I, group II and group III each other ($P < 0.05$) in body weight. As to right testis weight, there was difference between group I, group II and group III each other ($P < 0.05$). Epididymide organic coefficient in group II and group I were lower than that in group III ($P > 0.05$). Compared with group III, the counts amount of sperm and the rates of sperm mobility in group II and group I singnificantly increased ($P < 0.05$), and the rates of sperm aberration in group II and group I significantly decreased ($P < 0.05$), compared with group II, the sperm quality of group I descreased significantly ($P < 0.05$). The level of GnRH in three groups were significant difference between each groups ($P <0.05$). The level of FSH in three groups were significant difference between each groups ($P < 0.05$). The level of ICSH in three groups were no significant difference between each groups ($P > 0.05$). The level of T in Group I is significant lower than that of in Group II and Group III ($P < 0.05$). The level of E2 in Group I is significant higher than that of in Group II and Group III ($P < 0.05$).

Gupta (2007)[101] "The present study was undertaken to evaluate the effect of fluoride toxicity on the reproductive system of male rats. Sexually mature male Wistar rats were exposed to 2, 4, and 6 ppm sodium fluoride in their drinking water for 6 months

[100] Ma X[1], Cheng X, Li F, Guo J.[Experimental research on endocrine disturbing effect of fluorin on hypothalamus-hypophysis-testis axis in male rats]. Wei Sheng Yan Jiu. 2008 Nov;37(6):733-5. [Article in Chinese]

[101] Gupta RS[1], Khan TI, Agrawal D, Kachhawa JB. The toxic effects of sodium fluoride on the reproductive system of male rats. Toxicol Ind Health. 2007 Oct;23(9):507-13.

ad libitum. Sperm motility and density in cauda epididymis were assessed. Biochemical and histological analysis were performed in reproductive organs. Fluoride treatment brought about a significant decrease in the weight of testis, epididymis, and ventral prostate. The sperm motility and density were significantly reduced. There was a marked reduction in the number of primary spermatocyte, secondary spermatocyte, and spermatids. The Sertoli cell counts and their cross sectional surface areas were significantly decreased. The Leydig cell nuclear area and the number of mature Leydig cells were also significantly decreased. The protein content of the testis and epididymis were significantly reduced. Fructose in the seminal vesicles and cholesterol in testes were increased significantly. In conclusion, sodium fluoride administrated in drinking water of 2, 4, and 6 ppm concentration for 6 months to male rats adversely affected their fertility and reproductive system."

Jiang (2007)[102] OBJECTIVE: To study the damages of fluoride on the male reproductive system in rat testes. METHODS: A total of 30 male SD rats were randomly divided into control group, high, low dose fluorine treated groups, which were given normal saline ,20 mg/kg sodium fluoride, and 10mg/kg sodium fluoride respectively. After 39 days the change of the weight of rats and the number of sperms were observed. The change of telomerase reverse transcriptase(TERT) and proliferating cell nuclear antigen (PCNA) were observed by using in situ hybridization and radioimmunoassay respectively.RESULTS: The weight was (273.39 +/- 20.68), (240.00 +/- 21.39) g in NaF treated groups, which was lower than that in control group($P < 0.05$); The rate of TERT expression in germ cells of testes in NaF treated groups was (13.89 +/- 4.86)% and (6.33 +/- 4.42)% respectively, which was significantly lower than that in control group ($P < 0.05$). The rate of PCNA expression in germ cells of tests in NaF treated groups was (0.71 +/- 0.05)%, (0.60 +/- 0.08)% respectively, which also was significant lower than that in control group($P < 0.05$). The number of sperms was (18.31 +/- 1.20)10(10)/L, (9.17 +/- 1.38)10(10)/L, which was lower than that in control group ($P < 0.05$). CONCLUSION: Fluorine possibly damages the male reproductive system by reducing the expression of TERT and PCNA.

Oncu (2007)[103] (Note: Oncu's rats were given 0.7 mg/l NaF, the same as USPHS recommended "This experiment was designed to investigate the histological and lipid peroxidation effects of chronic fluorosis on testes tissues of first- and second-generation rats. Sixteen virgin female Wistar rats were mated with eight males (2:1) for approximately 12 h to obtain first-generation rats. Pregnant rats were divided into two groups: controls and fluoride-given group, each of which containing five rats. Pregnant rats in the fluoride-given group were exposed to a total dose of 30 mg/l sodium fluoride (NaF) in commercial drinking water containing 0.07 mg/l of NaF throughout the gestation and lactation periods. After the lactation period, the young

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[102] Jiang Q[1], Song XK, Cui QH, Chen LJ. [Effect of fluoride on expression of telomerase reverse transcriptase expression and proliferating cell nuclear antigen in germ cells of rats' testes]. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi. 2007 Feb;25(2):96-9. [Article in Chinese]

[103] Oncü M[1], Kocak A, Karaoz E, Darici H, Savik E, Gultekin F. Effect of long-term fluoride exposure on lipid peroxidation and histology of testes in first- and second-generation rats. Biol Trace Elem Res. 2007 Sep;118(3):260-8.

animals (first generation, F1) were exposed to the same dose of NaF in drinking water for 4 months. At the end of the 4 months of experimental period, nine randomly chosen male rats (F1) were killed and testes tissues were taken for histopathological and biochemical analysis. The remaining eight female rats were mated with four males (2:1) for approximately 12 h to obtain second-generation rats. Six female were identified as pregnant and treated with similarly throughout the gestation and the lactation periods. After the lactation period, the young male animals (second generation, F2) were also treated in the same way for 4 months. At the end of the 4 months of experimental period, nine randomly chosen male rats (F2) were killed and testes tissues were collected for histopathological and biochemical analysis. The rats in the control group were applied the same procedure without NaF administration. In biochemical analysis of the fluoride given F1 and F2 rats, it has been found that plasma fluoride levels and testes thiobarbituric acid reactive substance levels were significantly increased when compared with the control group. In F1 and F2 rats, similar histopathological changes were observed. In both groups, spermatogenesis was severely reduced. Spermatogonia and primary spermatocytes were normal, however, there was a widespread degeneration in other spermatogenic cell lines of the seminiferous epithelium. The histological structures of the Sertoli and interstitial Leydig cells were normally observed. It is concluded that chronic fluorosis exposure leads to a remarkable destruction in testes tissues of F1 and F2 rats via lipid peroxidation."

Dvoráková-Hortová K (2007)[104] Increasing infertility, due to pathological changes on sperm, has become a serious issue. Eco-toxicological effect of rising concentration of fluorides can be enhanced in the presence of aluminium ions by forming fluorometallic complexes, analogues of phosphate groups that interfere with the activity of G-proteins and P-type ATPases, which are part of several signalling pathways during sperm maturation. In order for sperm to gain fertilizing ability, they must undergo in the female reproductive tract, capacitation that includes tyrosine phosphorylation and consequent actin polymerization. The present paper reports the findings of 3-month oral toxicity in mice of fluorides at the concentrations 0, 1, 10, and 100ppm and their synergic action with aluminium at dose of 10ppm. There were no mortalities, clinical signs of discomfort or body weight loss during the experiment. The analysis revealed, for the concentrations of 10 and 100ppm, abnormalities of spermatogenesis and ability of epididymal spermatozoa to capacitate in vitro, as the result of decreased sperm head tyrosine phosphorylation and actin polymerization. The enhancing overload caused by fluorides represents a potential factor, having an impact on function of sperm, hence contributing to a growing infertility in the human population.

Zakrzewska (2006)[105] "RESULTS: The semen was diluted in 0.9% NaCl and was found

[104] Dvoráková-Hortová K[1], Sandera M, Jursová M, Vasinová J, Peknicová J. The influence of fluorides on mouse sperm capacitation. Anim Reprod Sci. 2008 Oct;108(1-2):157-70. Epub 2007 Aug 6.

[105] Zakrzewska H, Udala J. (2006). [In vitro influence of sodium fluoride on adenosine triphosphate (ATP) content in ram semen]. [Article in Polish]. Ann Acad Med Stetin. 52 Suppl 1:109-11

to contain 12.4 micromol ATP 10-(-9) spermatozoa. ATP content was reduced with rising concentrations of NaF: by 74.6% at 20 tmol/L; by 75.5% at 100 micromol/L; by 90.8% at 200 imol/L; and by 99.9% at 10(5) micromol/L. The correlation between ATP content and sperm motility was significant (r = 0.4990). There was no correlation between ATP content and sperm density."

Krasowska (2004)[106] "Previous work has shown that a high fluoride intake in rodents leads to histopathological changes in the germinal epithelium of testes that is associated with zinc deficiency. The purpose of this study was to determine whether supplemental dietary Zn would protect against testicular toxicity induced by fluoride in a small rodent, the bank vole. The 4-month exposure period to fluoride (200 microg/ml of drinking water) induced histopathological changes (hemorrhage in interstitium, necrosis and apoptosis in seminiferous tubule epithelium) which were accompanied by decreased testicular zinc concentration and increased lipid peroxidation. Supplemental dietary zinc (110-120 microg/g) together with fluoride treatment resulted in complete reversal of the fluoride-mediated effects. However, supplemented dietary Zn did not affect the accumulation of fluoride in the testes and bone. These data suggest that a zinc-enriched diet protects seminiferous tubules against fluoride toxicity by preventing the fluoride-induced testicular zinc deprivation."

Zakrzewska (2002)[107] "The activities of androgen-dependent enzymes—acid phosphatase (ACP), lactate dehydrogenase (LDH), and gamma-glutamyl transferase (y-GT-10S)—decreased significantly when the ejaculate was treated with NaF at concentrations of 20, 100, 200 µmol/L (0.38; 1.9; 3.8 ppm F-), but they returned to the initial value of the control at 0.1 mol/L (1900 ppm F-). . . . These changes undoubtedly affect the physiological functions of the sperm."

Ghosh (2002)[108] "This study examined the effect of sodium fluoride, a water pollutant important through the world, including India, on testicular steroidogenic and gametogenic activities in relation to testicular oxidative stress in rats. Sodium fluoride treatment at 20mg/kg/day for 29 days by oral gavage resulted in significant diminution in the relative wet weight of the testis, prostate, and seminal vesicle without alteration in the body weight gain. Testicular delta(5),3beta-hydroxysteroid dehydrogenase (HSD) and 17beta-HSD activities were decreased significantly along with significant diminution in plasma levels of testosterone in the fluoride-exposed group compared to the control. Epididymal sperm count was decreased significantly in the fluoride-treated group and qualitative examination of testicular sections

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[106] Krasowska A[1], Włostowski T, Bonda E. Zinc protection from fluoride-induced testicular injury in the bank vole (Clethrionomys glareolus). Toxicol Lett. 2004 Mar 7;147(3):229-35.

[107] Zakrzewska H, et al. (2002). In vitro influence of sodium fluoride on ram semen quality and enzyme activities. Fluoride 35: 153-160.

[108] Ghosh D[1], Das Sarkar S, Maiti R, Jana D, Das UB. Testicular toxicity in sodium fluoride treated rats: association with oxidative stress. Reprod Toxicol. 2002 Jul-Aug;16(4):385-90.

revealed fewer mature luminal spermatozoa in comparison to the control. The seminiferous tubules were dilated in treated animals. Fluoride treatment was associated with oxidative stress as indicated by an increased level of conjugated dienes in the testis, epididymis, and epididymal sperm pellet with respect to control. Peroxidase and catalase activities in the sperm pellet were decreased significantly in comparison to the control. The results of this experiment indicate that fluoride at a dose encountered in drinking water in contaminated areas exerts an adverse effect on the male reproductive system and this effect is associated with indicators of oxidative stress."

Susheela (1996)[109] "OBJECTIVE: The present study focuses on serum testosterone concentrations in patients with skeletal fluorosis, in order to assess the hormonal status in fluoride toxicity. METHODS: Serum testosterones were compared for patients afflicted with skeletal fluorosis (n = 30) and healthy males consuming water containing less than 1 ppm fluoride (Control 1, n = 26) and a second category of controls (Control 2, n = 16): individuals living in the same house as the patients and consuming same water as patients but not exhibiting clinical manifestations of skeletal fluorosis. RESULTS: Circulating serum testosterones in skeletal fluorosis patients were significantly lower than those of Control 1 at $p < 0.01$. Testosterone concentrations of Control 2 were also lower than those of Control 1 at $p < 0.05$ but were higher than those of the patient group. CONCLUSIONS: Decreased testosterone concentrations in skeletal fluorosis patients and in males drinking the same water as the patients but with no clinical manifestations of the disease compared with those of normal, healthy males living in areas nonendemic for fluorosis suggest that fluoride toxicity may cause adverse effects in the reproductive system of males living in fluorosis endemic areas."

Chinook (1994)[110] "Fluoride-treated sperm [4,750 ppm for 20 minutes] exhibited a high percent of morphologic abnormalities, including a large number (10.59%) of elongated heads and 2.1% amorphous heads. The tail also revealed splitting (2.19%), coiling (11.6%) and deflagellation (22.43%). A few sperm had bent necks, and 16.75% of spermatozoa showed a diminutive acrosome. . . . These changes may have caused loss of membrane integrity and reduced metabolic activity, which ultimately resulted in deterioration of forward progression rating. The treatment caused a significant enhancement in poor to fair forward progression and failure of good and excellent forward progression, leading to a significant decline in sperm motility. . . . The depleted sperm GSH in the present investigation strongly suggests that, like several exogenous compounds, fluoride is largely dependent upon glutathione for detoxification."

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[109] Susheela AK1, Jethanandani P., Circulating testosterone levels in skeletal fluorosis patients. J Toxicol Clin Toxicol. 1996;34(2):183-9.

[110] Chinoy NJ, Narayana MV. (1994). In vitro fluoride toxicity in human spermatozoa. Reprod Toxicol. 8(2):155-9.

Chubb (1985a)[111] “Our studies indicate that 3 ppm fluoride ions significantly inhibit testosterone secretion by rat testes perfused in vitro. . . . In conclusion, Oxypherol-E.T. contains contaminants that are toxic to endocrine organs. Fluoride ion may be the primary endocrine toxicant.”

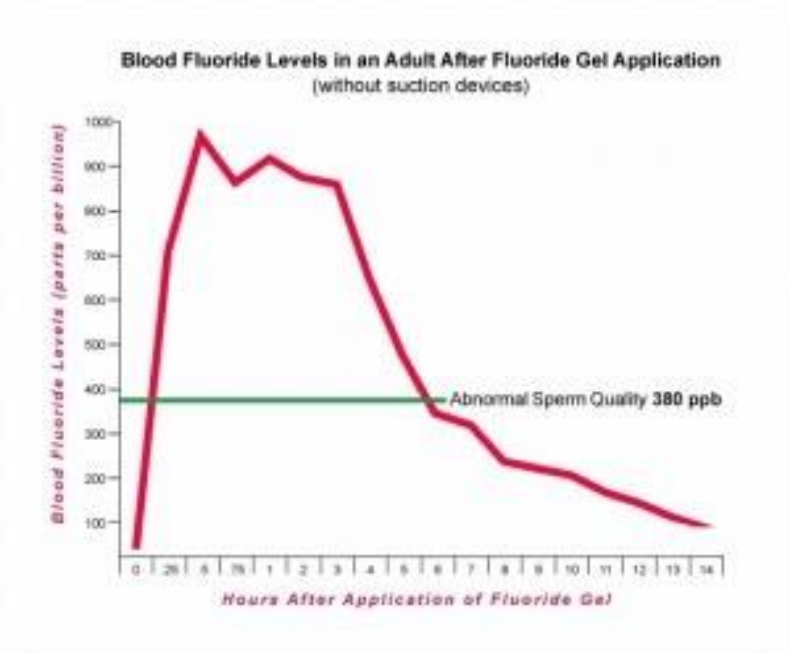
REVIEW BY FAN (2011)[112] "The enhancing overload caused by fluorides represents a potential factor, having an impact on function of sperm, hence contributing to a growing infertility in the human population.” (Animal Reproduction Science, 2008

“Male infertility is responsible for about 50% of the fertility problems that couples face. Infertility in males is often the result of reduced sperm court, abnormal sperm quality (e.g., reduced motility and altered morphology), or altered levels of sex hormones (e.g., reduced testosterone). A review of over 100 studies of sperm density from 1938 to 1996 found that human sperm count has significantly declined in North America and Europe since the 1940s. (Swan 2000) While the causes of this decline are not entirely known, fluoride exposure — particularly from high-concentration topical fluoride gels — must be considered as one of the potential contributing factors.

“In 2002 and again in 2006, researchers from Poland reported that exposing ram semen to 0.38 parts per million (20 umol/L) of fluoride for 5 hours was sufficient to “cause a statistically significant decrease in the motility of spermatoza and the number of intact acrosomes.” (Zakrzewska 2002). As the authors noted, these changes would “undoubtedly affect the physiological function of the sperm.” Prior to the Polish team’s findings, researchers from Texas found that infusing testis with higher, but still relatively modest, levels of fluoride (4.75 ppm) “unequivocally” inhibited the synthesis of testosterone. (Chubb 1985).

[111] Chubb C. (1985a). Reversal of the endocrine toxicity of commercially produced perfluorochemical emulsion. Biology of Reproduction 33(4):854-8.

[112] http://fluoridealert.org/issues/health/fertility/



"The Polish team's findings are of particular importance when considering that from the 1960s to the 1990s, the use of high-concentration topical fluoride gels produced blood concentrations in boys and men that far exceeded 0.38 ppm. In tests on both children and adults, the use of topical fluoride gels at the dental office has been found to produce blood fluoride concentrations as high as 1.2 ppm, or four times higher than the concentration found to damage sperm. (Ekstrand 1980, 1981). Further, the blood fluoride concentrations have been found to exceede 0.38 ppm for up to six hours after treatment (longer than the length of time that the Polish researchers exposed the semen). Although most dentists now use precautionary procedures (e.g., suction devices) to reduce blood fluoride concentrations following application of fluoride gels, available data shows that children will still routinely ingest enough fluoride from topical gels to reach blood fluoride concentrations exceeding 0.38 ppm.

"Consistent with the in vitro research, over 60 animal studies have found that fluoride adversely impacts the male reproductive system. The effects — which have been observed in rats, mice, chickens, and rabbits — include: (1) decreases in testosterone levels; (2) reduced sperm motility; (3) altered sperm morphology; (4) reduced sperm quantity; (5) increased oxidative stress; (6) and reduced capacity to breed. While the studies have generally used high doses, many of the studies have found effects at dosages that would produce blood fluoride concentrations far lower than the concentrations used in the in vitro research. See, e.g., Sun (2010); Dvoráková-Hortová (2008); Sharma (2008); Reddy (2007); Gupta (2007); Pushpalatha (2005). In one of the few studies to report blood fluoride concentrations, Mexican researchers reported that blood fluoride levels of 0.2 to 0.26 ppm for an eight week period caused increased oxidative stress, reductions in sperm motility and reduced fertility in male rats. Izquierdo-Vega, et al. (2008).

"While some studies have not found any effects of high fluoride dosages on the reproductive functions of male rats , these studies represent the distinct minority in the field. (Sprando & Collins 1996, 1997, and 1998). One possible explanation for the discrepancy in findings is the nutritional health of the tested animals. As with many other areas of fluoride research, nutritional deficiencies (e.g., protein) unequivocally exacerbate fluoride's reproductive effects, whereas nutritional supplementation (e.g., protein or anti-oxidants such as vitamin C) can significantly prevent or ameliorate these effects.

"Consistent with the in vitro and animal research, studies of human populations have reported associations between fluoride exposure and damage to the male reproductive system. Most notably, a scientist at the Food & Drug Administration reported in 1994 that populations in the United States with more than 3 ppm fluoride in their water had lower "total fertility rates" than populations with lower fluoride levels. (Freni 1994). While 3 ppm is a higher concentration than used in water fluoridation programs (0.7 to 1.2 ppm), it is still considered a "safe" level by the EPA. To date, no U.S. health agency has attempted to replicate Freni's findings. However, three studies of highly fluoride-exposed populations in China and India have found that high fluoride exposure is associated with reduced male fertility. (Chen 1997; Liu 1988; Neelam 1987). In addition, five studies from China, India, Mexico, and Russia.have found that high-fluoride exposure is associated with reduced male testosterone levels. (Hao 2010; Ortiz 2003; Susheela 1996; Michael 1996; Tokar 1977)." End of FAN quote.

G. ENTEROENDOCRINE (See the Pancreas for Pancreatic enteroendocrine)

Wikipedia: Enteroendocrine cells are specialized endocrine cells of the gastrointestinal tract and pancreas. They produce gastrointestinal hormones or peptides in response to various stimuli and release them into the bloodstream for systemic effect, diffuse them as local messengers, or transmit them to the enteric nervous system to activate nervous responses.[1][2] Enteroendocrine cells of the intestine are the most numerous endocrine cells of the body.[3][4][5] In a sense they are known to act as chemoreceptors, initiating digestive actions and detecting harmful substances and initiating protective responses.[6][7] Enteroendocrine cells are located in the stomach, in the intestine and in the pancreas. Intestinal

enteroendocrine cells are not clustered together but spread as single cells throughout the intestinal tract.[8]Hormones secreted include somatostatin, motilin, cholecystokinin, neurotensin, vasoactive intestinal peptide, and enteroglucagon.[9]

Searches did not readily find studies specifically evaluating the enteroendocrine cells and fluoride. We do have studies on fluoride's effect on the gastrointestinal cells as a group. If gastrointestinal cells are being harmed with fluoride, it is reasonable to expect enteroendocrine cells to be similarly involved.

Social (2010)[113] "Results reveal that (1) the urine fluoride levels decreased in 67% and 53% of the pregnant women respectively, who attended ANCs (antenatal clinic to reduce fluoride intake and improve diet) during 1st and 2nd trimester of pregnancy. (2) An increase in Hb upon withdrawal of fluoride followed by nutritional intervention in 73% and 83% respectively has also been recorded. (3) Body mass index (BMI) also enhanced. (4) The percentage of pre-term deliveries was decreased in sample group compared to control. (5) Birth weight of babies enhanced in 80% and 77% in sample group women who attended ANC in 1st and 2nd trimester respectively as opposed to 49% and 47% respectively in the control group. (6) The number of low birth weight babies was reduced to 20% and 23% respectively in sample as opposed to 51% and 53% in control groups."

NRC (2006)[114] "It is important to realize that GI effects depend more on the net concentration of the aqueous solution of fluoride in the stomach than on the total fluoride dose in the fluid or solid ingested. The presence of gastric fluids already in the stomach when the fluoride is ingested can affect the concentration of the fluoride to which the gut epithelium is exposed. The residual volume of stomach fluid ranges between 15 and 30 mL in people fasting overnight (Narchi et al. 1993; Naguib et al. 2001; Chang et al. 2004). Such volumes would decrease the fluoride concentration of a glass of drinking water by only about 10%. In Table 9-1, the concentrations of fluoride in the stomach were estimated from the mean reported fluoride exposures. A dilution factor was used when it was clear that the subjects already had fluid in their stomach. The results from the water fluoridation overfeed reports (concentrations of fluoride in the stomach between 20 and 250 mg/L) indicate that GI symptoms, such as nausea and vomiting, are common side effects from exposure to high concentrations of fluoride.

"Fluoride supplements are still routinely used today in areas where natural fluoride in the drinking water falls below 0.7 mg/L. In an early clinical trial using fluoride supplements, Feltman and Kosel (1961) administered fluoride tablets containing 1.2

[113] Susheela AK et al, Effective interventional approach to control anaemia in pregnant women. Current Science, May 25, 2010. 98(10):1320-30.
[114] National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C. p 229-230.

mg of fluoride or placebo tablets to pregnant mothers and children up to 9 years of age. They determined that about 1% of the subjects complained of GI symptoms from the fluoride ingredient in the test tablets. If it is assumed that the stomach fluid volume after taking the fluoride supplement was approximately 250 mL, the concentration to which the stomach mucosal lining was exposed was in the neighborhood of 5 mg/L. GI effects appear to have been rarely evaluated in the fluoride supplement studies that followed the early ones in the 1950s and 1960s. Table 9-1 suggests that, as the fluoride concentration increases in drinking water, the percentage of the population with GI symptoms also increases. The table suggests that fluoride at 4 mg/L in the drinking water results in approximately 1% of the population experiencing GI symptoms."

Connett (2012) provided an overview of fluoride and gastric mucosa: "When fluoride has been used (at doses of 18-34 mg/day) as an experimental treatment for osteoporosis, gastric pain is one of the two main side effects consistently encountered. To better understand how fluoride causes this effect, researchers have sought to determine how fluoride affects the tissue that lines the gastrointestinal tract.

In a study published in the British Medical Journal, the researchers gave a *single* dose of 20 mg/F to 12 healthy volunteers and then examined, both microscopically and macroscopically, the impact on the gastric mucosa. The examination revealed that the fluoride dose caused erosions (petechiae) in the stomach of *all* the subjects tested, with six of the subjects having similar effects in the antrum as well. Other findings were as follows:
"In four subjects a layer of clotted blood was found over a large part of the gastric mucosa… Three components of the gastric mucosa were affected by fluoride: the surface epithelium, the gastric pits, and the superficial stroma. The damaged epithelial cells were smaller than undamaged ones, and the vacuoles containing mucus were reduced in size or had disappeared. The most severely damaged epithelium was disrupted or totally lost. The most characteristic changes in the gastric pits were irregular dilation and flattening of the epithelial cells. There was also a noticeable loss of mucin."
SOURCE: Spak CJ, et al. (1989). Tissue response of gastric mucosa after ingestion of fluoride. British Medical Journal 298:1686-87. [See study]

Despite the fact that tissue damage was found in all 12 volunteers, only 4 of the volunteers experienced nausea. Thus, "using nausea as the first sign of fluoride toxicity might not be valid as all subjects showed mucosal damage."

In a follow-up study, published in 1990, the authors examined the impact of lower doses of fluoride to determine whether the use of self-applied topical gels could cause damage to children's gastric system. In the study, the volunteers ingested a single dose of just **3 to 9 mg** of fluoride, which is considerably lower than what some people ingest from higher-concentration professional fluoride gels. Despite using low

doses, the authors again found significant damage to the gastric mucosa. They described this damage as follows:
"After F exposure, histopathological changes were found in nine out of ten patients. The surface epithelium of the gastric mucosa showed the greatest effects: In two cases, there was a slight dilation of the gastric pits and a focal loss of surface epithelium. In some cases, the mucus-containing intercellular vacuoles were reduced in size, and focal hemorrhages within the epithelium occurred."
SOURCE: Spak CJ, et al. (1990). Studies of human gastric mucosa after application of 0.42% fluoride gel. Journal of Dental Research 69:426-9.

Interestingly, the authors note that they "could not find any correlation between the presence of mucosal injuries and the size of the ingested F dose." Based on this, they suggest that individual variability to fluoride may be a more important predictor of fluoride-induced gastric damage when low levels of fluoride are ingested. As they note: "The various reactions of the mucosa to F exposure are most likely due to individual variations in gastric fluid volume, gastric pH, and motility and mucosal resistance."
Such findings emphasize the difficulty of determining a uniform "safe" fluoride dose for an entire population. Indeed, if significant variability to fluoride is observed among 10 otherwise healthy humans, the variability is likely to be quite vast when studying the population as a whole, especially when including those with diseases that render one particularly susceptible to fluoride toxicity.

EXCERPTS FROM STUDIES EXAMINING FLUORIDE'S EFFECT ON GASTRIC MUCOSA IN HUMANS

"In a prospective case controlled study, we evaluated the adverse effects of long-term fluoride ingestion on the gastrointestinal tract. Ten patients with otosclerosis who were receiving sodium fluoride 30 mg/day for a period of 3-12 months, and 10 age- and sex-matched healthy volunteers were included… Seven subjects (70%) ingesting fluoride had abdominal pain, vomiting, and nausea. Petechiae, erosions, and erythema were seen on endoscopy in all the subjects, but not in the controls. Histological examination of the gastric antral biopsy showed chronic atrophic gastritis in all the subjects but in only one (10%) healthy volunteer. Scanning electron microscopic examination showed "cracked-clay" appearance, scanty microvilli, surface abrasions, and desquamated epithelium in the subjects ingesting fluoride, but not in the controls. We conclude that long-term fluoride ingestion is associated with a high incidence of dyspeptic symptoms as well as histological and electron microscopic abnormalities."
SOURCE: Das TK, et al. (1994). Toxic effects of chronic fluoride ingestion on the upper gastrointestinal tract. Journal of Clinical Gastroenterology 18(3):194-9.
"In a randomized double-blind study with two parallel groups of 10 male healthy volunteers each the response of gastric mucosa after a 7 days ingestion of sodium fluoride tablets (NaF) or sodium monofluorophosphate tablets (MFP) was compared. Gastroscopic evaluations were performed before treatment, day 1 and day 7… In the MFP-group no severe gastric lesions were observed, whereas in the NaF-group in 7 of

the 10 subjects significant gastric mucosal lesions including acute hemorrhages and free blood in the gastric lumen were found. The differences of the lesions scores in both groups were statistically significant (p = 0.0015)… In summary, under the experimental conditions used MFP is well tolerated by the stomach while NaF produces significant gastric mucosal lesions."
SOURCE: Muller P, et al. (1992). Sodium fluoride-induced gastric mucosal lesions: comparison with sodium monofluorophosphate. Z Gastroenterol. 30(4):252-4.
"Dental prophylaxis with APF gels (1.23%) may cause gastric distress as a side-effect. This gastric irritation is probably due to a direct toxic effect of fluoride (F), swallowed in conjunction with the treatment, on the gastric mucosa. The aim of the present study was to investigate whether–and to what extent–a dental treatment with 3 g of a 0.42%-F gel could affect the gastric mucosa due to inadvertent swallowing of the gel. Ten subjects underwent a control gastroscopy, and two weeks later, a second gastroscopy was performed two h after a F gel treatment. During the gastroscopy, the mucosa was examined and the injuries graded according to an arbitrary scale. Four biopsies of the antral and corpus regions of the stomach were taken and evaluated histologically. The mean (+/- SD) amount of F retained after the application was 5.1 +/- 2.1 mg, i.e., 40% of the applied amount of F. Petechiae and erosions were found in the mucosa in seven of the ten patients. The histopathological evaluation revealed changes in nine of ten patients, with the surface epithelium as the most affected component of the mucosa. The present study clearly shows that a treatment with a F gel of rather low F concentration may result in injuries to the gastric mucosa. The importance of current recommended guidelines so that the amount of F swallowed during a gel application can be minimized is emphasized. From a toxicological standpoint, the use of a low-F gel instead of a 1.23%-F gel in small children is recommended for avoidance of adverse gastric effects."
SOURCE: Spak CJ, et al. (1990). Studies of human gastric mucosa after application of 0.42% fluoride gel. Journal of Dental Research 69:426-9.
"We studied the response of the gastric mucosa after a single dose of fluoride. Twelve healthy volunteers (age range 22-45, four men and eight women) underwent two endoscopies after overnight fasts. One endoscopy was a control and the other was performed two hours after subjects ingested 20 ml sodium fluoride solution containing 20 mg fluoride (53 mmol/l)… After taking fluoride all subjects had petechiae or erosions (graded 3 or 4) in the body of the stomach and six had changes (graded 1-4) in the antrum. No petechiae or erosions were recorded in the oesophagus or the duodenum. In four subjects a layer of clotted blood was found over a large part of the gastric mucosa… Three components of the gastric mucosa were affected by fluoride: the surface epithelium, the gastric pits, and the superficial stroma. The damaged epithelial cells were smaller than undamaged ones, and the vacuoles containing mucus were reduced in size or had disappeared. The most severely damaged epithelium was disrupted or totally lost. The most characteristic changes in the gastric pits were irregular dilation and flattening of the epithelial cells. There was also a noticeable loss of mucin. Our study showed that one ingestion of fluoride at a dose used to treat osteoporosis affects the gastric mucosa… Symptoms like nausea and vomiting are not unusual when fluoride is used to treat osteoporosis. They also occur occasionally when high doses are used for dental prophylaxis. In our study only four subjects developed

nausea, which suggests that using nausea as the first sign of fluoride toxicity might not be valid as all our subjects showed mucosal damage."
SOURCE: Spak CJ, et al. (1989). Tissue response of gastric mucosa after ingestion of fluoride. British Medical Journal 298:1686-87.

H. Paraganglia

"Paranganglia," refers to the groups of chromaffin cells associated with the sympathetic system. "Paraganglia are neuroendocrine organs mainly comprising cells that take their origin in the neural crest. They secrete catecholamines or indolamines and peptides. They are divided into two groups, associated with the sympathetic or parasympathetic nervous systems."[115]

Research specifically evaluating fluoride's effect on paraganglial tissues is not readily available at this time from our search.

I. Pituitary Gland

The pituitary gland is about the size of a pea (0.018 oz) and sits at the base of the brain. The anterior pituitary regulates several physiological processes including stress, growth, reproduction, blood pressure, metabolism, salt/water regulation of kidneys, temperature, pain relief and lactation, while the intermediate lobe synthesizes and secretes melanocyte-stimulating hormone and the posterior lobe is functionally connected to the hypothalamus.

The effects of fluoride pesticides on the pituitary gland are reported at http://www.fluoridealert.org/wp-content/pesticides/effects.endocrine.pituitary.htm

J. Placenta.

The phrase "buyer beware" comes to mind (in a sad guilty way) when searching studies for the effect of fluoride on the placenta, very few exist. In our capitalistic society we expect the buyer, the patient, to be responsible for purchase, use, and safety, especially of fluoride. Apparently we adults expect the fetus to do adequate and quality research on the effects of fluoride on the placenta and themselves, because we adults sure have not. Why have we adults failed to protect the unborn?

In a 1952 issue of *Science* magazine,[116] Harold C. Hodge (chief toxicologist for the US Army's Manhattan Project) reported that women who drank artificially fluoridated water

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[115] Endocrine Pathology:: Differential diagnosis and Molecular Advances. Lloyd RV Editor. Chapter 12, Adrenal Medulla and Paraganglia by McNicol AM.

[116] Gardner DE, Smith FA, Hodge HC, Overton DE, Feltman R. The fluoride concentration of placental tissue as related to fluoride content in drinking water. *Science*. 1952;115(2982):208–209.
See also: Chlubek D, Poreba R, Machalinski B. Fluoride and calcium distribution in human placenta. *Fluoride*. 1998 31(3):131–136.

(1.0–1.2 ppm fluoride) averaged 2.09 ppm fluoride in their placentas, compared with 0.74 ppm fluoride in the placentas of women who drank nonfluoridated water (0.06 ppm fluoride). Maternal blood fluoride levels were also nearly three times higher (0.040 vs. 0.014 ppm).

Tskitishvili (2010)[117] "Oxidative stress with elevated intracellular Ca^{2+} concentration as well as endothelial dysfunction is a component of pre-eclampsia. Our aim was to investigate the oxidative stress-dependent expression of Endoglin and Ca^{2+}-binding S100B protein from villous and amniotic tissue cultures, and to assess sEng expression from S100B protein-stimulated endothelial cells. We initially examined Endoglin and Hydroxy-nonenal-(HNE)-modified proteins in the placentas and amnion obtained from women with pre-eclampsia (n = 8), and healthy controls (n = 8) by immunohistochemistry. To examine oxidative stress and the S100B protein effect on sEng expression from endothelial cells, normal villous and amniotic tissue cultures were stimulated by 4-HNE, sodium fluoride and xanthine/xanthine oxidase, whereas human umbilical vein endothelial cell cultures were treated with S100B protein in a dose- and time-dependent manner at 37°C in an environment of 95% air and 5% of CO_2. Culture supernatants were assessed using ELISA. Cell viability was determined using MTS assay. The concentrations of sEng and S100B protein were significantly increased in the villous and amniotic tissue culture supernatants under oxidative stress. S100B protein-stimulated endothelial cells released sEng into conditioned media with a significantly higher expression levels at a concentration of 200 pM–20 nM S100B by 2 h, whereas treated with 200 nM of S100B endothelial cells significantly expressed sEng by 12 h and stimulated the cell proliferation by the same period of time. Our findings show that oxidative stress affects sEng and S100B protein expression from villous and amniotic tissues, and picomolar and low nanomolar concentrations of S100B protein significantly up-regulate sEng release from endothelial cells leading to endothelial dysfunction."

Dlugosz (2009) "The aim of the study was to investigate the role of oestrogens in free radical detoxication upon exposure to fluoride. Interactions between xenobiotics and oestrogens need to be investigated, especially as many chemicals interact with the oestrogen receptor. It is still unknown whether free radical-generating xenobiotics can influence the antioxidative ability of oestradiol (E(2)). In an in vitro examination of human placental mitochondria, thiobarbituric active reagent species (TBARS), hydroxyl radical ((*)OH) generation and protein thiol (-SH) groups were detected. 17beta-E(2) was examined in physiological (0.15-0.73 nM) and experimental (1-10 microM) concentrations and sodium fluoride (NaF) in concentrations of 6-24 microM. E(2) in all the concentrations significantly decreased lipid peroxidation measured as the TBARS level, in contrast to NaF, which increased lipid peroxidation. Lipid

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Sastry GM, Mohanty S, Rao P. Role of placenta to combat fluorosis (in fetus) in endemic fluorosis area. *Natl J Integr Res Med*. 2010 Oct–Dec;1(4):16–19.

[117] E. Tskitishvili[1,3], N. Sharentuya[1], K. Temma-Asano[1], K. Mimura[1], Y. Kinugasa-Taniguchi[1], T. Kanagawa[1], H. Fukuda[1], T. Kimura[1], T. Tomimatsu[1] and K. Shimoya· Oxidative stress-induced S100B protein from placenta and amnion affects soluble Endoglin release from endothelial cells. Mol Hum Reprod. 2010 Mar;16(3):188-99. doi: 10.1093/molehr/gap104. Epub 2009 Nov 25.

peroxidation induced by NaF was decreased by E(2). The influence of E(2) on (*)OH generation was not very significant and depended on the E(2)concentration. The main mechanism of E(2) protection in NaF exposure appeared to be connected with the influence of E(2)on thiol group levels, not (*)OH scavenging ability. The E(2) in concentrations 0.44-0.73 nM and 1-10 microM significantly increased the levels of -SH groups, in contrast to NaF, which significantly decreased them. E(2) at every concentration reversed the harmful effects of NaF on -SH group levels. No unfavourable interactions in the influence of E(2) and NaF on TBARS production, (*)OH generation, or -SH group levels were observed. The results suggest that postmenopausal women could be more sensitive to NaF-initiated oxidative stress."

Srednicka (2007)[118] "The interactions in free radicals processes between cyclosporine A (CsA) and sodium fluoride (NaF) on in vitro model human placental mitochondria were evaluated. The level of malondialdehyde, hydroxyl radical generation and concentration of sulfhydryl groups of protein was measured. The results showed that CsA with NaF did not give any toxicological interactions with NaF in the area of measured parameters.

Hassunuma (2007)[119] Little information is available on the pathogenesis of fluorosis during the fetal and initial postnatal period. In the present study, female rats received 0 (control), 7 or 100 ppm of sodium fluoride in drinking water, one week before breeding and throughout gestation and nursing periods. The hemimandibles of the offspring were collected at 0, 7 and 14 days of postnatal life (n = 5) and processed for morphological analyses by light and electron microscopy, immunohistochemical analysis for amelogenin and morphometric study of enamel matrix and ameloblasts of incisors. The results showed a decrease in matrix production at the secretory phase at all study periods for the 100 ppm group. In this same group, the secretory ameloblasts showed reduction of enamel matrix secretion, disorganization of mitochondrial crests, large vacuoles at the apical portion of the cytoplasm, retention of intracisternal material and dilatation of some cisterns in the rough endoplasmic reticulum. In the groups of animals aged 7 and 14 days, analysis of variance showed significant reduction ($p<0.05$) in cytoplasmic volume of 23.80% and 24.75%, respectively, in relation to the control group. The smooth-ended maturation ameloblasts exhibited a large number of vacuoles with electron-dense endocytic matrix, suggesting a delay in the resorption process. Immunohistochemical analysis showed no difference in the intensity and labeling pattern of the enamel matrix in any study group. Interestingly, in offspring at the age of 14 days for the 7 ppm group, there was an increase in the matrix length at the secretory phase. Therefore, part of the excessive dose of sodium fluoride given to the mother in drinking water can reach the offspring through the placenta and mother's milk, causing morphological

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[118] Srednicka D1, Długosz A., Interactions in free radicals processes between cyclosporine A and sodium fluoride. Acta Pol Pharm. 2007 Nov-Dec;64(6):503-8.

[119] Hassunuma RM1, Zen Filho EV, Ceolin DS, Cestari TM, Taga R, de Assis GF. Ultrastructural and immunohistochemical study of the influence of fluoride excess on the development of rat incisor tooth buds. J Appl Oral Sci. 2007 Aug;15(4):292-8.

changes in ameloblasts and suggesting a reduction in secretion and a delay in matrix resorption.

Toyama (2001)[120] "This study sought to obtain a precise profile of fluoride concentrations at and near the neonatal line in deciduous incisors and canines from the naturally fluoridated area (1.0--1.3 parts/10(6) F in drinking water) of West Hartlepool and the non-fluoridated area (less than 0.1 parts/10(6) F in drinking water) of Leeds in England. An abrasive microsampling method was used to determine the distribution of fluoride and phosphorus concentrations. The profile of fluoride concentrations in 100-microm layers before and after the neonatal line, that is, in the prenatal and postnatal enamel, were significantly higher in teeth from the fluoridated than non-fluoridated areas. It was concluded that the fact that the fluoride concentrations were about the same prenatally and postnatally in deciduous enamel obtained from the fluoridated and non-fluoridated areas indicates that fluoride enters the prenatal deciduous enamel and that it is transferred through the placenta."

Li (1999)[121] "Whole embryo rotated culture technique was used to investigate the toxicity of combination of selenium, fluoride and arsenic on rat embryos at day 9.5 of gestation. The result of factorial analysis (3 x 3 x 3) showed that the main effect of combination of selenium, fluoride and arsenic on the developmental toxicity was synergistic. The mixtures with different level of these three chemicals in combination could result in different developmental toxicity. The low level combinations mainly caused teratogenic effect, and the high level combinations(selenium 2.0 micrograms + fluoride 10 micrograms + arsenic 1.0 microgram/ml culture media) caused lethal effect. The results suggested that the disorders of yolk-sac placenta in structure and function were one of teratogenic mechanisms for the combination of selenium, fluoride and arsenic."

Flores-Herrera (1999)[122] "This report describes an ATP-diphosphohydrolase activity associated with the inner membrane of human term placental mitochondria. An enriched fraction containing 30 per cent of the total protein and 80 per cent of the total ATP-diphosphohydrolase activity was obtained from submitochondrial particles. ATP-diphosphohydrolase activity was characterized in this fraction. The enzyme had a pH optimum of 8 and catalysed the hydrolysis of triphospho- and diphosphonucleosides other than ATP or ADP. Pyrophosphate was also hydrolysed, but AMP or other monoester phosphates were not. The activity of ATP-diphosphohydrolase was dependent on Mg(2 +), Ca(2 +)or Mn(2 +)and the enzyme substrate was the cation-nucleotide complex. An excess of free cation produced inhibition.ATP-diphosphohydrolase activity was stimulated at micromolar

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[120] Toyama Y1, Nakagaki H, Kato S, Huang S, Mizutani Y, Kojima S, Toyama A, Ohno N, Tsuchiya T, Kirkham J, Robinson C. Fluoride concentrations at and near the neonatal line in human deciduous tooth enamel obtained from a naturally fluoridated and a non-fluoridated area. Arch Oral Biol. 2001 Feb;46(2):147-53.

[121] Li Y1, Sun M, Wu D, Chen X. [The toxicity of combination of selenium, fluoride and arsenic on rat embryos]. Wei Sheng Yan Jiu. 1999 Mar 30;28(2):74-6.

[122] Flores-Herrera O1, Uribe A, Pardo JP, Rendón JL, Martínez F. A novel ATP-diphosphohydrolase from human term placental mitochondria. Placenta. 1999 Jul-Aug;20(5-6):475-84.

concentrations of calcium or magnesium in the presence of La-PPi. Negative cooperativity kinetics was observed with all substrates tested. The V(max)ranged from 150 to 300nmol of Pi released/mg/min. The [S](0.5)for nucleotides was 1-10m m and 182m m for PPi. The enzyme was inhibited by orthovanadate, but not by l -phenylalanine, oligomycin, sodium azide, P(1),P(5)-di(adenosine-5')pentaphosphate or sodium fluoride.The experimental evidence showing absence of inhibition by sodium azide and sodium fluoride, hydrolysis of pyrophosphate but not of monoester phosphates, and negative cooperativity suggested that this enzyme was a novel ATP-diphosphohydrolase."

Yuan (1998)[123] "Most inhibitors of S-adenosylhomocysteine (AdoHcy) hydrolase function as substrates for the "3'-oxidative activity" of the enzyme and convert the enzyme from its active form (NAD+) to its inactive form (NADH) (Liu, S., Wolfe, M. S., and Borchardt, R. T. (1992) Antivir. Res. 19, 247-265). In this study, we describe the effects of a mechanism-based inhibitor, 6'-bromo-5', 6'-didehydro-6'-deoxy-6'-fluorohomoadenosine (BDDFHA), which functions as a substrate for the "6'-hydrolytic activity" of the enzyme with subsequent formation of a covalent linkage with the enzyme. Incubation of human placental AdoHcy hydrolase with BDDFHA results in a maximum inactivation of 83% with the remaining enzyme activity exhibiting one-third of the kcat value of the native enzyme. This partial inactivation is concomitant with the release of both Br- and F- ions and the formation of adenine (Ade). The enzyme can be covalently labeled with [8-3H]BDDFHA, resulting in a stoichiometry of 2 mol of BDDFHA/mol of the tetrameric enzyme. The 3H-labeled enzyme retains its original NAD+/NADH content. Tryptic digestion and subsequent protein sequencing of the [8-3H]BDDFHA-labeled enzyme revealed that Arg196 is the residue that is associated with the radiolabeled inhibitor. The partition ratio of the Ade formation (nonlethal event) to covalent acylation (lethal event) is approximately 1:1. From these experimental results, a possible mechanism by which BDDFHA inactivates AdoHcy hdyrolase is proposed: enzyme-mediated water addition at the C-6' position of BDDFHA followed by elimination of Br- ion results in the formation of homoAdo 6'-carboxyl fluoride (HACF). HACF then partitions in two ways: (a) attack by a proximal nucleophile (Arg196) to form an amide bond after expulsion of F- ion (lethal event) or (b) depurination to form Ade and hexose-derived 6-carboxyl fluoride (HDCF), which is further hydrolyzed to hexose-derived 6-carboxylic acid (HDCA) and F- ion (nonlethal event). . . . Pharmacological modulation of intracellular methylation can be achieved through feedback inhibition of methyltransferase activity by AdoHcy (2). Intracellular AdoHcy concentrations can be elevated by decreasing AdoHcy hydrolase activity (27). Numerous nucleoside analogs capable of reversibly or irreversibly inhibiting AdoHcy hydrolase have been isolated or synthesized as potential antiviral, antiparasitic, antiarthritic, immunosuppresive, and antitumor agents (3-10). More recently, AdoHcy hydrolase inhibitors have been reported to be specially effective against fliovirus such as Ebola virus (28).

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[123] Yuan CS1, Wnuk SF, Robins MJ, Borchardt RT. A novel mechanism-based inhibitor (6'-bromo-5', 6'-didehydro-6'-deoxy-6'-fluorohomoadenosine) that covalently modifies human placental S-adenosylhomocysteine hydrolase. J Biol Chem. 1998 Jul 17;273(29):18191-7.

Tertrin-Clary (1998)[124] "1. Introduction: Protein kinase C (PKC) plays a fundamental role in the regulation of many signal transduction mechanisms activated in response to a variety of stimuli (hormones, growth factors, neurotransmitters). Molecular cloning and biochemical studies have revealed that this kinase consists of a family of at least 12 closely related isoforms classified into four groups based on their primary structure and cofactor requirements. . . . PKC appears to perform a variety of functions in vascular smooth muscle. Many studies have reported that the activation of PKC is associated with vascular smooth muscle contractility and plays a major role in growth-related signal transduction [2]. The feto-placental circulation provides for the metabolic needs of the fetus, and regulation of blood flow in this system is critical for fetal well-being and normal development. Stem villi vessels are considered to be the major sites of fetal placenta vascular resistance [3]. Since the placental vessels lack autonomic innervation, vascular tone is regulated by locally or humorally delivered vasoactive substances [4]. Endothelin-1 (ET-1), a 21 amino acid peptide, is a potent vasoactive agent that acts on the contractility of placental vessels [5]. Several studies have reported that activation of PKC may be a component of the signal cascade resulting in the effects of this peptide on contractility and cell division in vascular smooth muscles, such as rat cardiomyocytes [6-8], bovine cerebral arteries [9], human and rat renal artery [10,11], rat aorta [12] and the rat portal vein [13]. Specific high affinity binding sites for ET-1 have been described in the muscular layer of stem villi vessels [14], and Mondon et al. [15] demonstrated that these ET-1 vascular binding sites are coupled to a phosphoinositide-specific phospholipase C pathway that generates two intracellular messengers, DAG and CaP^{2+}, that are activators of PKC.

The objective of this study was to examine the presence of PKC activity in the muscular layer of human placental stem villi vessels. . . .

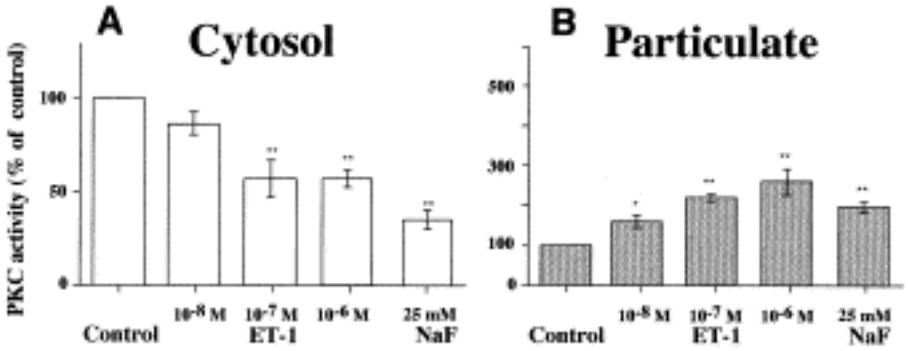

Fig. 1.
Chromatography of cytosolic and particulate-associated protein kinase C from human placental stem villi vessels on a DEAE-cellulose column. PKC activity in the eluted fractions was assayed as described in Section 2and is expressed cpm: (•) in the

[124] Tertrin-Clary C, Fournier T., Ferreè F. Regulation of protein kinase C in the muscular layer of human placental stem villi vessels. FEBS Lett. 1998 Jan 23;422(1):123-8.

presence of Ca^{2+}, phosphatidylserine and diolein, (○) in the presence of EGTA, without phosphatidylserine or diolein. Results are representative of three experiments."

Montherrat-Carret (1996)[125] "To evaluate the beneficial effect of prenatal fluoride supplementation, the presence of fluoride in hard tissues in two populations of human foetuses coming from fluoridated (> or = 0.7 parts/10(6) F in drinking water) and non-fluoridated areas (< or = 0.1 parts/10(6) F in drinking water) were compared by chemical analysis and X-ray microanalysis. The fluoride concentrations measured in maternal and venous cord blood confirmed that placental transfer of fluoride was passive when fluoride intake was low. Total fluoride contents of tooth germs and mandibular bone appeared to increase with fluoride level in drinking water. However, these concentrations were too low to be detected by X-ray microanalysis. Phosphorus and calcium total contents were identical in mandibular and femoral bone of both populations. In incisor germs, phosphorus and calcium concentrations in enamel and dentine close to the amelodentinal junction did not differ significantly between the two populations. It is suggested that the low fluoride concentrations in enamel and dentine formed in utero would not have a significant effect on acid solubility."

Anand (1996)[126] "Active glycine transport was demonstrated in microvillous (maternal-facing, BBM) and basal (fetal-facing, BCM) plasma membranes of the human term placental syncytiotrophoblast. . . Nicotine, insulin, sodium fluoride and sodium arsenate were inhibitors for both the vesicles."

Gupta (1993)[127] "Transplacental passage of fluorides was studied in 25 randomly selected neonates. Blood samples collected simultaneously from the mother and the umbilical cord showed that average fluoride concentration in the cord blood was 60% of that in mother's blood. When concentration in the mother's blood exceeded 0.4 ppm, the placenta acted as a selective barrier.

Malhotra (1993)[128] "The study was conducted on 25 healthy women residing in optimum fluoride areas, who were to deliver normally through vaginal route, to correlate the maternal and cord plasma fluoride levels and evaluate the placental transfer of fluoride. A wide variation was found in the maternal and cord plasma fluoride levels. In only 8 percent of the cases the fluoride levels in cord plasma were higher than maternal plasma. It was deduced that the placenta allows passive diffusion of fluoride from mother to foetus and does not act as a barrier."

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[125]Montherrat-Carret L1, Perrat-Mabilon B, Barbey E, Bouloc R, Boivin G, Michelet A, Magloire H. Chemical and X-ray analysis of fluoride, phosphorus, and calcium in human foetal blood and hard tissues. Arch Oral Biol. 1996 Dec;41(12):1169-78.

[126] Anand RJ1, Kanwar U, Sanyal SN. Transport of glycine in the brush border and basal cell membrane vesicles of the human term placenta. Biochem Mol Biol Int. 1996 Feb;38(1):21-30.

[127] Gupta S1, Seth AK, Gupta A, Gavane AG. Transplacental passage of fluorides. J Pediatr. 1993 Jul;123(1):139-41.

[128] Malhotra A1, Tewari A, Chawla HS, Gauba K, Dhall K. Placental transfer of fluoride in pregnant women consuming optimum fluoride in drinking water. J Indian Soc Pedod Prev Dent. 1993 Mar;11(1):1-3.

Vinals (1993)[129] "Fluoride is a nucleophilic reagent which has been reported to inhibit a variety of different enzymes such as esterases, asymmetrical hydrolases and phosphatases. In this report, we demonstrate that fluoride inhibits tyrosine kinase activity of insulin receptors partially purified from rat skeletal muscle and human placenta. . . . These data suggest: (i) that fluoride interacts directly and slowly with the insulin receptor, which causes inhibition of its phosphotransferase activity; (ii) that the binding site of fluoride is not structurally modified by receptor phosphorylation; and (iii) based on the fact that fluoride inhibits phosphotransferase activity in the absence of alterations in the binding of ATP, Mn2+ or insulin, we speculate that fluoride binding might affect the transfer of phosphate from ATP to the tyrosine residues of the beta-subunit of the insulin receptor and to the tyrosine residues of exogenous substrates."

The NRC (2006)[130] concluded in part: "The effects of fluoride on various aspects of endocrine function should be examined further, particularly with respect to a possible role in the development of several diseases or mental states in the United States. Major areas for investigation include the following: . . . thyroid disease (especially in light of decreasing iodine intake by the U.S. population). . . ."

[129]Viñals F1, Testar X, Palacín M, Zorzano A. Inhibitory effect of fluoride on insulin receptor autophosphorylation and tyrosine kinase activity. Biochem J. 1993 Apr 15;291 (Pt 2):615-22.
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1132568/

[130] "Fluoride in Drinking Water: A Scientific Review of EPA's Standards."
http://www.nap.edu/catalog/11571/fluoride-in-drinking-water-a-scientific-review-of-epas-standards

V. NRC (2006) REPORT ON THE ENDOCRINE SYSTEM

The following 9 pages are directly from pages 224-236 of the NRC's report's "Fluoride in Drinking Water: A Scientific Review of EPA's Standards."

"Effects on the Endocrine System

The endocrine system, apart from reproductive aspects, was not considered in detail in recent major reviews of the health effects of fluoride (PHS 1991; NRC 1993; Locker 1999; McDonagh et al. 2000a; WHO 2002; ATSDR 2003). Both the Public Health Service (PHS 1991) and the World Health Organization (WHO 2002) mentioned secondary hyperparathyroidism in connection with discussions of skeletal fluorosis, but neither report examined endocrine effects any further. The Agency for Toxic Substances and Disease Registry (ATSDR 2003) discussed four papers on thyroid effects and two papers on parathyroid effects and concluded that "there are some data to suggest that fluoride does adversely affect some endocrine glands." McDonagh et al. (2000a) reviewed a number of human studies of fluoride effects, including three that dealt with goiter and one that dealt with age at menarche. The following section reviews material on the effects of fluoride on the endocrine system—in particular, the thyroid (both follicular cells and parafollicular cells), parathyroid, and pineal glands. Each of these sections has its own discussion section. Detailed information about study designs, exposure conditions, and results is provided in Appendix E.

The follicular cells of the thyroid gland produce the classic thyroid hormones thyroxine (T4) and triiodothyronine (T3); these hormones modulate a variety of physiological processes, including but not limited to normal growth and development (Larsen et al. 2002; Larsen and Davies 2002; Goodman 2003). Between 4% and 5% of the U.S. population may be affected by deranged thyroid function (Goodman 2003), making it among the most prevalent of endocrine diseases (Larsen et al. 2002). The prevalence of subclinical thyroid dysfunction in various populations is 1.3-17.5% for subclinical hypothyroidism and 0.6-16% for subclinical hyperthyroidism; the reported rates depend on age, sex, iodine intake, sensitivity of measurements, and definition used (Biondi et

al. 2002). Normal thyroid function requires sufficient intake of iodine (at least 100 micrograms/day [µg/d]), and areas of endemic iodine deficiency are associated with disorders such as endemic goiter and cretinism (Larsen et al. 2002; Larsen and Davies 2002; Goodman 2003). Iodine intake in the United States (where iodine is added to table salt) is decreasing (CDC 2002d; Larsen et al. 2002), and an estimated 12% of the population has low concentrations of urinary iodine (Larsen et al. 2002).

The principal regulator of thyroid function is the pituitary hormone thyroid-stimulating hormone (TSH), which in turn is controlled by positive input from the hypothalamic hormone thyrotropin-releasing hormone (TRH) and by negative input from T4 and T3. TSH binds to G-protein-coupled receptors in the surface membranes of thyroid follicular cells (Goodman 2003), which leads to increases in both the cyclic adenosine monophosphate (cAMP) and diacylglycerol/inositol trisphosphate second messenger pathways (Goodman 2003). T3, rather than T4, probably is responsible for the feedback response for TSH production (Schneider et al. 2001). Some T3, the active form of thyroid hormone, is secreted directly by the thyroid along with T4, but most T3 is produced from T4 by one of two deiodinases (Types I and II1) in the peripheral tissue (Schneider et al. 2001; Larsen et al. 2002; Goodman 2003). T3 enters the nucleus of the target cells and binds to specific receptors, which activate specific genes.

Background

An effect of fluoride exposure on the thyroid was first reported approximately 150 years ago (Maumené 1854, 1866; as cited in various reports). In 1923, the director of the Idaho Public Health Service, in a letter to the Surgeon General, reported enlarged thyroids in many children between the ages of 12 and 15 using city water in the village of Oakley, Idaho (Almond 1923); in addition, the children using city water had severe enamel deficiencies in their permanent teeth. The dental problems were eventually attributed to the presence in the city water of 6 mg/L fluoride, and children born after a change in water supply (to water with <0.5 mg/L fluoride) were not so affected (McKay 1933); however, there seems to have been no further report on thyroid conditions in the village.

More recently, Demole (1970) argued that a specific toxicity of fluoride for the thyroid gland does not exist, because (1) fluoride does not accumulate in the thyroid; (2) fluoride does not affect the uptake of iodine by thyroid tissue; (3) pathologic changes in the thyroid show no increased frequency in regions where water is fluoridated (naturally or artificially); (4) administration of fluoride does not interfere with the prophylactic action of iodine on endemic goiter; and (5) the beneficial effect of iodine in threshold dosage to experimental animals is not inhibited by administration of fluoride, even in excessive amounts. Bürgi et al. (1984) also stated that fluoride does not potentiate the consequences of iodine deficiency in populations with a borderline or low iodine intake and that published data fail to support the hypothesis that fluoride has adverse effects on the thyroid (at doses recommended for caries prevention). McLaren (1976), however, pointed out the complexity of the system, the difficulties in making adequate comparisons of the various studies of fluoride and the thyroid, and evidence for fluoride accumulation in the thyroid and morphological and functional changes (e.g., changes in activity of adenylyl cyclase), suggesting that analytical methods could have limited the

definitiveness of the data to date. His review suggested that physiological or functional changes might occur at fluoride intakes of 5 mg/day.

Although fluoride does not accumulate significantly in most soft tissue (as compared to bones and teeth), several older studies found that fluoride concentrations in thyroid tissue generally exceed those in most other tissue except kidney (e.g., Chang et al. 1934; Hein et al. 1954, 1956); more recent information with improved analytic methods for fluoride was not located. Several studies have reported no effect of fluoride treatment on thyroid weight or morphology (Gedalia et al. 1960; Stolc and Podoba 1960; Saka et al. 1965; Bobek et al. 1976; Hara 1980), while others have reported such morphological changes as mild atrophy of the follicular epithelium (Ogilvie 1953), distended endoplasmic reticulum in follicular cells (Sundström 1971), and "morphological changes suggesting hormonal hypofunction" (Jonderko et al. 1983).

Fluoride was once thought to compete with iodide for transport into the thyroid, but several studies have demonstrated that this does not occur (Harris and Hayes 1955; Levi and Silberstein 1955; Anbar et al. 1959; Saka et al. 1965). The iodide transporter accepts other negatively charged ions besides iodide (e.g., perchlorate), but they are about the same size as iodide (Anbar et al. 1959); fluoride ion is considerably smaller and does not appear to displace iodide in the transporter.

Animal Studies

A number of studies have examined the effects of fluoride on thyroid function in experimental animals or livestock (for details, see Appendix E, Tables E-1, E-2, and E-3). Of these, the most informative are those that have considered both the fluoride and iodine intakes.

Guan et al. (1988) found that a fluoride intake of 10 mg/L in drinking water had little apparent effect on Wistar rats with sufficient iodine intake, but a fluoride intake of 30 mg/L in drinking water resulted in significant decreases in thyroid function (decreases in T4, T3, thyroid peroxidase, and 3H-leucine), as well as a decrease in thyroid weight and effects on thyroid morphology (Table E-2). In iodine-deficient rats, fluoride intake of 10 mg/L in drinking water produced abnormalities in thyroid function beyond that attributable to low iodine, including decreased thyroid peroxidase, and low T4 without compensatory transformation of T4 to T3.

Zhao et al. (1998), using male Kunmin mice, found that both iodine-deficient and iodine-excess conditions produced goiters, but, under iodine-deficient conditions, the goiter incidence at 100 days increased with increased intake of fluoride. At 100 days, the high-fluoride groups had elevated serum T4 at all concentrations of iodine intake and elevated T3 in iodine-deficient animals. High fluoride intake significantly inhibited the radioiodine uptake in the low- and normal-iodine groups.

Stolc and Podoba (1960) found a decrease in protein-bound iodine in blood in fluoride-treated female rats (3-4 mg/kg/day) fed a low-iodine diet but not in corresponding rats fed a larger amount of iodine. Both groups (low- and high-iodine) of fluoride-treated rats showed a reduced rate of biogenesis of T3 and T4 after administration of 131I compared with controls (Stolc and Podoba 1960).

Bobek et al. (1976) found decreases in plasma T4 and T3 as well as a decrease in free T4 index and an increase in T3-resin uptake in male rats given 0.1 or 1 mg of fluoride per day (0.4-0.6 or 4-6 mg/kg/day) in drinking water for 60 days.2 The authors suggested the possibility of decreased binding capabilities and altered thyroid hormone transport in blood.

Decreases in T4 and T3 concentrations have been reported in dairy cows at estimated fluoride doses up to 0.7 mg/kg/day with possible iodine deficiency (Hillman et al. 1979; Table E-3). Reduced T3 (Swarup et al. 1998) and reduced T3, T4, and protein-bound iodine (Cinar and Selcuk 2005) have also been reported in cows diagnosed with chronic fluorosis in India and Turkey, respectively.

Hara (1980) found elevated T3 and T4 at the lowest dose (approximately 0.1 mg/kg/day), decreased T3 and normal T4 at intermediate doses (3-4 mg/kg/day), and decreased TSH and growth hormone (indicating possible effects on pituitary function) at the highest doses (10-20 mg/kg/day). This was the only animal study of fluoride effects on thyroid function to measure TSH concentrations; however, full details (e.g., iodine intake) are not available in English.

Other studies have shown no effect of fluoride on the end points examined (Gedalia et al. 1960; Siebenhüner et al. 1984; Clay and Suttie 1987; Choubisa 1999; Table E-1). Choubisa (1999) looked only for clinical evidence of goiter in domestic animals (cattle and buffaloes) showing signs of enamel or skeletal fluorosis; no hormone parameters (e.g., T4, T3, TSH) were measured. Gedalia et al. (1960) also did not measure T4, T3, or TSH; radioiodine uptake, protein-bound iodine, and total blood iodine were all normal in rats receiving fluoride doses up to approximately 1 milligram per kilogram of body weight per day (mg/kg/day). Clay and Suttie (1987) reported no significant differences from control values for T4 concentration and T3 uptake in heifers fed up to 1.4 mg/kg/day; iodine intake is not stated but probably was adequate, and TSH was not measured.

Siebenhüner et al. (1984) carried out a special experiment involving iodine depletion of the thyroid before 6 days of fluoride treatment. No effects were seen on the parameters measured, including T3 and T4 concentrations; however, TSH was not measured. In addition, propylthiouracil (PTU), the agent used to deplete the thyroid of iodine, also has an inhibitory effect on deiodinases (Larsen et al. 2002; Larsen and Davies 2002); Siebenhüner et al. (1984) did not mention this second action of PTU and its relevance to the interpretation of the experimental results, and there was no control group without the PTU treatment.

Human Studies

Several authors have reported an association between endemic goiter and fluoride exposure or enamel fluorosis in human populations in India (Wilson 1941; Siddiqui 1960; Desai et al. 1993), Nepal (Day and Powell-Jackson 1972), England (Wilson 1941; Murray et al. 1948), South Africa (Steyn 1948; Steyn et al. 1955; Jooste et al. 1999), and Kenya (Obel 1982). Although endemic goiter is now generally attributed to iodine deficiency (Murray et al. 1948; Obel 1982; Larsen et al. 2002; Belchetz and Hammond 2003), some of the goitrogenic areas associated with fluoride exposure were not considered to be iodine deficient (Steyn 1948; Steyn et al. 1955; Obel 1982; Jooste et

al. 1999). Obel (1982) indicated that many cases of fluorosis in Kenya occur concurrently with goiter. Several authors raise the possibility that the goitrous effect, if not due to fluoride, is due to some other substance in the water (e.g., calcium or water hardness) that was associated with the fluoride concentration (Murray et al. 1948; Day and Powell-Jackson 1972) or that enhanced the effect of fluoride (Steyn 1948; Steyn et al. 1955). Dietary selenium deficiencies (e.g., endemic in parts of China and Africa or due to protein-restricted diets) can also affect normal thyroid function3 (Larsen et al. 2002); no information on dietary selenium is available in any of the fluoride studies. Appendix E summarizes a number of studies of the effects of fluoride on thyroid function in humans (see Table E-4).

Three studies illustrated the range of results that have been reported: (1) Gedalia and Brand (1963) found an association between endemic goiter in Israeli girls and iodine concentrations in water but found no association with fluoride concentrations (<0.1-0.9 mg/L). (2) Siddiqui (1960) found goiters only in persons aged 14-17 years; the goiters, which became less visible or invisible after puberty, were associated with mean fluorine content of the water (5.4-10.7 mg/L) and were inversely associated with mean iodine content of the water. (3) Desai et al. (1993) found a positive correlation ($P < 0.001$) between prevalence of goiter (9.5-37.5%) and enamel fluorosis (6.0-59.0%), but no correlation between prevalence of goiter and water iodine concentration ($P > 0.05$).

Day and Powell Jackson (1972) surveyed 13 villages in Nepal where the water supply was uniformly low in iodine (?1 µg/L; see Figure 8-1). Here the goiter prevalence (5-69%, all age groups) was directly associated with the fluoride concentration (<0.1 to 0.36 mg/L; $P < 0.01$) or with hardness, calcium concentration, or magnesium concentration of the water (all $P < 0.01$). Goiter prevalence of at least 20% was associated with all fluoride concentrations ? 0.19 mg/L, suggesting that fluoride might influence the prevalence of goiter in an area where goiter is endemic because of low iodine intake. The possibility of a nutritional component (undernutrition or protein deficiency) to the development of goiter was also suggested.

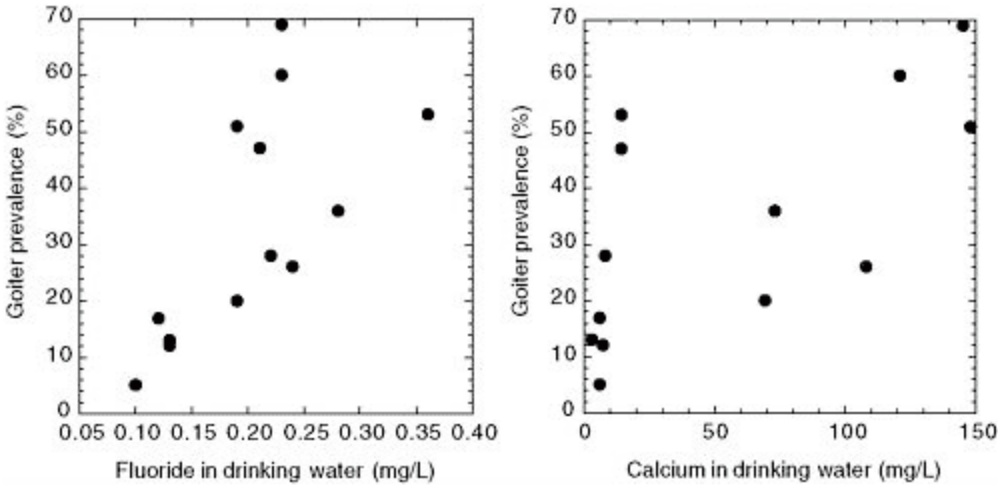
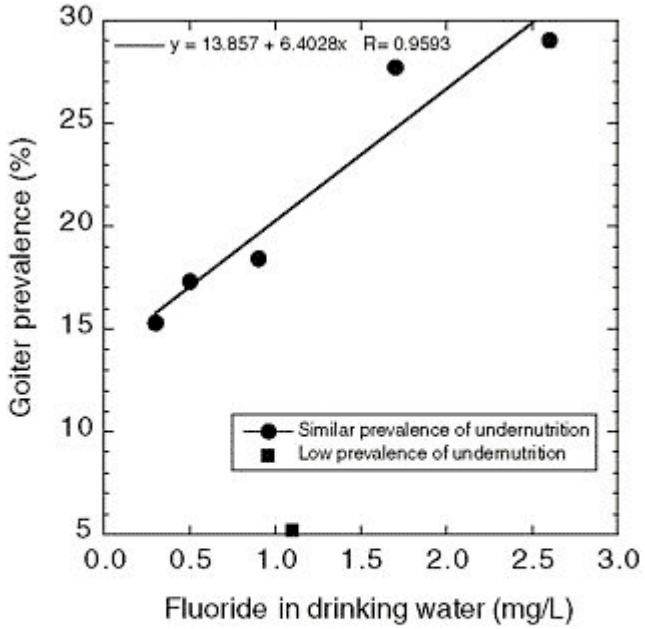


FIGURE 8-1 Goiter prevalence versus fluoride (left) and calcium (right) concentration in drinking water for 13 villages in Nepal with very low iodine concentrations. SOURCE: Day and Powell-Jackson 1972.

Jooste et al. (1999) examined children (ages 6, 12, and 15) who had spent their entire lives in one of six towns in South Africa where iodine concentrations in drinking water were considered adequate (median urinary iodine concentration exceeding 201 µg/L [1.58 µmol/L]; see Appendix E, Tables E-4 and E-5; Figure 8-2). For towns with low (0.3-0.5 mg/L) or near "optimal" (0.9-1.1 mg/L) fluoride concentrations in water, no relationship between fluoride and prevalence of mild goiter was found (5-18%); for the other two towns (1.7 and 2.6 mg/L fluoride), however, goiter prevalences were 28% and 29%, respectively, and most children had severe enamel mottling. These two towns (and one low-fluoride town) had very low proportions (0-2.2%) of children with iodine deficiency, defined as urinary iodine concentrations <100 µg/L (<0.79 µmol/L). The town with the lowest prevalence of goiter also had the lowest prevalence of under-nutrition; the two towns with the highest prevalence of goiter (and highest fluoride concentrations) did not differ greatly from the remaining three towns with respect to prevalence of under-nutrition. The authors suggested that fluoride or an associated goitrogen might be responsible for the goiters seen in the two towns with the highest fluoride concentrations but that some other factor(s) was involved in development of goiter in the other towns.



Several studies have compared various aspects of thyroid status in populations with different fluoride intakes (for details, see Appendix E, Table E-4). Leone et al. (1964) and Baum et al. (1981) reported no significant differences in thyroid status between populations with low (0.09-0.2 mg/L) and high (3-3.5 mg/L) fluoride concentrations

in the drinking water. Leone et al. (1964) looked only at protein-bound iodine and physical examination of the thyroid in adults; Baum et al. (1981) measured a number of parameters in teenagers, including T4, T3, and TSH. Neither study reported iodine status of the groups. Baum et al. (1981) showed but did not explain a decrease in thyroglobulin in girls in the high-fluoride group.

Bachinskii et al. (1985) examined 47 healthy persons, 43 persons with hyperthyroidism, and 33 persons with hypothyroidism. Prolonged consumption of “high-fluoride” drinking water (2.3 mg/L, as opposed to “normal” concentrations of 1 mg/L) by healthy persons was associated with statistically significant changes in TSH concentrations (increased), T3 concentrations (decreased), and uptake of radioiodine (increased), although the mean values for TSH and T3 were still within normal ranges (see Appendix E, Table E-6). The mean value of TSH for the healthy group (4.3 ± 0.6 milliunits/L; Table E-6) is high enough that one expects a few individuals to have been above the normal range (typically 0.5-5 milliunits/L; Larsen et al. 2002). These results were interpreted as indicating disruption of iodine metabolism, stress in the pituitary-thyroid system, and increased risk of developing thyroidopathy (Bachinskii et al. 1985).

Lin et al. (1991) examined 769 children (7-14 years old) for mental retardation in three areas of China, including an area with “high” fluoride (0.88 mg/L) and low iodine, an area with “normal” fluoride (0.34 mg/L) and low iodine, and an area where iodine supplementation was routine (fluoride concentration not stated). Ten to twelve children in each area received detailed examinations, including measuring thyroid 131I uptake and thyroid hormone concentrations. Children in the first area had higher TSH, slightly higher 131I uptake, and lower mean IQ than children in the second area. Children in the first area also had reduced T3 and elevated reverse T3, compared with children in the second area. The authors suggested that high fluoride might exacerbate the effects of iodine deficiency. In addition, the authors reported a difference in T3/rT3 (T3/reverse-T3) ratios between high- and low-fluoride areas and suggested that excess fluoride ion affects normal deiodination.

A recent study by Susheela et al. (2005) compared thyroid hormone status (free T4, free T3, and TSH) of 90 children with enamel fluorosis (drinking water fluoride ranging from 1.1 to 14.3 mg/L) and 21 children without enamel fluorosis (0.14-0.81 mg/L fluoride in drinking water) in areas where iodine supplementation was considered adequate.4 Forty-nine children (54.4%) in the sample group had “well-defined hormonal derangements”; findings were borderline in the remaining 41 children. The types of hormonal derangements included elevated TSH and normal T4 and T3 (subclinical hypothyroidism); low T3 and normal T4 and TSH (“low T3 syndrome”); elevated T3 and TSH and normal T4 (possible T3 toxicosis); elevated TSH, low T4, and normal T3 (usually indicative of primary hypothyroidism and iodine deficiency); and low T3, high TSH, and normal T4. All but the first category are considered to be associated with or potentially caused by abnormal activity of deiodinases. The authors concluded that fluoride in excess may be inducing diseases that have usually been attributed to iodine deficiency and that iodine supplementation may not be adequate when excess fluoride is being consumed.

Thyroid hormone disturbances were also noted in the control children, and urine and fluoride concentrations in the control children reflect higher fluoride intake than can be

accounted for by the drinking water alone (Susheela et al. 2005). Thus, the authors recommend that end points such as hormone concentrations should be examined with respect to serum or urinary fluoride concentrations, not just drinking water fluoride concentrations. In addition, they note that all hormone endpoints (T3, T4, and TSH) should be examined, lest some of the abnormalities be missed.

Mikhailets et al. (1996) detected thyroid abnormalities (moderate reduction of iodine uptake, low T3, normal T4, and increased TSH) in 165 aluminum workers with signs of chronic fluorosis and an estimated average fluoride intake of 10 mg/working day. A tendency toward increased TSH was observed with increased exposure time and with more severe fluorosis. Workers with more than 10 years of service had a significant decrease in T3 concentration in comparison to controls. The frequency of individuals with low concentrations of T3 (corresponding to hypothyroidism) was 65% among workers with more than 10 years of service and 54% among workers with Stage 2 fluorosis. The highest frequency of occurrence of low T3 (76%) was observed in people with chronic fluoride intoxication including liver damage (moderate cytolysis), suggesting a disorder in peripheral conversion of T4 to T3 (deiodination). The possibility of indirect effects of fluorine on enzymatic deiodination was also suggested.

Tokar? et al. (1989) and Balabolkin et al. (1995) have also reported thyroid effects in fluoride- or fluorine-exposed workers; full details of these studies are not available in English. Balabolkin et al. (1995) found that 51% of the workers examined had subclinical hypothyroidism with reduced T3.

No changes in thyroid function were detected in two studies of osteoporosis patients treated with NaF for 6 months or several years (Eichner et al. 1981; Hasling et al. 1987; for details, see Appendix E, Table E-7). These study populations are not necessarily representative of the general population, especially with respect to age and the fact that they usually receive calcium supplements. In an earlier clinical study to examine the reported effects of fluoride on individuals with hyperthyroidism, Galletti and Joyet (1958) found that, in 6 of 15 patients, both basal metabolic rate and protein-bound iodine fell to normal concentrations, and the symptoms of hyperthyroidism were relieved after fluoride treatment. Fluoride was considered clinically ineffective in the other 9 patients, although improvement in basal metabolic rate or protein-bound iodine was observed in some of them. In the 6 patients for whom fluoride was effective, tachycardia and tremor disappeared within 4-8 weeks, and weight loss was stopped. The greatest clinical improvement was observed in women between 40 and 60 years old with a moderate degree of thyrotoxicosis; young patients with the classic symptoms of Graves' disease did not respond to fluoride therapy. Radioiodine uptake tests were performed on 10 of the patients, 7 of whom showed an inhibitory effect on initial 131I uptake by the thyroid.

Discussion (Effects on Thyroid Function)

In studies of animals with dietary iodine sufficiency, effects on thyroid function were seen at fluoride doses of 3-6 mg/kg/day (Stolc and Podoba 1960; Bobek et al. 1976; Guan et al. 1988; Zhao et al. 1998); in one study, effects were seen at doses as low as 0.4-0.6 mg/kg/day (Bobek et al. 1976). In low-iodine situations, more severe effects on thyroid function were seen at these doses (Stolc and Podoba 1960; Guan et al. 1988; Zhao et al. 1998). Effects on thyroid function in low-iodine situations have also been noted at fluoride doses as low as 0.06 mg/kg/day (Zhao et al. 1998), ?0.7 mg/kg/day

(Hillman et al. 1979), and 1 mg/kg/day (Guan et al. 1988). Studies showing no effect of fluoride on thyroid function did not measure actual hormone concentrations (Gedalia et al. 1960; Choubisa 1999), did not report iodine intakes (Gedalia et al. 1960; Clay and Suttie 1987; Choubisa 1999), used fluoride doses (<1.5 mg/kg/day) below those (3-6 mg/kg/day) associated with effects in other studies (Gedalia et al. 1960; Clay and Suttie 1987), or did not discuss a possibly complicating factor of the experimental procedure used (Siebenhüner et al. 1984). Only one animal study (Hara 1980) measured TSH concentrations, although that is considered a “precise and specific barometer” of thyroid status in most situations (Larsen et al. 2002). Full details of Hara’s report are not available in English.

Goiter prevalence of at least 20% has been reported in humans exposed to water fluoride concentrations ? 0.2 mg/L (low-iodine situation; Day and Powell-Jackson 1972) or 1.5-3 mg/L (undernutrition, but adequate iodine; Jooste et al. 1999); however, other causes of goiter have not been ruled out. Bachinskii et al. (1985) showed increased TSH concentrations and reduced T3 concentrations in a population with a fluoride concentration of 2.3 mg/L in their drinking water (in comparison to a group with 1.0 mg/L), and Lin et al. (1991) showed similar results for a population with 0.88 mg/L fluoride in the drinking water (in comparison to a group with 0.34 mg/L); another study showed no effect at 3 mg/L (Baum et al. 1981). Among children considered to have adequate iodine supplementation, Susheela et al. (2005) found derangements of thyroid hormones in 54% of children with enamel fluorosis (1.1-14.3 mg/L fluoride in drinking water), and in 45-50% of “control” children without enamel fluorosis but with elevated serum fluoride concentrations. Mikhailets et al. (1996) observed an increase in TSH in workers with increased exposure time and with more severe fluorosis; low T3 was found in 65% of workers with more than 10 years of service and in 54% of workers with Stage 2 fluorosis. Several studies do not include measurements of T4, T3, or TSH (Siddiqui 1960; Gedalia and Brand 1963; Leone et al. 1964; Day and Powell-Jackson 1972; Teotia et al. 1978; Desai et al. 1993; Jooste et al. 1999).

Nutritional information (especially the adequacy of iodine and selenium intake) is lacking for many (iodine) or all (selenium) of the available studies on humans. As with the animal studies, high fluoride intake appears to exacerbate the effects of low iodine concentrations (Day and Powell-Jackson 1972; Lin et al. 1991). Uncertainty about total fluoride exposures based on water fluoride concentrations, variability in exposures within population groups, and variability in response among individuals generally have not been addressed. Although no thyroid effects were reported in studies using controlled doses of fluoride for osteoporosis therapy, the study populations are not necessarily representative of the general population with respect to age, calcium intake, and the presence of metabolic bone disease.

Thus, several lines of information indicate an effect of fluoride exposure on thyroid function. However, because of the complexity of interpretation of various parameters of thyroid function (Larsen et al. 2002), the possibility of peripheral effects on thyroid function instead of or in addition to direct effects on the thyroid, the absence of TSH measurements in most of the animal studies, the difficulties of exposure estimation in human studies, and the lack of information in most studies on nutritional factors (iodine, selenium) that are known to affect thyroid function, it is difficult to predict exactly what

effects on thyroid function are likely at what concentration of fluoride exposure and under what circumstances.

Suggested mechanisms of action for the results reported to date include decreased production of thyroid hormone, effects on thyroid hormone transport in blood, and effects on peripheral conversion of T4 to T3 or on normal deiodination processes, but details remain uncertain. Both peripheral conversion of T4 to T3 and normal deiodination (deactivation) processes require the deiodinases (Types I and II for converting T4 to T3 and Types I and III for deactivation; Schneider et al. 2001; Larsen et al. 2002; Goodman 2003). Several sets of reported results are consistent with an inhibiting effect of fluoride on deiodinase activity; these effects include decreased plasma T3 with normal or elevated T4 and TSH and normal T3 with elevated T4 (Bachinskii et al. 1985; Guan et al. 1988; Lin et al. 1991; Balabolkin et al. 1995; Michael et al. 1996; Mikhailets et al. 1996; Susheela et al. 2005). The antihyperthyroid effect that Galletti and Joyet (1958) observed in some patients is also consistent with an inhibition of deiodinase activity in those individuals.

The available studies have generally dealt with mean values of various parameters for the study groups, rather than with indications of the clinical significance, such as the fraction of individuals with a value (e.g., TSH concentration) outside the normal range or with clinical thyroid disease. For example, in the two populations of asymptomatic individuals compared by Bachinskii et al. (1985), the elevated mean TSH value in the higher-fluoride group is still within the normal range, but the number of individuals in that group with TSH values above the normal range is not given.

In the absence of specific information in the reports, it cannot be assumed that all individuals with elevated TSH or altered thyroid hormone concentrations were asymptomatic, although many might have been. For asymptomatic individuals, the significance of elevated TSH or altered thyroid hormone concentrations is not clear. Belchetz and Hammond (2003) point out that the population-derived reference standards (e.g., for T4 and TSH) reflect the mean plus or minus two standard deviations, meaning that 5% of normal people have results outside a given range. At the same time, healthy individuals might regulate plasma T4 within a “personal band” that could be much more narrow than the reference range; this brings up the question of whether a disorder shifting hormone values outside the personal band but within the population reference range requires treatment (Davies and Larsen 2002; Belchetz and Hammond 2003). For example, early hypothyroidism can present with symptoms and raised TSH but with T4 concentrations still within the reference range (Larsen et al. 2002; Belchetz and Hammond 2003).

Subclinical hypothyroidism is considered a strong risk factor for later development of overt hypothyroidism (Weetman 1997; Helfand 2004). Biondi et al. (2002) associate subclinical thyroid dysfunction (either hypo or hyperthyroidism) with changes in cardiac function and corresponding increased risks of heart disease. Subclinical hyperthyroidism can cause bone demineralization, especially in postmenopausal women, while subclinical hypothyroidism is associated with increased cholesterol concentrations, increased incidence of depression, diminished response to standard psychiatric treatment, cognitive dysfunction, and, in pregnant women, decreased IQ of their offspring (Gold et al. 1981; Brucker-Davis et al. 2001). Klein et al. (2001) report an

inverse correlation between severity of maternal hypothyroidism (subclinical or asymptomatic) and the IQ of the offspring (see also Chapter 7).

A number of authors have reported delayed eruption of teeth, enamel defects, or both, in cases of congenital or juvenile hypothyroidism (Hinrichs 1966; Silverman 1971; Biggerstaff and Rose 1979; Noren and Alm 1983; Loevy et al. 1987; Bhat and Nelson 1989; Mg'ang'a and Chindia 1990; Pirinen 1995; Larsen and Davies 2002; Hirayama et al. 2003; Ionescu et al. 2004). No information was located on enamel defects or effects on eruption of teeth in children with either mild or subclinical hypothyroidism. The possibility that either dental fluorosis (Chapter 4) or the delayed tooth eruption noted with high fluoride intake (Chapter 4; see also Short 1944) may be attributable at least in part to an effect of fluoride on thyroid function has not been studied." (End quote of NRC)

VI. FLUORIDE, IODINE AND GOITER[131]

A reasonably consistent body of animal and human research shows that fluoride exposure worsens the impact of iodine deficiency. (Gas'kov 2005; Hong 2001; Wang

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131 Additional References

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2001; Zhao 1998; Xu 1994; Lin 1991; Ren 1989; Guan 1988).[132] Iodine is needed for T3 and T4 hormone production and thus an adequate iodine intake is considered important for the proper thyroid function.

Researchers report an iodine deficiency coupled with fluoride exposure produces a more damaging effect on neurological development than iodine deficiency alone. (Hong 2001; Xu 1994; Lin 1991; Ren 1989).[133] The studies, which utilize childhood intelligence as the metric for assessing neurological health, have found that fluoride levels in water as low as 0.9 ppm can worsen the IQ effect of iodine deficiency. (Lin 1991).[134] Studies have reported an association between fluoride and reduced IQ among children with adequate iodine intake, (Choi 2012),[135] and iodine deficiency appears to lower the threshold at which fluoride damages the brain, (Xu 1994; Guan 1988).[136] and dental fluorosis. (Zhao 1998; see also Pontigo-Loyola 2008).[137]

Iodine deficiency is still a public health concern in the United States. (CDC 1998). More than 11% of all Americans, and more than 15% of American women of child-bearing age, presently have urine iodine levels less than 50 mcg/L (Caldwell et al., 2008),[138] indicating moderate to severe iodine deficiency. An additional 36% of reproductive-aged women in the U.S. are considered mildly iodine deficient (<100 mcg/L urinary iodine). Without success, the National Research Council has therefore called for studies investigating the interactive effects of fluoride and iodine on US populations.

The Fluoride Goiter Iodine Connection

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[133] Ibid #6.

[134] Ibid #6

[135] Choi AL, et al. (2012). Developmental Fluoride Neurotoxicity: A Systematic Review and Meta-Analysis. *Environmental Health Perspectives* 2012 Jul 20. [Epub ahead of print]

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Studies dating back to the 19th century have implicated fluoride as a possible cause of goitre. Goitre (aka goiter) is an enlargement of the thyroid gland that in some cases can produce visible swelling in the neck. Although the main cause of goitre is iodine deficiency, it can also be caused by other things, including hypothyroidism and goitrogens (substances that cause goitre). Studies that have examined human populations with adequate intake of iodine have reported mixed results about fluoride's ability to produce goitre. (NRC 2006; Burgi 1984; McLaren 1969).[139] The research has been more consistent, however, where the examined populations had either excessive iodine intakes, or deficient iodine intakes. (Gas'kov 2005; Hong 2001; Wang 2001; Xu 1994; Yang 1994; Lin 1986).[140] Most of this latter research was initially published in either Russian or Chinese and was only recently translated into English by the Fluoride Action Network. Accordingly, previous reviews of fluoride/goitre research (e.g., NRC 2006) were not able to take these studies into account. As such, the evidence linking fluoride to goitre for populations with excessive, or deficient, iodine exposure is stronger than previously recognized.

Dogs have been found to suffer a high incidence of hypothyroidism, the relationship between fluoride contamination and thyroid disease in pets deserves further attention, particularly since it was fluoride's production of goiter in dogs that first prompted the idea that fluoride could be an anti-thyroid agent. (Maumene 1854).[141]

A consistent body of animal and human research shows that fluoride exposure worsens the impact of an iodine deficiency. Iodine is the basic building block of the T3 and T4 hormones and thus an adequate iodine intake is essential for the proper functioning of the thyroid gland. When iodine intake is inadequate during infancy and early childhood, the child's brain can suffer permanent damage, including mental retardation.[142]

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See also

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In China, researchers have repeatedly found that an iodine deficiency coupled with fluoride exposure produces a significantly more damaging effect on neurological development than iodine deficiency alone. In the first study to investigate the issue,

Ren (1989) "From the results it is evident that disrupted child intellectual development is among the effects on the human body from a harmful environment containing both high fluoride and low iodine, and this disruption is clearly much more serious than the effects of iodine deficiency alone."[143]

In 1991, a UNICEF-funded study concluded that fluoride levels of just 0.9 ppm (less than the level added to many water supplies for fluoridation) were sufficient to worsen the effects of iodine deficiency. The authors found that, when compared to children with iodine deficiency in a low-fluoride area, the children with iodine deficiency in the 0.9 ppm area had increased TSH levels, reduced T3, reduced intelligence, retarded bone development, and reduced hearing. According to the authors:

"Statistically significant differences existed between these areas, suggesting that a low iodine intake coupled with high fluoride intake exacerbates the central nervous lesions and the somatic developmental disturbance of iodine deficiency."[144]

In 1994, Xu and colleagues measured the IQ rates of children living in 8 areas with differing levels of both iodine and fluoride in exposure. Of all the areas studied, the region with the high fluoride/low iodine content had the lowest IQ. In addition, when compared against the low-iodine area, the high fluoride/low iodine area had a significantly higher rate of thyroid swelling. According to the authors:
"A higher chance of one being affected by thyroid swelling is likewise more prevalent in regions containing a high amount of fluoride but low amount of iodine, and regions where a relatively lower amount of iodine is detected. We believe that in a region where the level of iodine is low, but fluoride is significantly elevated, the level of toxicity in thyroid swelling could increase."[145]

Wang (2004) "In comparison with control rats, the learning and memory ability of the offspring rats was depressed by high fluoride, low iodine, or the combination of high fluoride and low iodine. Brain protein was decreased by low iodine and even more by the combined interaction of high fluoride and low iodine. The activity of cholinesterase (ChE) in the brain was affected to some extent by high fluoride and low iodine but was especially affected by high fluoride and low iodine together."[146]

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[143] Ren D, et al. (1989). A study of the intellectual ability of 8-14 year-old children in high fluoride, low iodine areas. Chinese Journal of Control of Endemic Diseases 4(4):251 (republished in Fluoride 2008; 41:319-20).

[144] SOURCE: Lin Fa-Fu; et al (1991). The relationship of a low-iodine and high-fluoride environment to subclinical cretinism in Xinjiang. Endemic Disease Bulletin 6(2):62-67 (republished in Iodine Deficiency Disorder Newsletter Vol. 7(3):24-25).

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[146] Wang J, et al. (2004). Effects of high fluoride and low iodine on biochemical indexes of the brain and learning-memory of offspring rats. Fluoride 37(4): 201-208.

Hong (2001) “The IQ results of this study show no significant difference between the average IQs of those children from the high fluoride only areas and the high fluoride/high iodine areas, however the result from the high fluoride/low iodine group show statistically significant differences as compared to that of the low fluoride/low iodine group.”[147]

The interactive effects of fluoride and low iodine on neurological health is consistent with other research showing that fluoride intensifies the anti-thyroid effects of iodine deficiency, and vice versa.

Guan (1988) “This study reveals that the degree of impairment of thyroid morphology and function is related with the amount of fluorine taken by rats. Goiter occurs in rats with iodine deficiency. Damage to the thyroid is observed in rats on iodine deficient diet and highly fluorinated water [30 ppm]. These changes are much more severe than in rats on a normal level iodine diet and highly fluorinated water. This seems to suggest that competitive antagonistic action exists between fluorine and iodine in the thyroid gland.”[148]

An animal study by Zhao et al (1998) found that fluoride and low iodine have “mutually interacting effects” on the thyroid gland, as evident by changes in thyroid weight, time-specific alterations in thyroid hormone levels, increased bone fluoride content, and increased severity of dental fluorosis. As with other studies, Zhao found that fluoride has interactive effects with iodine excess as well. [See study]

More recently, a team of Russian researchers studied a population with iodine deficiency that was exposed to varying levels of fluoride air pollution. The team found that indices of thyroid disease, including stunted growth and thyroid swelling, were more severe, and prophylactic measures less effective, in the population with heavier exposure to fluoride pollution. According to the authors:
“Natural iodine deficiency and ambient air pollution with fluorine compounds were examined for their combined influence on the prevalence and severity of iodine-deficiency disorders. The excess intake of fluorine was shown to increase the incidence of thyroid diseases and to lower anthropometric indices in children. The preventive measures performed to eliminate iodine-deficiency disorders under intensive ambient air pollution with fluorine compounds were found to be insufficiently effective.”[149]

Fluoride, Low Iodine, and Dental Fluorosis

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[147] Hong F, et al. (2001). Research on the effects of fluoride on child intellectual development under different environments. Chinese Primary Health Care 15(3):56-57 (republished in Fluoride 2008; 41(2):156–60).
[148] Guan ZZ, et al. (1988). Synergistic action of iodine-deficiency and fluorine-intoxication on rat thyroid. Chinese Medical Journal 101(9):679-84.
[149] Gas’kov AIu, et al. (2005). [The specific features of the development of iodine deficiencies in children living under environmental pollution with fluorine compounds]. [Article in Russian] Gig Sanit. 2005 Nov-Dec;(6):53-5.

As noted above, the animal study by Zhao (1998) found that iodine deficiency worsened the severity of dental fluorosis in the fluoride-treated rats. Xu (1994) found far higher rates of dental fluorosis in a population with low iodine exposure, than a similar population with adequate iodine exposure. Although both communities had 0.8 ppm fluoride in the water, the rate of dental fluorosis was 89% in the low-iodine area, which was more than double the fluorosis rate (40%) in the area with adequate iodine. (Similar to dental fluorosis in the USA).

More recently, a research team in Mexico reported a high rate of fluorosis in an area known for iodine deficiency. (Pontigo-Loyola 2008). Since the rate of fluorosis was higher than would be expected under normal circumstances, the authors suggested that iodine deficiency could be one of the factors contributing to the high rate. According to the authors,

"The hypothesized relationship between iodine deficiency and increased prevalence of fluorosis appears to be relevant to Hidalgo."[150]

Iodine Deficiency in the United States

Over the past few decades, the rate of iodine deficiency has increased in the United States. According to the National Research Council (NRC), "Iodine intake in the United States (where iodine is added to table salt) is decreasing, and an estimated 12% of the population has low concentrations of urinary iodine." (NRC 2006). In light of this trend, the NRC has called upon researchers to begin studying the endocrine and neurological effects that fluoride exposures may be having on the health of people with low iodine intake. As the NRC stated in 2006:

"The effects of fluoride on various aspects of endocrine function should be examined further, particularly with respect to a possible role in the development of several diseases or mental states in the United States. Major areas for investigation include the following: thyroid disease (especially in light of decreasing iodine intake by the U.S. population)."

GOITER HISTORY

Goitre (goiter) is an enlargement of the thyroid gland that in some cases can produce visible swelling in the neck. The suggested main deficiency cause of goitre is iodine. Goitre can also be caused by other things, including hypothyroidism and substances that cause goitre (goitrogens).

Since as far back as the 19th century, fluoride has been identified as a possible goitrogen. In the research to date, studies that have examined human populations with adequate intake of iodine have reported mixed results about fluoride's ability to produce

[150]Pontigo-Loyola AP, et al. (2008). Dental fluorosis in 12- and 15-year-olds at high altitudes in above-optimal fluoridated communities in Mexico. Journal of Public Health Dentistry 68(3):163-6.

goitre. (NRC 2006; Burgi 1984; McLaren 1969). Where, however, the examined populations had either excessive iodine intakes, or deficient iodine intakes, the research has been more consistent in finding a goitrogenic effect from fluoride. (Gas'kov 2005; Hong 2001; Wang 2001; Xu 1994; Yang 1994; Lin 1986). Since most of this latter research was initially published in either Russian or Chinese and was only recently translated into English by the Fluoride Action Network, the NRC's review of fluoride's goitrogenic potential (e.g, NRC 2006) was not able to take this evidence into account. As such, the evidence linking fluoride to goitre is stronger than previously determined, at least for populations with excessive, or deficient, exposure to iodine.

Origins of the Fluoride/Goitre Connection:

Fluoride was first suspected to be a goitrogen in 1854, when Maumeme reported producing goitre in a dog after 4 months of daily fluoride exposure (9 to 55 mg/day). Based on this and subsequent research in the early 20th century, doctors in Europe and South America began using fluoride as a medical treatment for hyperthyroidism (over-active thyroids). (McLaren 1969). As a goitrogen, doctors believed fluoride could suppress the thyroid's function and thereby alleviate symptoms in people with overly active thyroids. Subsequent clinical research found merit in this idea, as a daily fluoride treatment of just 2 to 5 mg/day was found capable of reducing thyroid function in a group of hyperthyroid patients. (Galletti & Joyet 1958). Ultimately, however, more effective treatments were discovered and the use of fluoride was phased out by the 1960s. (Merck Index 1968).

Fluoride & Goitre in Humans:

Note: the NRC (2006) review did not include the last decade of research and more studies have been translated.

NRC (2006):

"Three studies illustrated the range of results that have been reported: (1) Gedalia and Brand (1963) found an association between endemic goiter in Israeli girls and iodine concentrations in water but found no association with fluoride concentrations (<0.1-0.9 mg/L). (2) Siddiqui (1960) found goiters only in persons aged 14-17 years; the goiters, which became less visible or invisible after puberty, were associated with mean fluorine content of the water (5.4-10.7 mg/L) and were inversely associated with mean iodine content of the water. (3) Desai et al. (1993) found a positive correlation ($P < 0.001$) between prevalence of goiter (9.5-37.5%) and enamel fluorosis (6.0-59.0%), but no correlation between prevalence of goiter and water iodine concentration ($P > 0.05$)."

The NRC did not have access to a series of Chinese studies that FAN[151] has subsequently translated that provide data on the relationship between fluoride and goitre in communities with either iodine excess, or iodine deficiency. In these studies, fluoride's capacity to increase the goitre rate has been consistently demonstrated,

[151] FAN, Fluoride Action Network. www.fluoridealert.org

suggesting that the relationship between fluoride and goitre is stronger and more easily detected in populations (and individuals) with sub-optimal iodine intakes.

Meng (2013)“ Fluoride, a goitrogenic substance in drinking water, is another contributing factor to high GP. The fluoride concentration of drinking water was as high as 1.00 mg/kg in Chongqing municipality, which led Chongqing to have the highest GP (18.37%, 18 of 98) amongst all study areas.”[152]

Gas’kov (2005)“ Analysis of the simultaneous action of factors of the environment (iodine deficits and fluorosis) has shown that the basic cause of enlargement of the thyroid in children is an excessive intake of fluorine. Increasing the amount of iodine absorbed under conditions of excessive intake of fluorine cannot be an effective prophylactic measure directed at the elimination of iodine deficiency states.”[153]

Hong F (2001) “In endemic areas with high fluoride and high iodine, there was greater prevalence of both fluorosis and goiter than in the areas with only one of these two factors. . . . The high fluoride/low iodine group had an increased rate of goiter as compared to low fluoride/low iodine group, possibly stemming from the toxic effects of fluoride interacting with and aggravating the damage caused by a low iodine environment.”[154]

Wang X (2001) “In high iodine and high fluorine areas, the goiter and dental fluorosis rates of children aged from 8 to 12 were clearly higher than the control point, indicating that high iodine and high fluorosis have worse effects on children’s thyroid and teeth.”[155]

Yang (1994) “For children 15 or younger, the rate of thyroid swelling was 29.8% (96/322), and the rate of dental fluorosis reached 72.98% (235/322). In the control group, the rates were 16.13% (15/93) and 18.28 (17/93), respectively, with P<0.01 in all cases, indicating that the harm caused by a high fluoride-high iodine environment

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[152] Meng F, et al. (2013). Assessment of iodine status in children, adults, pregnant women and lactating women in iodine-replete areas of China. PLoS One 8(11):e81294.
[153] Gas’kov A, et al. (2005). The specific features of the development of iodine deficiencies in children living under environmental pollution with fluorine compounds. *Gig Sanit.* Nov-Dec;(6):53-5.
[154] Hong F, et al. (2001). Research on the effects of fluoride on child intellectual development under different environments. Chinese Primary Health Care 15(3):56-57 (republished in Fluoride 2008; 41(2):156–60).
[155] Wang X, et al. (2001). Effects of high iodine and high fluorine on children’s intelligence and thyroid function. Chinese Journal of Endemiology 20(4):288-90.

is particularly serious in the case of children."[156]

Lin F (1986) "In the lower alluvial plains, endemic goiter occurred concomitantly with endemic fluorosis and the contents of iodine in both water and urine were higher, but did not reach the level found in countries where goiter could be attributed to excess intake of iodine.The fact that in the circumstances of the lower uptake of I in thyroid for 24 hours and normal values of T3, T4, TSH, endemic goiter still was slightly prevalent indicated that fluoride also was a factor responsible for goiter."[157]

Jooste (1999) "OBJECTIVE: The study was undertaken to investigate whether endemic goitre still exists in the Northern Cape Province of South Africa more than 55 years after it was reported and, if so, whether iodine deficiency, or fluoride in the drinking water, is linked to the goitres. DESIGN: Cross-sectional study of children in three pairs of towns. SUBJECTS: The 6-, 12- and 15-year-old children (n = 671) who had been lifetime residents in two Northern Cape towns with low levels, two towns with near optimal levels and two towns with high levels of fluoride in the drinking water were recruited through the schools as study participants. RESULTS: Endemic goitre was found in all the towns except one, ranging from 5% to 29%. Iodine deficiency did not prevail in the study area because the median urinary iodine concentration, exceeding 1.58 micromol/l in all but one of the towns, indicated a more than adequate iodine consumption. The drinking water and, to a lesser extent, iodised salt were important sources of iodine. No relationship was found between fluoride in the water and the mild goitre prevalence (5% to 18%) in the four towns with either a low or near optimal fluoride content in the water. The causal factor(s) responsible for the goitres in these four towns were not clear from our data. However, the prevalence of goitre was higher (28% and 29%) in the two towns with high levels of fluoride in the water. CONCLUSION: These results indicate that either a high fluoride level in the water or another associated goitrogen, other than iodine deficiency, may have been responsible for these goitres."[158]

Desai (1993) "We examined 22,276 individuals for presence of goitre and dental fluorosis and estimated the fluoride and iodine content of their drinking water. Overall goitre and dental fluorosis prevalences were 14.0% and 12.2%, respectively, and were significantly and positively correlated. No significant relationship was observed between water iodine level and goitre. In the study area only 0.3% of cases were visible goitre (Grade-II and above) and all goitre cases were euthyroid.

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[156] Yang Y, et al. (1994). The effects of high levels of fluoride and iodine on intellectual ability and the metabolism of fluoride and iodine. Chinese Journal of Epidemiology 15(4):296-98 (republished in Fluoride 2008; 41:336-339).

[157] Lin F, et al. (1986). A preliminary approach to the relationship of both endemic goiter and fluorosis in the valley of Manasi River, Xin-Jiang to environmental geochemistry. Chinese Journal of Endemiology 5(1):53-55.

[158] Jooste PL, et al. (1999). Endemic goitre in the absence of iodine deficiency in schoolchildren of the Northern Cape Province of South Africa. European Journal of Clinical Nutrition 53(1):8-12.

This suggests that fluoride-induced goitres are brought about by anatomical or structural changes rather than functional changes."[159]

Obel (1982)" Areas which have endemic goitre in Kenya are highlands in the central parts of the country where there are no lakes from which iodide-rich foodstuffs, such as fish, could be found. Iodized salt has been mandatorily available in Kenya for many years. Indeed, most of the cases of goitre from these areas do not show iodide deficiency on biochemical evaluation. Many of these patients manifest clinical and laboratory findings of simple goitre (normal plasma levels of thyroxine, triiodothyronin, thyroid stimulating hormone, and normal iodine uptake values). It therefore would appear unlikely that absolute iodide deficiency per se would account for endemic goitre in Kenya. . . . It is interesting that the same areas which suffer from endemic goitre in Kenya also have the highest prevalence of fluorosis in the country. Indeed, many cases of fluorosis in Kenya have concurrent fluorosis."[160]

Day (1972). "The prevalence of goitre in 17 Himalayan villages has been estimated. Water-samples from each village were taken, and levels of iodine, fluoride, and hardness determined. In 13 villages wide variations in goitre prevalence were not attributable to differences in iodine intake, which remained constant within a narrow range. Instead, variations in goitre prevalence were found to correlate closely with the fluoride content (p=0-74; P<0-01) and with the hardness (p=0.77; P<0-01) of the water in each village. The effects of fluoride and water hardness seem to be independent."[161]

Siddiqui (1969) "With regard to the slight and temporary enlargement of the thyroid encountered in the age group 14-17 (type b), detailed scrutiny of the data . . . reveals that with a fall in mean fluorine content of the water from 10.7 mg/l in Kamaguda to 5.4 mg/l in Yellareddyguda, there was a corresponding progressive fall in the incidence of pubertal goiters from 40% in Kamaguda to 9% in Yellareddyguda, However, associated with the fall in fluorine content there was also a rise in mean iodine of the water. The figures can be interpreted to indicate that, so far as type b goiters are concerned, (1) fluorine may be actually goitrogenic, and (2) high concentrations of iodine may have a goiter-preventing effect. Investigations in other areas, where the variations in fluorine content are not associated with variations in iodine content of the type encountered here, may throw light on this particular problem."[162]

Steyn DG, et al. 1955. In 1936 while on an investigation into poisoning of man and animal by subterranean waters in the North-Western Cape Province, one of us

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[159] Desai VK, et al. (1993). Epidemiological study of goitre in endemic fluorosis district of Gujarat. Fluoride. 26(3):187-90.
[160] Obel AO. (1982). Goitre and fluorosis in Kenya. East African Medical Journal 59:363-365.
[161] Day TK, Powell-Jackson PR. (1972). Fluoride, water hardness, and endemic goitre. Lancet 1:1135-1138.
[162] Siddiqui AH. (1969). Incidence of simple goiter in areas of endemic fluorosis in Nalgonda district, Andhra Pradesh, India. Fluoride 2(2): 200-05.

[D.G.S. (126-129)] encountered several cases of goitre in European women living on farms. Enquiries made, revealed that a fair percentage of people, especially women, settling in this part of the country developed enlargement of the thyroid gland within 10 to 15 years after having entered the area. This was a puzzling phenomenon as the North Western Cape Province is known to be rich in iodine. It was realized that endemic goitre in this area could not possibly be the result of primary iodine deficiency in the soil, food and water. It was thought that the cause must be sought in the drinking water. The area is semi-arid and all drinking water, except that of towns and farms situated on the Orange River, is drawn from wells and boreholes. It was also known that the subterranean waters in the North-Western Cape Province generally contain harmful quantities of fluorine. It was considered that there was a possibility that fluorine has an antithyroid (goitrogenic) action. After having consulted the literature and conducting some experiments upon rats, it was realized that fluorine is a goitrogenic agent and that endemic goitre in the North-Western Cape Province is due not to an inherent primary iodine deficiency but chiefly to the general presence of harmful quantities of fluorine in the drinking-water. It is possible that the large quantities of calcium generally present in the subterranean waters in that area, enhances the goitrogenic effect of fluorine. Generally speaking the diet of the people is very satisfactory as it included a good percentage of meat with vegetables, fruit and bread. A large percentage of the vegetables and fruit is imported."[163]

Wilson (1941) "The distribution of endemic goitre in the Punjab and in England is related to the geological distribution of fluorine and to the distribution of human dental fluorosis (mottled enamel). Inquiry showed the presence of dental fluorosis among school-children in two areas of Somerset where two previous observers had recorded a high incidence of goitre, and the absence of dental fluorosis in an adjoining area selected as control where endemic goitre was absent."[164]

[163] Steyn DG, et al. 1955. Endemic goitre in the Union of South Africa and some neighbouring territories. Union of South Africa. Department of Nutrition.

[164] Wilson DC. (1941). Fluorine in the aetiology of endemic goitre. The Lancet 15(6129): 212-213.

Liu H (2013) “Excessive iodide and fluoride coexist in the groundwater in many regions, causing a potential risk to the human thyroid. To investigate the mechanism of iodide- and fluoride-induced thyroid cytotoxicity, human thyroid follicular epithelial cells (Nthy-ori 3-1) were treated with different concentrations of potassium iodide (KI), with or without sodium fluoride (NaF). . . . Collectively, excessive iodide and/or fluoride is cytotoxic to the human thyroid. Although these data do not manifest iodide could induce the IRE1 pathway, the cytotoxicity followed by exposure to fluoride alone or in combination with iodide may be related to IRE1 pathway-induced apoptosis. Furthermore, exposure to the combination of excessive iodide and fluoride may cause interactive effects on thyroid cytotoxicity.”[165]

Liu (2012) “Endemic fluorosis is a serious problem in public health. Previous studies have indicated that patients with thyroid goiters usually live in fluoride-affected areas. However, the mechanism of goitrogenesis caused independently by fluoride is still unclear. The principle objective of this study was to investigate the possible roles of nitric oxide (NO) and vascular endothelial growth factor (VEGF) in the genesis of fluoride-induced nodular goiters. . . . The results showed that the average relative weight of the thyroid glands of rats in the fluoride-treated groups was significantly higher than that in control rats ($p<0.05$). The proliferation and dilatation of capillary blood vessels, enlarged follicles with excessive colloid, and obvious nodules were found in the thyroid glands of fluoride-treated rats. Compared to the control group, the expression of VEGF mRNA in the thyroid gland and the serum NO levels in the fluoride-treated groups were significantly increased ($p<0.05$). Furthermore, the deposition of VEGF in epithelial and follicular cells of the thyroid gland was significantly higher in fluoride-treated groups than in the control group. These results suggested that abnormal expression of VEGF induced by fluoride can lead to the proliferation of vascular endothelial cells in the thyroid gland. Accordingly, VEGF oversecreted locally by vascular endothelial cells might contribute to the proliferation of epithelial and follicular cells, resulting in the formation of hyperplastic nodules and enlargement of the thyroid gland. Furthermore, we proposed that there might be a positive feedback mechanism between NO and VEGF expression in fluoride-induced goiter formation. It was concluded that angiogenic and vasodilative factors such as VEGF and NO must be involved in fluoride-induced thyroid goitrogenesis.”[166]

Zeng Q (2012) “To explore the toxic effect of fluoride on the human thyroid cells (Nthy-ori 3-1) and its mechanism. . . . To Nthy-ori 3-1 cells, fluoride under experimental concentrations decreases cell viability, improve the LDH leakage rate, and ROS level. It

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[165]Liu H et al, The role of the IRE1 pathway in excessive iodide- and/or fluoride-induced apoptosis in Nthy-ori 3-1 cells in vitro. Toxicol Lett. 2014 Jan 30;224(3):341-8. doi: 10.1016/j.toxlet.2013.11.001. Epub 2013 Nov 11.

[166] Liu G[1], Zhang W, Jiang P, Li X, Liu C, Chai C. Role of nitric oxide and vascular endothelial growth factor in fluoride-induced goitrogenesis in rats. Environ Toxicol Pharmacol. 2012 Sep;34(2):209-17. doi: 10.1016/j.etap.2012.04.003. Epub 2012 Apr 10.

blocks the cells in S phase and induce cell apoptosis."[167]

Liu (2012) "Endemic fluorosis is a serious problem in public health. Previous studies have indicated that patients with thyroid goiters usually live in fluoride-affected areas. . . It was concluded that angiogenic and vasodilative factors such as VEGF and NO must be involved in fluoride-induced thyroid goitrogenesis."[168]

Bashar (2011) "High-fluoride (100 and 200 ppm) water was administered to rats orally to study the fluoride-induced changes on the thyroid hormone status, the histopathology of discrete brain regions, the acetylcholine esterase activity, and the learning and memory abilities in multigeneration rats. Significant decrease in the serum-free thyroxine (FT4) and free triiodothyronine (FT3) levels and decrease in acetylcholine esterase activity in fluoride-treated group were observed. Presence of eosinophilic Purkinje cells, degenerating neurons, decreased granular cells, and vacuolations were noted in discrete brain regions of the fluoride-treated group. In the T-maze experiments, the fluoride-treated group showed poor acquisition and retention and higher latency when compared with the control. The alterations were more profound in the third generation when compared with the first- and second-generation fluoride-treated group. Changes in the thyroid hormone levels in the present study might have imbalanced the oxidant/antioxidant system, which further led to a reduction in learning memory ability. Hence, presence of generational or cumulative effects of fluoride on the development of the offspring when it is ingested continuously through multiple generations is evident from the present study."[169]

Cai (2009) "Objective: To observe the effects of fluoride on thyroid morphology, thyroid peroxidase and serum thyroid hormones. Methods: One-month ablactating SD rats were randomly divided into groups: the control group low-fluoride group, middle-fluoride group, high-fluoride group; fed with water containing different fluoride concentration by adding NaF respectively. Rats were sacrificed after being fed for six months. The morphology of thyroid was observed through light microscope. The TPO activity was measured with upgrade guaiacol method. Radio-immunoassay

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[167] Zeng Q et al. [Studies of fluoride on the thyroid cell apoptosis and mechanism]. [Article in Chinese] Journal; Zhonghua Yu Fang Yi Xue Za Zhi. 2012 Mar;46(3):233-6.

[168] Liu G, Zhang W, Jiang P, Li X, Liu C, Chai C. Role of nitric oxide and vascular endothelial growth factor in fluoride-induced goitrogenesis in rats. Environ Toxicol Pharmacol. 2012 Sep;34(2):209-17. doi: 10.1016/j.etap.2012.04.003. Epub 2012 Apr 10.

[169] Basha PM[1], Rai P, Begum S. Fluoride toxicity and status of serum thyroid hormones, brain histopathology, and learning memory in rats: a multigenerational assessment. Biol Trace Elem Res. 2011 Dec;144(1-3):1083-94. doi: 10.1007/s12011-011-9137-3. Epub 2011 Jul 14.

was used to detect serum thyroid hormones. Results: The major changes included increased follicles with colloid accumulation in high fluoride groups. With the dose of fluoride increasing, TPO activity significantly decreased as compared with the control group (P0.05). FT4 levels of the high-fluoride were significantly lower compared with the control group (P0.05). Conclusions: Chronic fluoride excess leads to definite histological changes in rat thyroid, inhibiting TPO activity so that level of thyroid hormone is decreased,which shows that fluoride can cause goiter, and cause abnormal changes of thyroid metabolism function."[170]

Zang (2008) "To investigate the mechanism of goiter caused by fluoride, goiter model of SD rats was produced by administering sodium fluoride in drinking water. Histological section of thyroid gland was made, and inducible nitric oxide synthase (iNOS) and vessel endothelial growth factor (VEGF) were determined by RT-PCR. Results showed that the capillary vessels in thyroid glands of the rats treated with fluoride proliferated and an obvious nodular goiter occurred in the fluoride-treated rats. Compared with the control, the contents of iNOS and VEGF in the thyroid glands of the rats with fluorosis was increased significantly (P0.05).It was concluded from the results that the mechanism of goiter caused by fluoride was that fluoride induced the over-expressions of iNOS and VEGF mRNAs in thyroid gland, which caused hyperplasia of capillary vessels."[171]

Shen (2004) "OBJECTIVE: Investigating the influence of combined iodine and fluoride on phospholipid and fatty acid composition in brain cells of rats. METHODS: Five groups of rats were provided with deionized drinking water containing 0 and 150 mg/L NaF, and containing both 150 mg/L NaF and 0.003, 0.03 or 3 mg/L KI respectively for 5 months. Then phospholipid and fatty acid composition were determined using liquid chromatography. RESULTS: The phospholipid composition had no obvious change. The high concentration fluoride (150 mg/L) and high concentration Iodine (3 mg/L) with high concentration fluoride could cause significant changes of the fatty acid composition in brain cells of rats, the proportion of unsaturated fatty acid (C18:2) was significantly decreased and the saturated fatty acid (C12:0) increased obviously. The antagonistic action of 0.03 mg/L KI drinking water on this kind of influence induced by 150 mg/L NaF was the most evident, whereas that of 3 mg/L KI was action of synergetic toxicity. CONCLUSION: Fluorosis had obvious influence on phospholipid and fatty acid composition in brain cells of rats, and its mechanism might be associated with action of lipid peroxidation, and 0.03 mg/L KI is the optimal concentration for the antagonistic action with this influence from fluorosis."[172]

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[170] Cai Q, Li Hong. (2009). Effects of Fluoride on the Thyroid Morphology and Thyroid Peroxidase and Serum Thyroid Hormones. Journal of Liaoning Medical University.

[171] Zhang W, et al. (2008). Expressions of iNOS and VEGF mRNAs in thyroid gland of rat with goiter induced by fluoride. Chinese Veterinary Science.

[172] Shen X, et al. (2004). [Influence of combined iodine and fluoride on phospholipid and fatty acid composition in brain cells of rats]. Wei Sheng Yan Jiu 33(2):158-61. [Article in Chinese]

Zhao (1998) “fluorine also affected the thyroid changes induced by ID [iodine deficiency] or IE [iodine excess]. After 100 days of treatment, fluorine showed some stimulatory effect on the thyroid in ID conditions and inhibitory effect in IE conditions. After 150 days, however, the effects of fluorine on the thyroid reversed as compared with that of 100 days. On the other hand, difference of iodide intake could also increase the toxic effects of FE on the incisors and bones.”[173]

Burg (1984)[174] Burgi and colleagues published a critique of then-existing research linking fluoride to thyroid dysfunction, including goitre and included studies which failed to find a relationship between fluoride and goiters.

[173] Zhao W, et al. (1998). Long-term effects of various iodine and fluorine doses on the thyroid and fluorosis in mice. Endocrine Regulation 32(2):63-70.
Environ Toxicol Pharmacol. 2014 Jul;38(1):332-40. doi: 10.1016/j.etap.2014.06.008. Epub 2014 Jun 27.

[174] Burgi H, et al. (1984): Fluorine and the Thyroid Gland: A Review of the Literature. *Klin Wochenschr*. 1984 Jun 15;62(12):564-9.

Bill Osmunson DDS, MPH
Audrey Adams

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
GENERAL COUNSEL

February 14, 2013

Gerald Steel, PE
7303 Young Road NW
Olympia, WA 98502

Dear Mr. Steel:

This is in response to your letter of December 28, 2012 to EPA Administrator Lisa Jackson in which you asked several questions about the status of an MOU between EPA and the Federal Drug Administration (FDA) published in 1979. I am replying on behalf of her.

Your first question is whether, from the viewpoint of EPA, the purpose of a 1979 Memorandum of Understanding (MOU) between EPA and the Federal Drug Administration (FDA) was "to take away from FDA, and give to EPA, responsibility for regulating public drinking water additives intended for preventative health care purposes and unrelated to contamination of public drinking water?" Your second question is whether, if that was the purpose of the 1979 MOU, the MOU was terminated through a subsequent Federal Register notice.

The answer to your first question is no, so there is no need to address your second question. The purpose of the MOU was not to shift any responsibilities between the Agencies. Rather, it was to help facilitate effective coordination of our respective legal authorities. Under the Safe Drinking Water Act (SDWA), EPA is the lead federal agency with responsibility to regulate the safety of public water supplies. EPA does not have responsibility for substances added to water solely for preventative health care purposes, such as fluoride, other than to limit the addition of such substances to protect public health or to prevent such substances from interfering with the effectiveness of any required treatment techniques. SDWA Section 1412(b)(11); see also A Legislative History of the Safe Drinking Water Act, Committee Print, 97th Cong, 2d Session (February 1982) at 547. The Department of Health and Human Services (HHS), acting through the FDA, remains responsible for regulating the addition of drugs to water supplies for health care purposes.

The 1979 MOU was intended to address contamination of drinking water supplies as a result of direct or indirect additives to drinking water, not to address the addition of substances solely for preventative health purposes. 44 Fed. Reg. 42775 (July 20, 1979) ("EPA and FDA agree: (1) that *contamination* of drinking water from the use and application of direct and indirect additives and other substances poses a potential public health problem...")(emphasis added). It was intended to avoid potentially duplicative regulation of "food", which FDA had, in the past, considered to include drinking water. 44 Fed. Reg. 42775 (July 20, 1979). The MOU did not address drugs or other substances added to water for health care purposes.

I hope that this has adequately answered your inquiry. Please do not hesitate to contact Carrie Wehling of my staff (202-564-5492) if you have further questions about this.

Sincerely,

Steven M. Neugeboren
Associate General Counsel
Water Law Office

WASHINGTON STATE
Board of Health
ALWAYS WORKING FOR A SAFER AND HEALTHIER WASHINGTON

DATE: June 9, 2010

TO: Washington State Board of Health Members

FROM: Environmental Health Committee:
Karen VanDusen, Keith Higman, and John Austin

SUBJECT: **PETITION FOR RULE MAKING: WATER FLUORIDATION, WAC 246-290-220 AND WAC 246-290-460**

Background and Summary:

On May 11, 2010, the Washington State Board of Health received a petition for rule making in the form of an e-mailed letter from Bill Osmunson, DDS, MPH, president of Washington Action for Safe Water. The petition asks the Board to amend WAC 246-290-460 and WAC 246-290-220, sections in the Board's rules for Group A public water supplies. The first requested amendment would change the allowable concentration of a fluoridation additive from a range specified in rule to a range approved by the U.S. Food and Drug Administration (FDA). The second would change the requirement that drinking water fluoridation additives meet Standard 60 of the National Sanitation Foundation (NSF) and American National Standards Institute (ANSI) to a requirement the additives be approved by FDA under a New Drug Application.

RCW 34.05.330 provides the opportunity for anyone to petition the Board with a request to adopt, amend, or repeal any of its rules. Upon receipt of such a petition, the Board has sixty days to initiate rule making, deny the petition, or address concerns raised by the petitioner by alternate means. Board policy number 2005-001 sets forth the procedures followed by the Board when it receives such a request. According to this policy, the chair may either decide on the request and instruct the executive director to respond or take the request to the full Board for discussion and possible action.

Chair Higman has worked with the Board's Environmental Health (EH) Committee to review the petition and make a recommendation for action. Ned Therien, Board staff, will summarize this rule making petition and EH Committee recommendations for the Board. Please refer to materials behind Tab 16 for additional information.

Recommended Board Action

The Environmental Health Committee recommends the Board adopt the following motion:

> ***Motion**: The Board denies the petition for rule making from Dr. William Osmunson dated May 11, 2010 because the U.S. Food and Drug Administration has a memorandum of understanding with the U.S. Environmental Protection Agency clarifying that the latter agency has authority for regulating tap water.*

Discussion:

The Board has authority under RCW 43.20-050(2) to adopt rules for Group A public water supplies "necessary to assure safe and reliable public drinking water and to protect public health." The Board has further responsibility under RCW 70.142.010 to establish standards for chemical contaminants in public drinking water and "consider the best available scientific information in establishing the standards." The Board has adopted such rules in chapter 246-290 WAC. These rules set both a maximum contaminant level (MCL) for fluoride in drinking water and a lower allowable concentration range if fluoride is added to drinking water. These rules also require that drinking water additives meet NSF/ANSI Standard 60.

RCW 57.08.012 gives each water district the authority to decide whether to ask the electors of the water district to vote on adding fluoride to its tap water. The Board does not appear to have authority to adopt rules related to a water district deciding whether to fluoridate. The Board's authority is to regulate allowable concentration levels and method of approval of water additives.

Dr. Osmunson asked the Board of Pharmacy in 2009 to designate fluoride a poison under chapter RCW 69.38 RCW, Poisons—sales and manufacturing. Dr. Osmunson asserted that fluoridation of public water supplies was the therapeutic administration of fluoride and should be controlled by the laws for legend drugs. The Pharmacy Board's response was that RCW 57.08.012, by being more specific, supersedes the general statutory authority under which it regulates drugs.

For fluoride in drinking water, this Board has adopted the U.S. Environmental Protection Agency (EPA) primary MCL of 4 parts per million (ppm) and secondary MCL of 2 ppm under WAC 246-290-310. These standards are primarily intended for naturally occurring fluoride. The Board has adopted under WAC 246-290-460 an allowable concentration range for artificial fluoridation of public tap water. This range is 0.8–1.3 ppm and is based on the Centers for Disease Control and Prevention (CDC) "optimal" recommended levels to help prevent tooth decay. The Board has adopted under WAC 246-290-220 requirements that drinking water additives meet NSF/ANSI Standard 60. These organizations have developed these standards in association with EPA and the American Water Works Association.

CDC recommends public tap water be fluoridated to an "optimal" target concentration of 0.7–1.2 ppm to help prevent cavities. This is a range of target concentrations and the actual target for a given water supplier would be based on a five-year average of the maximum daily air temperature for the supplier's service area. CDC recommends the concentration be controlled within a range no less than 0.1 ppm below and no more than 0.5 ppm above a supplier's target concentration. For example, if the target concentration is determined to be 0.9 ppm, the control range would be between 0.8 ppm and 1.4 ppm. The Board's standard of 0.8–1.3 ppm in WAC 246-290-460 was set based on different target concentrations across the state, which fall between 0.9 ppm and 1.1 ppm. The allowable range permits a variation of no more than 0.4 above the target concentration for the warmest part of the state. Therefore, the Board's rule is more stringent than the CDC recommendation.

The National Research Council (NRC) Committee on Fluoride in Drinking Water issued a report in 2006 titled *FLUORIDE IN DRINKING WATER: A Scientific Review of EPA's Standards.* It

recommended the MCL for fluoride be lowered from 4 ppm, but did not recommend a new level. It concluded that 2 ppm seemed safe, but might be high enough to cause moderate tooth discoloration (less that 15% of children). It did not specifically address the issue of the CDC-recommended 0.7 - 1.2 ppm concentration range for adding fluoride to a water supply. On March 29, 2010, EPA published in the *Federal Register* an announcement of a six-year review of the MCLs for 71 chemicals, one of which was fluoride. It requested public comments on the reviews by May 28, 2010. EPA's conclusion is that it does not have information at this time that warrants it making a change to the MCL for fluoride, but studies are continuing.

CDC considers drinking water fluoridation one of the top ten great public health achievements of the 20th century. A series of surgeon general statements, the last issued in 2004, have strongly supported fluoridation of community water systems. CDC states that the 2006 National Research Council report supports CDC's recommended "optimal" fluoridation levels as being safe. CDC further states that the most common chemical used for fluoridation, fluorosilicic acid, and related compounds are derived in high purity from the gypsum and phosphate fertilizer manufacturing process. CDC cautions against the overuse of fluoride-containing products to control total intake. In a telephone call between Ned Therien and William Bailey, DDS, MPH, U.S. Public Health Service, on May 21 of this year, Captain Bailey stated that CDC is continually reviewing data regarding the "optimal" level and safety of tap water fluoridation. He also stated that EPA is currently doing risk assessment reviews of dose-response, source contribution, and the potential for carcinogenicity of fluoride.

In 1979, EPA and FDA finalized a memorandum of understanding regarding regulating fluoride levels in drinking water. They concluded the 1974 Safe Drinking Water Act gives EPA authority for regulating chemicals in tap water, while FDA has authority for chemicals in bottled water. Under CFR Title 21, Section 165.110, FDA has set a limit for fluoride added to bottled water in the U.S. of between 0.7 and 1.7 ppm, depending on annual average maximum air temperature for the location where bottled. In a May 21 e-mail exchange between Ned Therien and John V. Kelsey, DDS, MBA, Dental Team Leader, Division of Dermatology and Dental Products, FDA, Dr. Kelsey confirmed that FDA does not have regulatory responsibility for public water supplies, but rather that is the responsibility of EPA. He said if the Board accepted the language proposed in the petition, it effectively would ban public water fluoridation in Washington.

The Washington State Department of Health encourages community water fluoridation as a public health measure. State Health Officer Maxine Hayes, MD, MPH, issued a statement in support of community water fluoridation in 2006. The department's Oral Health Program echoes the recommendations of CDC on community water fluoridation and provides warnings about the overuse of fluoridated products. Many health professional associations support CDC's recommendations on community water fluoridation, including the American Dental Association, American Medical Association, American Academy of Family Physicians, and American Public Health Association.

The EH Committee concludes:

- EPA is the lead federal agency for regulating the maximum levels of contaminants and additives in tap water under the Safe Drinking Water Act.
- FDA has relinquished any authority it might have for regulating fluoride levels in tap water under the memorandum of understanding with EPA.
- The Board cannot direct a federal agency to take action.
- The State Board of Pharmacy has stated it cannot regulate tap water fluoridation under its authority.
- An NRC committee evaluated the scientific evidence of the health effects of fluoride in drinking water and published a report in 2006 that concluded fluoride levels in drinking water below 2 ppm are safe for health.
- EPA announced completion of a review of MCLs in the Federal Register in March 2010 that concluded it did not have evidence to revise the MCL for fluoride.
- EPA will be conducting additional reviews regarding fluoride levels in drinking water.
- EPA recognizes NSF/ANSI Standard 60 as appropriate for the approval of drinking water additives.
- The range of 0.8 ppm to 1.3 ppm fluoride in WAC 246-290-460 is within the control range (0.1 ppm below to 0.5 ppm above) recommended by CDC for target "optimal" concentrations based on average maximum temperatures in various regions of Washington.

The EH Committee recommends the Board deny Dr. Osmunson's petition for rule making on the grounds that FDA has stated it has no intention to regulate fluoride levels or approve additives for tap water. Therefore, adopting the proposed rule changes would, essentially, prohibit all tap water fluoridation in Washington and make Board rules conflict with RCW 57.08.012.

The EH Committee considers much of the discussion in the petition to make points that go beyond the requested rule changes and are not pertinent to its decision. However, the Committee recommends the Department of Health monitor EPA evaluations of safe drinking water levels for fluoride and recommendations from CDC for "optimal" fluoride levels, and that the Department propose rule amendments based on any changes. The Committee further recommends the next time the Department undertakes a major review of chapter 246-290 WAC, it consider proposing the word "optimal" in section 460(3) be changed to a phrase such as "generally regarded as safe." The Committee further recommends the Board continue to review legal points raised in the petition concerning state law and Attorney General opinions.

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Prenatal Fluoride Exposure and Cognitive Outcomes in Children at 4 and 6–12 Years of Age in Mexico

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Background: Some evidence suggests that fluoride may be neurotoxic to children. Few of the epidemiologic studies have been longitudinal, had individual measures of fluoride exposure, addressed the impact of prenatal exposures or involved more than 100 participants.

Objective: Our aim was to estimate the association of prenatal exposure to fluoride with offspring neurocognitive development.

Methods: We studied participants from the Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) project. An ion-selective electrode technique was used to measure fluoride in archived urine samples taken from mothers during pregnancy and from their children when 6–12 y old, adjusted for urinary creatinine and specific gravity, respectively. Child intelligence was measured by the General Cognitive Index (GCI) of the McCarthy Scales of Children's Abilities at age 4 and full scale intelligence quotient (IQ) from the Wechsler Abbreviated Scale of Intelligence (WASI) at age 6–12.

Results: We had complete data on 299 mother–child pairs, of whom 287 and 211 had data for the GCI and IQ analyses, respectively. Mean (SD) values for urinary fluoride in all of the mothers ($n = 299$) and children with available urine samples ($n = 211$) were 0.90 (0.35) mg/L and 0.82 (0.38) mg/L, respectively. In multivariate models we found that an increase in maternal urine fluoride of 0.5 mg/L (approximately the IQR) predicted 3.15 (95% CI: −5.42, −0.87) and 2.50 (95% CI −4.12, −0.59) lower offspring GCI and IQ scores, respectively.

Conclusions: In this study, higher prenatal fluoride exposure, in the general range of exposures reported for other general population samples of pregnant women and nonpregnant adults, was associated with lower scores on tests of cognitive function in the offspring at age 4 and 6–12 y. https://doi.org/10.1289/EHP655

Introduction

Community water, salt, milk, and dental products have been fluoridated in varying degrees for more than 60 y to prevent dental caries, while fluoride supplementation has been recommended to prevent bone fractures (Jones et al. 2005). In addition, people may be exposed to fluoride through the consumption of naturally contaminated drinking water, dietary sources, dental products, and other sources (Doull et al. 2006). Whereas fluoride is added to drinking water [in the United States at levels of 0.7–1.2 mg/L (Doull et al. 2006)] to promote health, populations with exceptionally high exposures, often from naturally contaminated drinking water, are at risk of adverse health effects, including fluorosis.

In the United States, the U.S. Environmental Protection Agency (EPA) is responsible for establishing maximum permissible concentrations of contaminants, including fluoride, in public drinking-water systems. These standards are guidelines for restricting the amount of fluoride contamination in drinking water, not standards for intentional drinking-water fluoridation. In 2006 the U.S. EPA asked the U.S. National Research Council (NRC) to reevaluate the existing U.S. EPA standards for fluoride contamination, including the maximum contaminant level goal (MCLG, a concentration at which no adverse health effects are expected) of 4 mg/L, to determine if the standards were adequate to protect public health (Doull et al. 2006). The committee concluded that the MCLG of 4 mg/L should be lowered because it puts children at risk of developing severe enamel fluorosis, and may be too high to prevent bone fractures caused by fluorosis (Doull et al. 2006). The Committee also noted some experimental and epidemiologic evidence suggesting that fluoride may be neurotoxic (Doull et al. 2006).

The National Toxicology Program (NTP) recently reviewed animal studies on the effects of fluoride on neurobehavioral outcomes and concluded that there was a moderate level of evidence for adverse effects of exposures during adulthood, a low level of evidence for effects of developmental exposures on learning and memory, and a need for additional research, particularly on the developmental effects of exposures consistent with those resulting from water fluoridation in the United States (Doull et al. 2006; NTP 2016). Human studies have shown a direct relationship between the serum fluoride concentrations of maternal venous blood and cord blood, indicating that the placenta is not a barrier to the passage of fluoride to the fetus (Shen and Taves, 1974). Fluoride was shown to accumulate in rat brain tissues after chronic exposures to high levels, and investigators have speculated that accumulation in the hippocampus might explain effects on learning and memory (Mullenix et al. 1995). An experimental study on mice has shown that fluoride exposure may have adverse effects on neurodevelopment, manifesting as both cognitive and behavioral abnormalities later in life (Liu et al. 2014).

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Most epidemiologic studies demonstrating associations between fluoride exposure and lower neuropsychological indicators have been conducted in populations living in regions with endemic fluorosis that are exposed to high levels of fluoride in contaminated drinking water. The epidemiologic evidence is limited, however, with most studies using an ecologic design to estimate childhood exposures based on neighborhood measurements of fluoride (e.g., drinking water levels) rather than personal exposure measures. Moreover, almost all existing studies of childhood outcomes are cross-sectional in nature, rendering them weak contributors towards causal inference.

The main objective of this study was to assess the potential impact of prenatal exposures to fluoride on cognitive function and test hypotheses related to impacts on overall cognitive function. We hypothesized that fluoride concentrations in maternal urine samples collected during pregnancy, a proxy measure of prenatal fluoride exposure, would be inversely associated with cognitive performance in the offspring children. Overall, to our knowledge, this is one of the first and largest longitudinal epidemiologic studies to exist that either address the association of early life exposure to fluoride to childhood intelligence or study the association of fluoride and cognition using individual biomarker of fluoride exposure.

Methods

This is a longitudinal birth cohort study of measurements of fluoride in the urine of pregnant mothers and their offspring (as indicators of individual prenatal and postnatal exposures to fluoride, respectively) and their association with measures of offspring cognitive performance at 4 and 6–12 y old. The institutional review boards of the National Institute of Public Health of Mexico, University of Toronto, University of Michigan, Indiana University, and Harvard T.H. Chan School of Public Health and participating clinics approved the study procedures. Participants were informed of study procedures prior to signing an informed consent required for participation in the study.

Participants

Mother–child pairs in this study were participants from the successively enrolled longitudinal birth cohort studies in Mexico City that comprise the Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) project. Of the four ELEMENT cohorts [that have been described elsewhere (Afeiche et al. 2011)], Cohort 1 and Cohort 2B recruited participants at birth and did not have archived maternal-pregnancy urine samples required for this analysis; they were thus excluded. Mothers for Cohort 2A ($n = 327$) and 3 ($n = 670$) were all recruited from the same three hospitals in Mexico City that serve low-to-moderate income populations. Cohort 2A was an observational study of prenatal lead exposure and neurodevelopmental outcomes in children (Hu et al. 2006). Women who were planning to become pregnant or were pregnant were recruited during May 1997–July 1999 and were considered eligible if they consented to participate; were ≤ 14 wk of gestation at the time of recruitment; planned to stay in the Mexico City study area for at least 5 y; did not report a history of psychiatric disorders, high-risk pregnancies, gestational diabetes; did not report current use of daily alcohol, illegal drugs, and continuous prescription drugs; and were not diagnosed with preeclampsia, renal disease, circulatory diseases, hypertension, and seizures during the index pregnancy.

Cohort 3 mothers were pregnant women (≤ 14 wk of gestation) recruited from 2001 to 2003 for a randomized trial of the effect of calcium supplementation during pregnancy on maternal blood lead levels (Ettinger et al. 2009). Eligibility criteria were the same as for Cohort 2A, and 670 agreed to participate.

Exposure Assessment

By virtue of living in Mexico, individuals participating in the study have been exposed to fluoridated salt (at 250 ppm) (Secretaría-de-Salud 1995, 1996) and to varying degrees of naturally occurring fluoride in drinking water. Previous reports, based on samples taken from different urban and rural areas, indicate that natural water fluoride levels in Mexico City may range from 0.15 to 1.38 mg/L (Juárez-López et al. 2007; Martínez-Mier et al. 2005). Mean fluoride content for Mexico City's water supply is not available because fluoride is not reported as part of water quality control programs in Mexico.

Mother–child pairs with at least one archived urine sample from pregnancy and measures of neurocognitive function in the offspring were included in this study. In terms of when the archived samples were collected, the pregnant mothers were invited for assessments with the collection of samples during trimester 1 (13.6 ± 2.1 wk for Cohort 3 and 13.7 ± 3.5 wk for Cohort 2A), trimester 2 (25.1 ± 2.3 wk for Cohort 3 and 24.4 ± 2.9 wk for Cohort 2A), and trimester 3 (33.9 ± 2.2 wk for Cohort 3 and 35.0 ± 1.8 wk for Cohort 2A).

A spot (second morning void) urine sample was targeted for collection during each trimester of pregnancy of ELEMENT mothers as well as the offspring children at the time of their measurements of intelligence at 6–12 y old. The samples were collected into fluoride-free containers and immediately frozen at the field site and shipped and stored at -20°C at the Harvard T. H. Chan School of Public Health (HSPH), and then at -80°C at the University of Michigan School of Public Health (UMSPH).

A procedure for urine analysis of fluoride described elsewhere (Martínez-Mier et al. 2011) was adapted and modified for this study. The fluoride content of the urine samples was measured using ion-selective electrode-based assays. First, 3 M sulfuric acid saturated with hexamethyldisiloxane (HMDS) was added to the sample to allow fluoride to diffuse from the urine for 20–24 hr. The diffused fluoride was allowed to collect in 0.05 M of sodium hydroxide on the interior of the petri dish cover. Once the diffusion was complete, 0.25 M of acetic acid was added to the sodium hydroxide to neutralize the solution and then analyzed directly using a fluoride ion-selective electrode (Thermo Scientific Orion, Cat#13-642-265) and pH/ISE meter (Thermo Scientific Orion, Cat#21-15-001). All electrode readings (in millivolts) were calculated from a standard curve. Analyses were performed in a Class 100/1,000 clean room. Quality control measures included daily instrument calibration, procedural blanks, replicate runs, and the use of certified reference materials (Institut National de Santé Publique du Québec, Cat #s 0910 and 1007; NIST3183, Fluoride Anion Standard). Urinary fluoride concentrations were measured at the UMSPH and the Indiana University Oral Health Research Institute (OHRI) as previously described (Thomas et al. 2016). A validation study comparing measures taken by the two labs in the same samples revealed a between-lab correlation of 0.92 (Thomas et al. 2016).

There were a total of 1,484 prenatal samples measured at the UMSPH lab. All of these samples were measured in duplicate. Of these, 305 (20%) of them did not meet the quality control criteria for ion-selective electrode-based methods (i.e., RSD $<20\%$ for samples with F level <0.2 ppm or RSD $<10\%$ when F level >0.2 ppm) (Martinez-Mier et al. 2011). Of these 305, 108 had a second aliquot available and were successfully measured at the OHRI lab in Indiana (sufficient urine volume was not available for the remaining 197 samples). The OHRI lab in Indiana also measured an additional 289 samples. Of the 397

total samples measured at the OHRI lab in Indiana, 139 (35%) were measured in duplicate, for which >95% complied with the quality control criteria above; thus, all 139 values were retained. The remaining 258 (65%) were not measured in duplicate because of limitations in available urine volume, but were included in the study given the excellent quality control at the OHRI lab. In total, we ended up with 1,576 prenatal urine samples with acceptable measures of fluoride.

Of these 1,576 urine samples, 887 also had data on urinary creatinine and were associated with mother–offspring pairs who had data on the covariates of interest and GCI or IQ in the offspring. The urinary creatinine data were used to correct for variations in urine dilution at the time of measurement (Baez et al. 2014). Creatinine-adjusted urinary fluoride concentrations were obtained for each maternally derived sample by dividing the fluoride concentration (MUF) in the sample by the sample's creatinine concentration (MUC), and multiplying by the average creatinine concentration of samples available at each trimester ($MUC_{average}$) using the formula: $(MUF/MUC) \times MUC_{average}$. The values of average creatinine concentration used for the $MUC_{average}$ at each trimester were derived from the larger pool of trimester-1, -2, and -3 samples from Cohorts 2A and 3 examined in our previous report on maternal fluoride biomarker levels (Thomas et al. 2016): 100.81, 81.60, and 72.41 (mg/L), respectively. For each woman, an average of all her available creatinine-adjusted urinary fluoride concentrations during pregnancy (maximum three samples and minimum one sample) was computed and used as the exposure measure (MUF_{cr}). For children, as creatinine measurements were not available, urinary fluoride values (CUF) were corrected for specific gravity (SG) using the formula $CUFsg = CUF(1.02-1)/(SG-1)$ (Usuda et al. 2007).

After calculating MUF_{cr} for the 887 urine samples noted above, 10 values of MUF_{cr} were identified as extreme outliers (>3.5 SDs) and were dropped, leaving 877 measures of MUF_{cr}. These 877 measures of MUF_{cr} stemmed from 512 unique mothers. Of these 512, 71 participants had measurements from each of the three trimesters; 224 had measurements from two of the three trimesters (74, T1 and T2; 131, T1 and T3; and 19, T2 and T3); and 217 had measurements from only one of the trimesters (159, T1; 34, T2; and 24, T3).

Measurement of Outcomes

At age 4 y, neurocognitive outcomes were measured using a standardized version of McCarthy Scales of Children's Abilities (MSCA) translated into Spanish (McCarthy 1991). MSCA evaluates verbal, perceptual-performance, quantitative, memory, and motor abilities of preschool-aged children, and it has previously been successfully used in translated versions (Braun et al. 2012; Julvez et al. 2007; Kordas et al. 2011; Puertas et al. 2010). For this analysis, we focused on the General Cognitive Index (GCI), which is the standardized composite score produced by the MSCA (McCarthy 1991). For children 6–12 y old a Spanish-version of the Wechsler Abbreviated Scale of Intelligence (WASI) (Wechsler 1999) was administered. WASI includes four subtests (Vocabulary, Similarities, Block Design, and Matrix Reasoning), which provide estimates of Verbal, Performance, and Full-Scale IQ (Wechsler 1999). Both tests were administered by a team of three psychologists who were trained and supervised by an experienced developmental psychologist (L.S.). This team of three psychologists applied all of the McCarthy tests as well as the WASI-FSIQ tests. At the time of follow-up visits (age 4 and 6–12 y), each child was evaluated by one of the psychologists who was blind to the children's fluoride exposure. The inter-examiner reliability of the psychologists was evaluated by having all three psychologists participate in assessments on a set of 30 individuals. For these 30, the inter-examiner reliability of the psychologists was evaluated by calculating the correlation in GCI scores by two of the psychologists with the scores of a third psychologist whom they observed applying the test in all three possible combinations with 10 participants for each observers–examiner pair (i.e., psychologist A (applicant) was observed by psychologist B and psychologist C; psychologist B (applicant) was observed by psychologist A and psychologist C; and psychologist C (applicant) was observed by psychologist A and psychologist B). The mean observer–examiner correlation was 0.99. All raw scores were standardized for age and sex (McCarthy 1991). Inter-examiner reliability was not examined on the WASI test.

Measurement of Covariates

Data were collected from each subject by questionnaire on maternal age (and date of birth), education, and marital status at the first pregnancy visit; on birth order, birth weight, and gestational age at delivery; and on maternal smoking at every prenatal and postnatal visit. Gestational age was estimated by registered nurses. Maternal IQ was estimated using selected subtests of the Wechsler Adult Intelligence Scale (WAIS)-Spanish (Information, Comprehension, Similarities, and Block Design), which was standardized for Mexican adults (Renteria et al. 2008; Wechsler et al. 1981). Maternal IQ was measured at the study visit 6 mo after birth or at the 12-mo visit if the earlier visit was not completed.

The quality of the children's individual home environments was assessed using an age-appropriate version of the HOME score. However, the measure was not available for all observations because it was only added to on-going cohort evaluation protocols beginning in April 2003, when a version of the HOME score instrument that is age-appropriate for children 0–5 y old was adopted, following which a version of the HOME score instrument that is age-appropriate for children ≥6 y old was adopted in September 2009 (Caldwell and Bradley 2003). Thus, we adjusted for HOME score using the measures for 0- to 5-y-old children in the subset of children who had this data in our analyses of GCI, and we adjusted for HOME score using the measures for >6-y-old children in the subset of children who had this data in our analyses of IQ.

Statistical Analyses

Univariate distributions and descriptive statistics were obtained for all exposure variables, outcome variables, and model covariates. For each variable, observations were classified as outliers if they were outside the bounds of the mean ± 3.5 SDs. Primary analyses were conducted with exposure and outcome outliers excluded. Statistical tests of bivariate associations were conducted using chi-square tests for categorical variables and analysis of variance (ANOVA) to compare the means of the outcomes or exposure within groups defined according to the distribution of each covariate. Spearman correlation coefficients were used to measure the correlation between MUF_{cr} and CUF_{sg}. Regression models were used to assess the adjusted associations between prenatal fluoride and each neurocognitive outcome separately. Generalized additive models (GAMs) were used to visualize the adjusted association between fluoride exposure and measures of intelligence [SAS statistical software (version 9.4; SAS Institute Inc.)]. Because the pattern appeared curvilinear, and because GAMs do not yield exact *p*-values for deviations from linearity, we used a Wald *p*-value of a quadratic term of fluoride exposure to test the null hypothesis that a quadratic model fit the data better

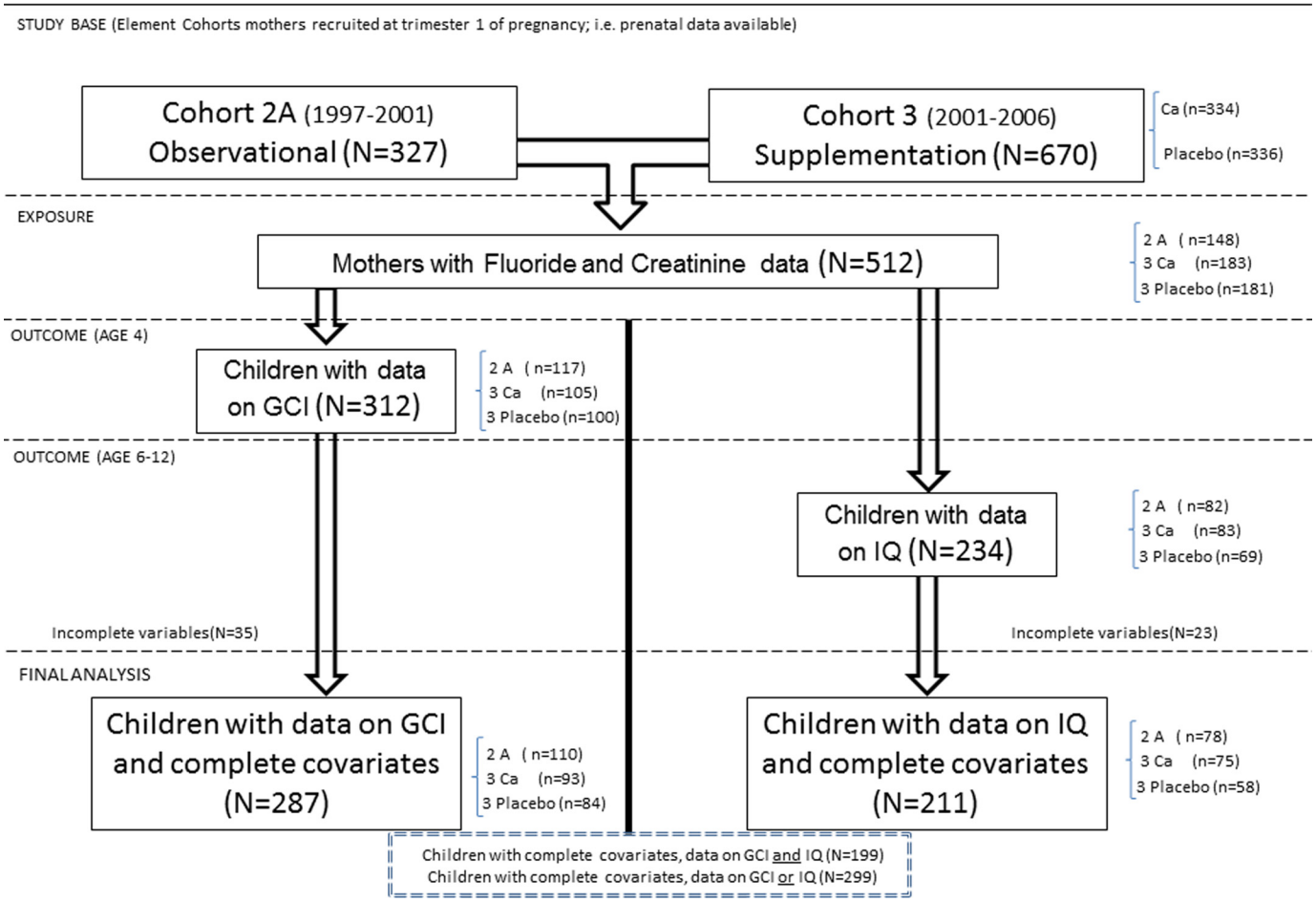

Figure 1. Flowchart describing source of mother–offspring subject pairs, fluoride and cognition study. Cohort 2A was designed as an observational birth cohort of lead toxicodynamics during pregnancy, with mothers recruited early during pregnancy from 1997 to 2001. Cohort 3 was designed as a randomized double-blind placebo-controlled trial of calcium supplements, with mothers recruited early during pregnancy from 2001 to 2006. "Ca" denotes subjects who were randomized to the calcium supplement; "placebo" denotes subjects who were randomized to the placebo. GCI is the McCarthy Scales General Cognitive Index (administered at age 4 y). IQ is the Wechsler Abbreviated Intelligence Scales Intelligence Quotient (administered at age 6–12 y and age-adjusted).

than the model assuming a linear relationship, and thus obtained a *p*-value for deviation from linearity of the fluoride–outcome associations. Residual diagnostics were used to examine other model assumptions and identify any additional potentially influential observations. Visual inspection of default studentized residual versus leverage plot from SAS PROC REG did not identify potential influential observations. Visual inspection of the histogram of the residuals did not indicate lack of normality; however, a fanning pattern in the residual versus predicted value plot indicated lack of constant variance (data not shown). Hence, robust standard errors were obtained using the "empirical" option in SAS PROC GENMOD.

Our overall strategy for selecting covariates for adjustment was to identify those that are well known to have potential associations with either fluoride exposure or cognitive outcomes and/or are typically adjusted for as potential confounders in analyses of environmental toxicants and cognition. All models were adjusted for gestational age at birth (in weeks), birthweight (kilograms), birth order (first born yes vs. no), sex, and child's age at the time of the neurocognitive test (in years). All models were also adjusted for maternal characteristics including marital status (married vs. others), smoking history (ever-smoker vs. never-smoker), age at delivery, IQ, and education (itself also a proxy for socioeconomic status). Finally, all models adjusted for potential cohort effects by including indicator variables denoting from which cohort (Cohort 2A, Cohort 3 + Ca supplement, and Cohort 3 -placebo) the participants came. We used 0.5 mg/L, which was close to the interquartile range of MUF_{cr} for the analyses of both GCI (IQR = 0.45) and IQ (IQR = 0.48), as a standard measure of incremental exposure. SAS statistical software (version 9.4; SAS Institute Inc.) was used for all data analyses described.

Sensitivity Analyses

Models were further adjusted for variables that relate to relatively well-known potential confounders (but for which we were missing a significant amount of data) and variables that were less-well known but possible confounders. The HOME scores were subject to sensitivity analyses because, as noted in the "Methods" section, they were not added to the subject evaluation protocols until 2003, resulting in a significantly smaller subsample of participants with this data. Models of the association between prenatal fluoride exposure (MUF_{cr}) and IQ at 6–12 y old were also adjusted for the child's urine fluoride concentration at 6–12 y of

Table 1. Comparisons across cohorts with respect to the distributions of biomarkers of exposure to prenatal fluoride (MUF_{cr}), prenatal lead (maternal bone Pb), prenatal mercury (maternal blood Hg), and contemporaneous childhood fluoride (CUF_{sg}); and cognitive outcomes (GCI and IQ).

							Percentiles				
Analysis	Measurement	Cohort	*N*	Mean	SD	Min	25	50	75	Max	*p*-Value[a]
GCI Analysis	GCI	Cohort 3-Ca	84	96.88	14.07	50	88	96	107	124	0.997
		Cohort 3-placebo	93	96.80	13.14	50	89	96	105	125	
		Cohort 2A	110	96.95	15.46	56	88	98	110	125	
		Total[b]	287	96.88	14.28	50	88	96	107	125	
	MUF_{cr} (mg/L)	Cohort 3-Ca	84	0.92	0.41	0.28	0.60	0.84	1.14	2.36	0.57
		Cohort 3-placebo	93	0.87	0.34	0.23	0.62	0.82	1.10	2.01	
		Cohort 2A	110	0.92	0.33	0.23	0.68	0.86	1.11	2.14	
		Total[b]	287	0.90	0.36	0.23	0.65	0.84	1.11	2.36	
	Maternal bone Pb (μg/g)	Cohort 3-Ca	62	7.30	7.37	0.05	0.75	4.40	12.93	26.22	<0.01
		Cohort 3-placebo	43	9.21	7.31	0.11	1.50	8.60	13.97	27.37	
		Cohort 2A	62	13.60	11.36	0.15	5.35	10.52	19.46	47.07	
		Total[c]	167	10.13	9.41	0.05	2.37	8.22	15.37	47.07	
	Maternal blood Hg (μg/L)	Cohort 3-Ca	38	3.32	1.40	0.73	2.40	3.00	4.15	7.06	0.12
		Cohort 3-placebo	28	2.80	1.33	1.27	1.89	2.53	3.40	7.22	
		Cohort 2A	75	4.53	5.61	0.77	2.30	3.24	4.37	35.91	
		Total[c]	141	3.86	4.25	0.73	2.20	3.08	4.15	35.91	
IQ Analysis	IQ	Cohort 3-Ca	58	94.91	9.86	76	87	96	100	120	0.69
		Cohort 3-placebo	75	96.29	9.63	75	89	97	102	124	
		Cohort 2A	78	96.47	13.20	67	87	96	107	131	
		Total[d]	211	95.98	11.11	67	88	96	107	131	
	MUF_{cr} (mg/L)	Cohort 3-Ca	58	0.89	0.38	0.29	0.57	0.84	1.10	1.85	0.86
		Cohort 3-placebo	75	0.87	0.35	0.23	0.61	0.82	1.11	2.01	
		Cohort 2A	78	0.90	0.34	0.23	0.67	0.85	1.09	2.14	
		Total[d]	211	0.89	0.36	0.23	0.64	0.82	1.07	2.14	
	Maternal bone Pb (μg/g)	Cohort 3-Ca	67	6.97	7.20	0.05	0.76	4.36	11.73	26.22	<0.01
		Cohort 3-placebo	48	9.07	7.42	0.11	1.00	8.49	14.41	27.37	
		Cohort 2A	62	13.60	11.36	0.15	5.35	10.52	19.46	47.07	
		Total[e]	177	9.86	9.33	0.05	2.29	7.95	15.22	47.07	
	Maternal blood Hg (μg/L)	Cohort 3-Ca	43	3.25	1.41	0.51	2.43	2.87	4.02	7.06	0.067
		Cohort 3-placebo	31	2.66	1.36	0.78	1.81	2.40	3.26	7.22	
		Cohort 2A	75	4.53	5.61	0.77	2.30	3.24	4.37	35.91	
		Total[e]	149	3.77	4.16	0.51	2.19	2.90	4.11	35.91	
	CUF_{sg} (mg/L)	Cohort 3-Ca	71	0.84	0.4	0.31	0.53	0.78	1.12	2.8	0.29
		Cohort 3-placebo	53	0.85	0.38	0.35	0.57	0.75	1.14	1.85	
		Cohort 2A	65	0.76	0.34	0.18	0.51	0.7	0.89	1.76	
		Total[e]	189	0.82	0.38	0.18	0.54	0.73	1.01	2.8	
All available measurements	GCI	Cohort 3-Ca	133	97.32	13.67	50	88	96	107	124	0.57
		Cohort 3-placebo	149	95.99	13.07	50	88	96	106	125	
		Cohort 2A	150	97.57	14.63	56	88	99	109	131	
		Total[f]	432	96.95	13.80	50	88	96	107	131	
	IQ	Cohort 3-Ca	91	95.92	10.15	76	88	95	103	120	0.92
		Cohort 3-placebo	114	96.56	9.84	75	89	96	102	124	
		Cohort 2A	111	96.25	12.67	67	87	95	105	131	
		Total[f]	316	96.27	10.97	67	88	96	103	131	
	MUF_{cr} (mg/L)	Cohort 3-Ca	181	0.89	0.36	0.28	0.64	0.83	1.09	2.36	0.11
		Cohort 3-placebo	183	0.84	0.31	0.02	0.61	0.81	1.02	2.01	
		Cohort 2A	148	0.91	0.35	0.23	0.67	0.86	1.10	2.15	
		Total[f]	512	0.88	0.34	0.02	0.64	0.82	1.07	2.36	
	Maternal bone Pb (μg/g)	Cohort 3-Ca	97	7.07	7.26	0.01	0.83	4.36	11.78	26.22	<0.01
		Cohort 3-placebo	74	9.15	8.38	0.11	0.85	8.62	13.41	40.8	
		Cohort 2A	86	13.77	11.30	0.15	5.49	10.52	20.58	47.07	
		Total[f]	257	9.91	9.51	0.01	2.01	7.64	15.31	47.07	
	Maternal blood Hg (μg/L)	Cohort 3-Ca	55	3.03	1.41	0.51	2.12	2.77	3.62	7.06	0.09
		Cohort 3-placebo	48	2.87	2.09	0.34	1.82	2.37	3.34	13.47	
		Cohort 2A	104	4.06	4.88	0.77	2.14	3.10	4.16	35.91	
		Total[f]	207	3.51	3.70	0.34	2.07	2.80	3.79	35.91	
	CUF_{sg} (mg/L)	Cohort 3-Ca	104	0.84	0.39	0.31	0.56	0.75	1.07	2.80	0.227
		Cohort 3-placebo	84	0.90	0.46	0.35	0.58	0.75	1.09	2.89	
		Cohort 2A	96	0.79	0.34	0.18	0.53	0.73	0.92	2.11	
		Total[f]	284	0.84	0.40	0.18	0.57	0.74	1.00	2.89	

[a]Analysis of variance across cohorts.
[b]Total number of subjects included in GCI main analysis.
[c]Total number of subjects included in GCI sensitivity analysis.
[d]Total number of subjects included in IQ main analysis.
[e]Total number of subjects included in IQ sensitivity analysis.
[f]Total number of subjects with available measurements, combining Cohort 2A and Cohort 3.

Table 2. Analysis comparing subjects with and without data of interest [n (%) or mean ± SD] with respect to characteristics of mothers and children and sensitivity analysis covariates.

Characteristic	GCI analysis		IQ analysis	
	Included	Excluded	Included	Excluded
Total number[a]	287	710	211	786
Sex				
Female	160 (56%)	244 (47%)	116 (55%)	288 (48%)
Male	127 (44%)	275 (53%)	95 (45%)	307 (52%)
Birth order				
First child	96 (33%)	184 (35%)	93 (32%)	279 (36%)
≥2nd child	191 (67%)	335 (65%)	118 (68%)	507 (65%)
Birth weight (kg)	3.11 ± 0.45	3.11 ± 0.44	3.11 ± 0.46	3.11 ± 0.43
Gestational age (wk)	38.66 ± 1.84	38.58 ± 1.68	38.56 ± 1.80	38.63 ± 1.72
Age at outcome assessment (y)	4.04 ± 0.05	4.05 ± 0.05	8.50 ± 1.31	8.83 ± 1.64
Maternal age at delivery (y)	26.78 ± 5.53	26.49 ± 5.37	27.16 ± 5.61	26.41 ± 5.36
Maternal education (y)[b]	10.63 ± 2.76	10.75 ± 3.08	10.80 ± 2.85	10.69 ± 3.03
Maternal IQ[c]	88.63 ± 12.17	89.27 ± 14.6	89.01 ± 12.45	88.27 ± 13.00
Marital status[d]	3.11 ± 0.45	3.11 ± 0.44	3.11 ± 0.46	3.11 ± 0.43
Married	201 (70%)	493 (70%)	149 (71%)	544 (69%)
Other	86 (30%)	216 (30%)	62 (29%)	240 (31%)
Maternal smoking[e]				
Ever	141 (49%)	335 (51%)	102 (48%)	374 (51%)
Never	146 (51%)	325 (49%)	109 (52%)	362 (49%)
Cohort				
Cohort 3-Ca	93 (32%)	241 (34%)	76 (36%)	259 (33%)
Cohort 3-placebo	84 (29%)	252 (36%)	59 (28%)	278 (35%)
Cohort 2A	110 (38%)	217 (31%)	78 (37%)	249 (32%)
Sensitivity Analyses				
HOME score[f]	$N^{\dagger} = 138$	$N^{\ddagger} = 87$	$N^{\dagger} = 124$	$N^{\ddagger} = 55$
	35.24 ± 6.31	33.23 ± 6.55	35.54 ± 7.46	35.8 ± 7.44
SES[g]	$N^{\dagger} = 188$	$N^{\ddagger} = 110$	$N^{\dagger} = 199$	$N^{\ddagger} = 98$
	6.35 ± 2.43	6.94 ± 2.72	6.36 ± 2.41	6.98 ± 2.79
Maternal Bone Pb (μg/g)[h]	$N^{\dagger} = 167$	$N^{\ddagger} = 91$	$N^{\dagger} = 177$	$N^{\ddagger} = 80$
	9.26 ± 10.55	8.97 ± 10.32	9.02 ± 10.43	9.48 ± 10.55
Maternal Blood Hg (μg/L)[i]	$N^{\dagger} = 141$	$N^{\ddagger} = 67$	$N^{\dagger} = 149$	$N^{\ddagger} = 58$
	3.86 ± 4.25	2.76 ± 1.95	3.77 ± 4.16	2.83 ± 2.01
CUF_{sg}[j] (mg/L)			$N^{\dagger} = 124$	$N^{\ddagger} = 55$
			35.54 ± 7.46	35.8 ± 7.44

[a]The total number of subjects ($n = 997$) are all mother–offspring pairs who participated in the original Cohort 2A and Cohort 3 studies.
[b]Maternal education at the time of the child's birth.
[c]Maternal IQ measured at 6 mo after child's birth.
[d]Mother's marital status at the time of the child's birth.
[e]History of any maternal smoking.
[f]HOME score measured using the separate age-appropriate instruments pertaining to children of ≤5 y old; and children >5 y old.
[g]Family socioeconomic status (SES) measured by questionnaire of family possessions at follow-up.
[h]Maternal patella bone lead measured by KXRF after birth.
[i]Maternal average blood mercury during pregnancy.
[j]Children's specific gravity–corrected urinary fluoride measured at the time of each child's IQ test (6–12 y old).
$N^{\dagger}$ Number of subjects with measurements of MUF_{cr}, cognitive outcome, main covariates, and sensitivity covariates (they are included in the sensitivity model).
$N^{\ddagger}$ Number of subjects with measurements of sensitivity covariates, but missing data on exposure, outcomes, or main covariates (they are excluded from the sensitivity model).

age (CUF_{sg}), a measure that was collected in a significantly smaller subset of individuals, to evaluate the potential role of contemporaneous exposure. Associations between prenatal fluoride exposure (MUF_{cr}) and GCI at 4 y old could not be adjusted for contemporaneous fluoride exposure because urine samples were not collected from children when the MSCA (from which the GCI is derived) was administered. Maternal bone lead measured by a 109-Cd K-X-ray fluorescence (KXRF) instrument at 1 mo postpartum, a proxy for lead exposure from mobilized maternal bone lead stores during pregnancy (Hu et al. 2006), was included in the model to test for the possible confounding effect of lead exposure during pregnancy. We focused on the subset of women who had patella bone lead values because these were found to be most influential on our previous prospective study of offspring cognition (Gomaa et al. 2002). Average maternal mercury level during pregnancy was also tested for being a potential confounder (Grandjean and Herz 2011). Mercury was measured as total mercury content in the subsample of women who had samples of archived whole blood samples taken during pregnancy with sufficient volume to be analyzed using a Direct Mercury Analyzer 80 (DMA-80, Milestone Inc., Shelton, CT, USA) as previously described (Basu et al. 2014).

To address the potential confounding effect of socioeconomic status (SES) we conducted sensitivity analyses that adjusted our model for SES (family possession score). The socioeconomic questionnaire asked about the availability of certain items and assets in the home. Point values were assigned to each item, and SES was calculated based on the sum of the points across all items (Huang et al. 2016). Given that the calcium intervention theoretically could have modified the impact of fluoride, in examining our results, we repeated the analyses with and without the Cohort 3 participants who were randomized to the calcium intervention to omit any potential confounding effect of this intervention. Another sensitivity test was performed to examine the potential effect of the psychologist who performed the WASI test by including tester in the regression model. The information about psychologists who performed the WASI was available for 75% of participants, as recording this data was

Table 3. Distributions of maternal creatinine-adjusted urinary fluoride (MUF_{cr}) and offspring cognitive scores across categories of main covariates.

	GCI Analysis					IQ Analysis				
Covariate	*n*	MUF_{cr}[a]	*p*-Value	GCI (Age 4)	*p*-Value	*n*	MUF_{cr}[a]	*p*-Value	IQ (Age 6–12)	*p*-Value
Mothers										
Age										
≥25 y	123	0.88 ± 0.36	0.45	96.22 ± 14.12	0.50	88	0.89 ± 0.37	0.98	95.75 ± 11.64	0.80
<25 y	164	0.92 ± 0.36		97.37 ± 14.43		123	0.89 ± 0.35		96.15 ± 10.76	
Education										
<12 y	153	0.91 ± 0.4	0.92	94.22 ± 14.23	0.001	111	0.87 ± 0.37	0.53	93.09 ± 10.54	<0.001
12 y	97	0.89 ± 0.34		98.56 ± 14.46		70	0.93 ± 0.35		98.29 ± 10.72	
>12 y	37	0.89 ± 0.42		103.49 ± 11.21		30	0.85 ± 0.31		101.3 ± 11.16	
Marital status										
Married	201	0.90 ± 0.37	0.81	96.40 ± 14.46	0.39	62	0.90 ± 0.35	0.79	96.55 ± 11.06	0.63
Other	86	0.91 ± 0.33		98.00 ± 13.88		149	0.88 ± 0.36		95.74 ± 11.16	
Smoking										
Ever smoker	141	0.90 ± 0.36	0.80	97.77 ± 13.9	0.30	102	0.90 ± 0.36	0.56	97.21 ± 10.7	0.12
Nonsmoker	146	0.91 ± 0.35		96.01 ± 14.63		109	0.87 ± 0.35		94.83 ± 11.41	
HOME score [b]										
Mid-low ≤30	49	0.88 ± 0.37	0.47	90.73 ± 13.36	<0.001	32	0.87 ± 0.36	0.85	89.88 ± 8.45	0.011
High >30	137	0.92 ± 0.38		99.29 ± 14.61		92	0.88 ± 0.38		99.05 ± 11.65	
Maternal IQ										
Mid-low ≤85	116	0.95 ± 0.35	0.09	93.16 ± 15.04	<0.001	86	0.92 ± 0.36	0.23	91.26 ± 9.72	<0.001
High >85	171	0.87 ± 0.36		99.4 ± 13.21		125	0.86 ± 0.35		99.23 ± 10.87	
Children										
Sex										
Boy	127	0.94 ± 0.36	0.09	93.93 ± 13.98	0.002	95	0.96 ± 0.38	0.008	96.82 ± 12.02	0.32
Girl	160	0.87 ± 0.36		99.22 ± 14.12		116	0.83 ± 0.32		95.29 ± 10.31	
Birthweight										
≥3.5 kg	241	0.91 ± 0.36	0.57	96.52 ± 14.36	0.33	201	0.89 ± 0.36	0.88	95.66 ± 11.29	0.58
<3.5 kg	46	0.87 ± 0.35		98.76 ± 13.88		10	0.88 ± 0.34		97.38 ± 9.42	
Gestational age										
≤39 wk	192	0.90 ± 0.35	0.90	96.66 ± 14.23	716	146	0.89 ± 0.36	0.712	95.71 ± 11.62	0.65
>39 wk	95	0.90 ± 0.37		97.32 ± 14.46		65	0.88 ± 0.34		96.58 ± 9.91	
First child										
Yes	96	0.91 ± 0.38	0.75	99.97 ± 12.87	0.009	68	0.88 ± 0.36	0.91	97.00 ± 11.00	0.36
No	191	0.90 ± 0.35		95.32 ± 14.73		143	0.89 ± 0.36		95.50 ± 11.17	
CUF_{sg} [c]										
≥0.80 mg/L						112	0.86 ± 0.32	0.49	96.80 ± 11.16	0.37
<0.80 mg/L						77	0.90 ± 0.38		95.37 ± 10.31	

[a]Maternal creatinine-adjusted urinary fluoride (mg/L).
[b]Home Observation for the Measurement of the Environment (HOME) score, measured using the separate age-appropriate instruments pertaining to children of ≤5 y old; and children >5 y old.
[c]Child contemporaneous specific gravity–adjusted urinary fluoride (available at the time of each child's IQ test).

added later to the study protocol. We also re-ran models with exposure outliers included as a sensitivity step. Finally, we ran models that focused on the cross-sectional relationship between children's exposure to fluoride (reflected by CUF_{sg}) and IQ score, unadjusted; adjusting for the main covariates of interest; and adjusting for prenatal exposure (MUF_{cr}) as well as the covariates of interest.

Results

Flow of Participants

Of the 997 total mothers from two cohorts evaluated, 971 were eligible after removing mothers <18 y old. Of these 971, 825 had enough urine sample volume to measure fluoride in at least one trimester urine sample, and of these 825 participants, 515 participants had urine samples with previously measured creatinine values, enabling calculation of creatinine-adjusted urinary fluoride (MUF_{cr}) concentrations. Of these 515, 3 participants were excluded based on the 10 extreme outlier values identified for MUF_{cr} (see the "Methods" section, "Exposure Assessment" subsection) and not having any other MUF_{cr} values to remain in the analysis. Thus, we had a total of 512 participants (mothers) with at least one value of MUF_{cr} for our analyses (Figure 1).

Of these 512 mothers, 312 had offspring with outcome data at age 4 (i.e., GCI), and 234 had offspring with outcome data at age 6–12 (i.e., IQ). Of these, complete data on all the covariates of main interest (as specified in the "Methods" section) were available on 287 mother–child pairs for the GCI analysis and 211 mother–child pairs for the IQ analysis. A total of 299 mother–child pairs had data on either GCI or IQ, and 199 mother–child pairs had data on both GCI and IQ (Figure 1).

Number of Exposure Measures per Subject

In terms of repeated measures of MUF_{cr} across trimesters, of the 287 participants with data on GCI outcomes; 25 participants had MUF_{cr} data for all three trimesters (11 from Cohort 2A and 14 from Cohort 3), 121 participants had MUF_{cr} data from two trimesters (48 from Cohort 2A and 73 from Cohort 3), and 141 participants had MUF_{cr} data from one trimester (51 from Cohort 2A and 90 from Cohort 3). Of the 211 participants with data on IQ outcomes, 10 participants had MUF_{cr} data for all three trimesters (6 from Cohort 2A and 4 from Cohort 3), 82 participants had data from two trimesters (32 from Cohort 2A and 50 from Cohort 3), and 119 participants had data from one trimester (40 from Cohort 2A and 79 from Cohort 3).

Comparisons across the Cohorts

In terms of the mother–child pairs who had data on all covariates as well as data on either GCI or IQ ($n = 299$), the mean (SD)

Table 4. Multivariate regression models: unadjusted and adjusted differences in GCI and IQ per 0.5 mg/L higher maternal creatinine-adjusted urinary fluoride (MUF_{cr}).

	GCI			IQ		
Estimate	*n*	β (95%CI)	*p*-Value	*n*	β ± S.E (95%CI)	*p*-Value
Unadjusted	287	−3.76 (−6.32, −1.19)	<0.01	211	−2.37 (−4.45, −0.29)	0.03
model A[a]	287	−3.15 (−5.42, −0.87)	0.01	211	−2.50 (−4.12, −0.59)	0.01
Model A − HOME	138	−3.63 (−6.48, −0.78)	<0.01	124	−2.36 (−4.48, −0.24)	0.03
Model A + HOME	138	−3.76 (−7.08, −0.45)	0.03	124	−2.49 (−4.65, −0.33)	0.02
Model A − CUF_{sg}				189	−1.79 (−3.80, 0.22)	0.08
Model A + CUF_{sg}				189	−1.73 (−3.75, 0.29)	0.09
Model A − SES	188	−4.55 (−7.23, −1.88)	0.01	199	−2.10 (−4.02, −0.18)	0.03
Model A + SES	188	−4.45 (−7.08, −1.81)	0.01	199	−2.10 (−4.06, −0.15)	0.04
Model A − Pb	167	−5.57 (−8.48, −2.66)	<0.01	177	−3.21 (−5.17, −1.24)	<0.01
Model A + Pb	167	−5.63 (−8.53, −2.72)	<0.01	177	−3.22 (−5.18, −1.25)	<0.01
Model A − Hg	141	−7.13 (−10.26, −4.01)	<0.01	149	−4.59 (−7.00, −2.17)	<0.01
Model A + Hg	141	−7.03 (−10.19, −3.88)	<0.01	149	−4.58 (−6.99, −2.16)	<0.01
Model A − Ca	194	−3.67 (−6.57, −0.77)	0.01	136	−3.23 (−5.88, −0.57)	0.02

[a]Coefficients from linear regression models adjusted for gestational age, weight at birth, sex, parity (being the first child), age at outcome measurement, and maternal characteristics including smoking history (ever smoked during the pregnancy vs. nonsmoker), marital status (married vs. others), age at delivery, IQ, education, and cohort (Cohort 3-Ca, Cohort 3-placebo and Cohort 2A). Model A–HOME, model A for subset of cases who have data on Home Observation for the Measurement of the Environment (HOME) scores (but the model did not include HOME score). Model A + HOME, model A for subset of cases with HOME score, adjusted for HOME score. Model A − CUF_{sg}, model A for subset of cases who have data on child contemporaneous specific gravity–adjusted urinary fluoride CUF_{sg} (but the model did not include CUF_{sg}). Model A + CUF_{sg}, model A for subset of cases with CUF_{sg}, adjusted for CUF_{sg}. Model A−SES, model A for subset of cases who have data on socioeconomic status (family possession measured by questionnaire of family possessions) (but the model did not include SES). Model A + SES, model A for subset of cases with SES data, adjusted for SES. Model A–Pb, model A for subset of cases who have data on maternal bone lead (but the model did not include maternal bone lead). Model A + Pb, model A for subset of cases with data on maternal bone lead, adjusted for maternal bone lead. Model A−Hg, model A for subset of cases who have data on maternal blood mercury (but the model did not include maternal blood mercury). Model A + Hg, model A for subset of cases who have data on maternal blood mercury, adjusted for maternal blood mercury. Model A − Ca, model A for subset of cases who did not receive the Ca supplement (they received the placebo).

values of creatinine–corrected urinary fluoride for the mothers was 0.90 (0.36) mg/L. The distributions of the urinary fluoride, outcomes (GCI and IQ), and additional exposure variables examined in our sensitivity analyses (maternal bone lead, maternal blood mercury, and children's contemporaneous urinary fluoride) across the three cohort strata (Cohort 3-Calcium, Cohort 3-placebo, and Cohort 2A) and all strata combined are shown in Table 1 for the mother–child pairs who had data for the GCI outcome ($n = 287$) and the IQ outcome ($n = 211$). The distributions showed little variation across the cohort strata except for bone lead and possibly blood mercury, for which, in comparison with Cohort 3, Cohort 2A clearly had higher mean bone lead levels ($p < 0.001$) and possibly higher blood mercury levels ($p = 0.067$). The mean (SD) values of specific gravity–corrected urinary fluoride for the children who had these measures (only available for those children who had IQ; $n = 189$) were 0.82 (0.38) mg/L.

In terms of the comparability of the participants across Cohort 2A and Cohort 3 with respect to our covariates, the distribution of the covariates was very similar with the exception of age of the offspring when IQ was measured, for which the mean ages were 7.6 and 10.0 y, respectively; and birth weight in the GCI analysis, for which Cohort 3 participants were slightly heavier than Cohort 2 participants (see Table S1).

GCI versus IQ Scores

There was a significant correlation between GCI at 4 y and IQ at 6–12 y old (Spearman $r = 0.55$; $p < 0.01$). There was no significant correlation between prenatal MUF_{cr} and offspring CUF_{sg} (Spearman $r = 0.54$, $p = 0.44$).

Comparisons of Participants in Relation to Missing Data

In comparing the participants who were included for the GCI and IQ analyses with the participants who were not included (based on data missing on GCI, IQ or other covariates), the distribution of covariates were similar except for sex, for which the proportion of females was somewhat higher in the included versus excluded group for both the GCI and IQ analyses (Table 2).

In terms of the sensitivity analyses, for each sensitivity variable of interest, we compared participants who had data on our exposures, outcomes, covariates, and the sensitivity variable of interest (and were thus included in the sensitivity analysis) versus participants who had data on the sensitivity variable of interest but were missing data on the exposure, outcomes, and/or covariates of interest (and were thus excluded from the sensitivity analysis; Table 2). It can be seen that for each sensitivity analysis, most of the participants with data on the sensitivity variable of interest also had data on the exposures, outcomes, and covariates and were therefore included in the sensitivity analysis. In addition, the distributions appeared to be similar comparing those included with those excluded in each sensitivity analysis (means were within 10% of each other), with the exception of maternal blood Hg, for which the mean levels for those included were 28.5% and 24.9% higher than the mean levels for those excluded in the GCI and IQ analyses, respectively.

Comparisons of GCI and IQ across Covariates

Table 3 shows mean and SD values for MUF_{cr} and offspring cognitive scores across categories of the covariates. In the participants with GCI data, the offspring cognitive scores were higher among mothers with higher levels of education, measured IQ, and HOME scores for both analyses; and scores were higher among first children and girls. In the IQ analysis a statistically significant difference was observed in MUF_{cr} as a function of child sex. No significant differences in MUF_{cr} values across levels of other covariates were observed. A modest difference (not statistically significant), was observed in MUF_{cr} as a function of maternal IQ ($p = 0.09$), and MUF_{cr} as a function of child sex ($p = 0.09$). Among other co-variates there were significant differences in age ($p < 0.01$) in both analyses.

Regression Models of GCI

Before adjustment, a 0.5 mg/L increase in MUF_{cr} was negatively associated with GCI at 4 y old [mean score −3.76; 95% confidence interval (CI): −6.32, −1.19] (Table 4). The association was somewhat attenuated after adjusting for the main covariates

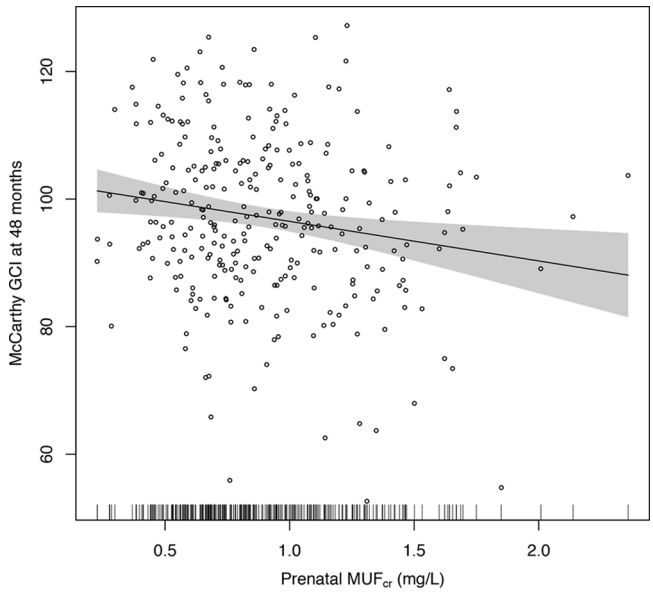

Figure 2. Adjusted association of maternal creatinine-adjusted urinary fluoride (MUF_{cr}) and General Cognitive Index (GCI) scores in children at age 4 y. Adjusted for gestational age, weight at birth, sex, parity (being the first child), age at outcome measurement, and maternal characteristics including smoking history (ever smoked vs. nonsmoker), marital status (married vs. others), age at delivery, IQ, education, and cohort (Cohort 3-Ca, Cohort 3-placebo and Cohort 2A). Shaded area is 95% confidence interval. Short vertical bars on the *x*-axis reflect the density of the urinary fluoride measures. Individual data points are individual observations, $n = 287$.

(model A, −3.15; 95% CI: −5.42, −0.87). The smooth plot of the association between GCI and maternal prenatal urinary fluoride from an adjusted GAM model suggested a linear relation over the exposure distribution (Figure 2).

Regression Models of IQ

A 0.5 mg/L increase in prenatal fluoride was also negatively associated with IQ at age 6–12 y based on both unadjusted (−2.37; 95% CI: −4.45, −0.29) and adjusted models (−2.50; 95% CI: −4.12, −0.59) (Table 4). However, estimates from the adjusted GAM model suggest a nonlinear relation, with no clear association between IQ scores and values below approximately 0.8 mg/L, and a negative association above this value (Figure 3A). There was a nonsignificant improvement in the fit of the model when a quadratic term was added to the linear model ($p = 0.10$).

Sensitivity Analyses

In sensitivity analyses, adjustment for HOME score increased the magnitude of the association between MUF_{cr} and GCI, though the difference was less pronounced when associations with and without adjustment for HOME score were both estimated after restricting the model to the subset of 138 children with HOME score data (Table 4). The association of IQ scores with MUF_{cr} did not substantially change after adding HOME score to the model (Table 4).

The association between MUF_{cr} and IQ was attenuated slightly after adjusting for contemporaneous children's urinary fluoride (CUF_{sg}) and comparing estimates with [−1.73 (95% CI: −3.75, 0.29)] and without [−1.94 (95% CI: −4.15, 0.26)] adjustment for CUF_{sg} among the 189 children with this data (Table 4). In addition, the evidence of nonlinearity was more pronounced, with no clear evidence of an association for MUF_{cr} <1.0 mg/L

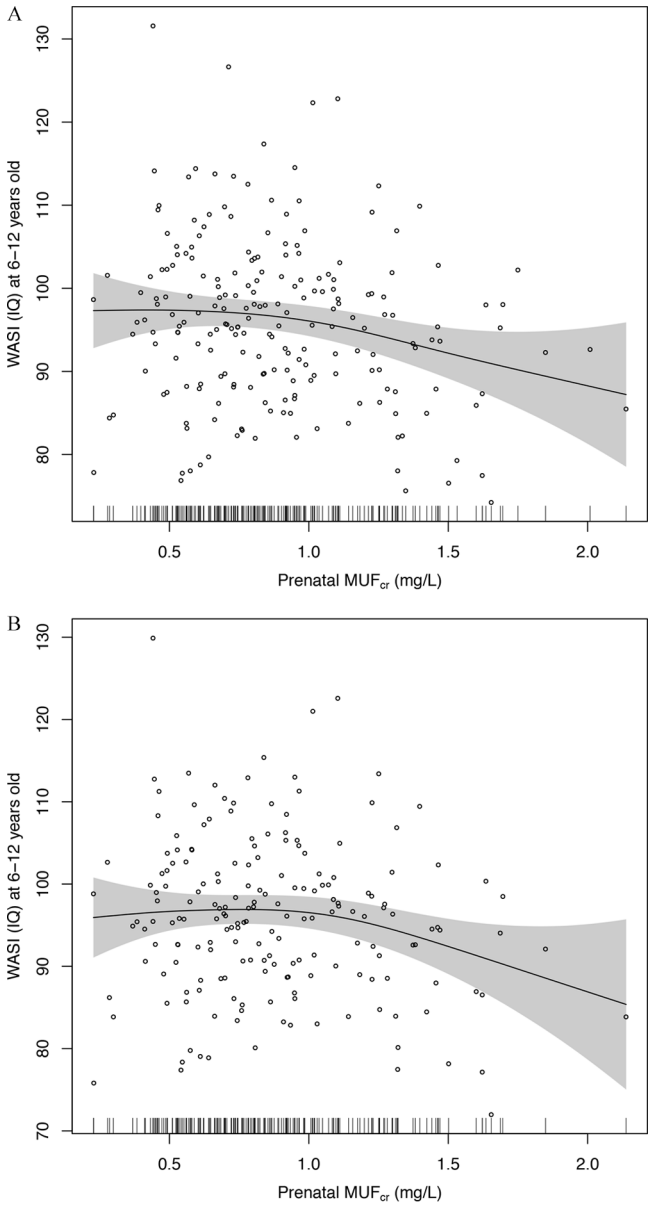

Figure 3. (A) Adjusted association of maternal creatinine-adjusted urinary fluoride (MUF_{cr}) and children's IQ at age 6–12 y. Adjusted for gestational age, weight at birth, sex, parity (being the first child), age at outcome measurement, and maternal characteristics including smoking history (ever smoked vs. nonsmoker), marital status (married vs. others), age at delivery, IQ, education, and cohort (Cohort 3-Ca, Cohort 3-placebo and Cohort 2A). Short vertical bars on the *x*-axis reflect the density of the urinary fluoride measures. Individual data points are individual observation, $n = 211$. (B) Association of maternal creatinine-adjusted urinary fluoride ($MUFU_{cr}$) and children's IQ at age 6–12 y, adjusted for specific gravity–adjusted child urinary fluoride (CUF_{sg}). Adjusted for gestational age, weight at birth, sex, parity (being the first child), age and CUF_{sg} at outcome measurement, and maternal characteristics including smoking history (ever smoked vs. nonsmoker), marital status (married vs. others), age at delivery, IQ, education. and cohort (Cohort 3-Ca, Cohort 3-placebo and Cohort 2A). Shaded area is 95% confidence interval. Short vertical bars on the *x*-axis reflect the density of the urinary fluoride measures. Individual data points are individual observation, $n = 189$.

based on the GAM model (Figure 3B), and a significant improvement in model fit when a quadratic term was added to the linear regression model ($p = 0.01$).

When we restricted models to subsets of children with available data for each additional covariate, there was little difference

between adjusted and unadjusted associations between MUF_{cr} and GCI or IQ when socioeconomic status (family possession), maternal bone lead, and blood mercury, were added to models (Table 4). However, the effect estimates associated with MUF_{cr} for these analyses appear to be higher in the subsets with available data for these variables.

Adding tester (psychologist who performed WASI) in the model did not substantially change the results (data not shown). In the sensitivity analyses in which we excluded Cohort 3 participants who received the calcium supplement, we continued to observe a negative association between MUF_{cr} and GCI [0.5 mg/L increase in MUF_{cr} associated with 3.67 lower GCI (95% CI: −6.57, −0.77), $n = 194$]; and between MUF_{cr} and IQ [0.5 mg/L increase in MUF_{cr} associated with 3.23-lower IQ (95% CI: −5.88, −0.57), $n = 136$].

In sensitivity analyses in which we re-ran models that included the 10 outliers with respect to fluoride exposure (for each of seven participants already in our models, an additional value of MUF_{cr} [from a different trimester]; for three participants, a value of MUF_{cr} that then allowed the participants to be added to our models), the results did not change in any meaningful way (data not shown). There were no outliers with respect to cognitive outcomes.

Independent Influence of Child Fluoride Exposure

Finally, in models that focused on the cross-sectional relationship between children's exposure to fluoride (reflected by their specific gravity–adjusted urinary fluoride levels) and IQ score and that contained the main covariates of interest, there was not a clear, statistically significant association between contemporaneous children's urinary fluoride (CUF_{sg}) and IQ either unadjusted or adjusting for MUF_{cr}. A 0.5 mg/L increase in CUF_{sg} was associated with a 0.89 lower IQ (95% CI: −2.63, 0.85) when not adjusting for MUF_{cr}; and 0.77-lower IQ (95% CI: −2.53, 0.99), adjusting for MUF_{cr} ($n = 189$).

Discussion

In our study population of Mexican women and children, which accounted for two of the three cohorts included in the ELEMENT study, higher prenatal exposure to fluoride (as indicated by average creatinine-adjusted maternal urinary fluoride concentrations during pregnancy) was associated with lower GCI scores in children at approximately 4 y old, and with lower Full-Scale IQ scores at 6–12 y old. Estimates from adjusted linear regression models suggest that mean GCI and IQ scores were about 3 and 2.5 points lower in association with a 0.5 mg/L increase in prenatal exposure, respectively. The associations with GCI appeared to be linear across the range of prenatal exposures, but there was some evidence that associations with IQ may have been limited to exposures above 0.8 mg/L. In general, the negative associations persisted in sensitivity analyses with further adjustment for other potential confounders, though the results of sensitivity analyses were based on subsets of the population with available data.

Overall, our results are somewhat consistent with the ecological studies suggesting children who live in areas with high fluoride exposure (ranging from 0.88 to 11.0 mg/L fluoride in water, when reported) have lower IQ scores than those who live in low-exposure or control areas (ranging from 0.20 to 1.0 mg/L fluoride in water) (Choi et al. 2012) and with results of a pilot study of 51 children (mean age 7 y) from southern Sichuan, China, that reported that children with moderate or severe dental fluorosis (60% of the study population) had lower WISC-IV digit span scores than other children (Choi et al. 2015). A distinction is that our study, which was longitudinal with repeated measures of exposure beginning in the prenatal period, found associations with respect to prenatal fluoride exposures.

To our knowledge, the only other study that is similar to ours was only recently published. Valdez Jiménez et al. (2017) studied the association of prenatal maternal urinary fluoride levels (not corrected for dilution) and scores on the Bayley Scales of Infant Development II among 65 children evaluated at age 3–15 mo (average of 8 mo). The mothers in their study had urinary fluoride levels of which the means at each of the three trimesters of pregnancy (1.9, 2.0, 2.7 mg/L) were higher than the mean MUF_{cr} in our participants (0.88 mg/L) (Valdez Jiménez et al. 2017). These levels of exposure were found to be associated with statistically significantly lower scores on the Bayley Scales' Mental Development Index (MDI) score after adjusting for gestational age, age of child, a marginality index, and type of drinking water (Valdez Jiménez et al. 2017). By comparison, our study had much longer periods of follow-up and larger sample sizes, controlled for a much larger set of covariates and sensitivity variables, and used creatinine–corrected urinary fluoride measures (which, by adjusting for urinary dilution effects, provides a more reliable measure of internal fluoride exposure).

With respect to understanding the generalizability of our findings to other populations, there are very few studies that measured prenatal fluoride levels among women derived from population-based samples. Gedalia et al. (1959) measured urinary fluoride in multiple samples collected from each of 117 healthy pregnant women living in Jerusalem, where fluoride in the water was approximate 0.50 mg/L, and reported mean levels per person that ranged from 0.29 to 0.53 mg/L. However, these analysis were not conducted utilizing modern analytical techniques. In a study of 31 pregnant women living in Poland, Opydo-Szymaczek and Borysewicz-Lewicka (2005) measured urinary fluoride in healthy pregnant women patients of a maternity hospital in Poland, where fluoride in the water ranged from 0.4 to 0.8 mg/L, and found a mean level of 0.65 mg/L for women in their 28th week of pregnancy, 0.84 mg/L in their 33rd week, and 1.30 mg/L in healthy non-pregnant women of similar age. This would suggest that the mothers in our study, who had a mean MUF_{cr} value of 0.90 mg/L, had fluoride exposures slightly higher than prior-mentioned populations.

In terms of comparing our findings with other studies of fluoride (using urinary fluoride as a biomarkers of exposure) and intelligence (i.e., those not involving prenatal exposures), of the 27 epidemiologic studies on fluoride and IQ reviewed by Choi et al. in their 2012 meta-analysis, only 2 had measures of urinary fluoride. Both were of urinary fluoride measures in children (not pregnant mothers), and neither corrected for dilution (either by correcting for urinary creatinine or specific gravity). Of these two, in comparison with the urinary fluoride levels of both our mothers (0.88 mg/L) and our children (0.82 mg/L), the mean levels of children's urinary fluoride were higher in the non-fluorosis (1.02 mg/L) and high-fluorosis (2.69 mg/L) groups found by Li et al. (1995) as well as the control (1.5 mg/L) and high-fluorosis (5.1 mg/L) groups described by Wang et al. (2007).

Among the limitations of our study are that we measured fluoride in spot (second morning void) urine samples instead of 24-hr urine collections. However, others have noted a close relationship between the fluoride concentrations of early morning samples and 24-hr specimens (Watanabe et al. 1994; Zohouri et al. 2006). Another limitation relates to the potential differences in the distribution of covariates over our study cohorts, raising the issue of potential bias. In the analyses we conducted across cohorts, we saw that, in comparison with Cohort 3, Cohort 2A clearly had

higher mean bone lead levels ($p < 0.001$) and possibly higher blood mercury levels ($p = 0.067$). However, we saw no other differences and the differences in these measures have a clear likely explanation: Cohort 2A had bone lead levels measured in 1997–2001 and Cohort 3 had bone lead levels measured in 2001–2005. Given that environmental lead and mercury exposures were steadily decreasing during this time interval (due to the phase-out of lead from gasoline), this difference likely relates to an exposure–time–cohort effect. We do not anticipate that this phenomenon would have introduced a bias in our analyses of fluoride and cognition controlling for bone lead.

Another limitation relates to the missing data that pertain to our covariate and sensitivity variables. In the comparisons of participants in relation to missing data (Table 2A,B), the proportion of females was somewhat higher in the included versus excluded group for both the GCI and IQ analyses, and the mean levels of maternal blood Hg for those included were 28.5% and 24.9% higher than the mean levels for those excluded in the GCI and IQ analyses, respectively. We also note that the coefficients for the associations between fluoride on cognition varied substantially in some of the sensitivity analyses, particularly with respect to the subgroups of participants who have data on SES, lead exposure, and mercury exposure (of which, for the latter, the effect estimates almost doubled). We do not have a ready explanation for this phenomenon, given that there is no obvious way that each of the selection factors governing which mothers had these measurements (discussed above) could have influenced the fluoride–cognition relationship. Nevertheless, it is not possible to entirely rule out residual confounding or in the population as a whole (that might have been detected had we had full data on larger sample sizes) or bias (should the subpopulations that had the data for analysis have a different fluoride–cognition relationship than those participants who were excluded from the analyses).

Other limitations include the lack of information about iodine in salt, which could modify associations between fluoride and cognition; the lack of data on fluoride content in water given that determination of fluoride content is not reported as part of the water quality monitoring programs in Mexico; and the lack of information on other environmental neurotoxicants such as arsenic. We are not aware of evidence suggesting our populations are exposed to significant levels of arsenic or other known neurotoxicants; nevertheless, we cannot rule out the potential for uncontrolled confounding due to other factors, including diet, that may affect urinary fluoride excretion and that may be related to cognition.

Another potential limitation is that we adjusted maternal urinary fluoride levels based on urinary creatinine, whereas we adjusted children's urinary fluoride levels based on urinary specific gravity; however, these two methods are almost equivalent in their ability to account for urinary dilution. We also had no data to assess the inter-examiner reliability of the testers administering the WASI test; however, the excellent reliability of these same testers in administering the McCarthy tests provides some reassurance that the WASI tests were conducted in a consistent manner.

Finally, our ability to extrapolate our results to how exposures may impact on the general population is limited given the lack of data on fluoride pharmacokinetics during pregnancy. There are no reference values for urinary fluoride in pregnant women in the United States. The Centers for Disease Control and Prevention has not included fluoride as one of the population exposures measured in urine or blood samples in its nationally representative sampling. The WHO suggests a reference value of 1 mg/L for healthy adults when monitoring renal fluoride excretion in community preventive programs (Marthaler 1999). As part of the NRC's review of the fluoride drinking-water standard, it was noted that healthy adults exposed to optimally fluoridated water had urinary fluoride concentrations ranging from 0.62 to 1.5 mg/L.

Conclusion

In this study, higher levels of maternal urinary fluoride during pregnancy (a proxy for prenatal fluoride exposure) that are in the range of levels of exposure in other general population samples of pregnant women as well as nonpregnant adults were associated with lower scores on tests of cognitive function in the offspring at 4 and 6–12 y old.

Community water and salt fluoridation, and fluoride toothpaste use, substantially reduces the prevalence and incidence of dental caries (Jones et al. 2005) and is acknowledged as a public health success story (Easley 1995). Our findings must be confirmed in other study populations, and additional research is needed to determine how the urine fluoride concentrations measured in our study population are related to fluoride exposures resulting from both intentional supplementation and environmental contamination. However, our findings, combined with evidence from existing animal and human studies, reinforce the need for additional research on potential adverse effects of fluoride, particularly in pregnant women and children, and to ensure that the benefits of population-level fluoride supplementation outweigh any potential risks.

Acknowledgments

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Washington State Board of Health

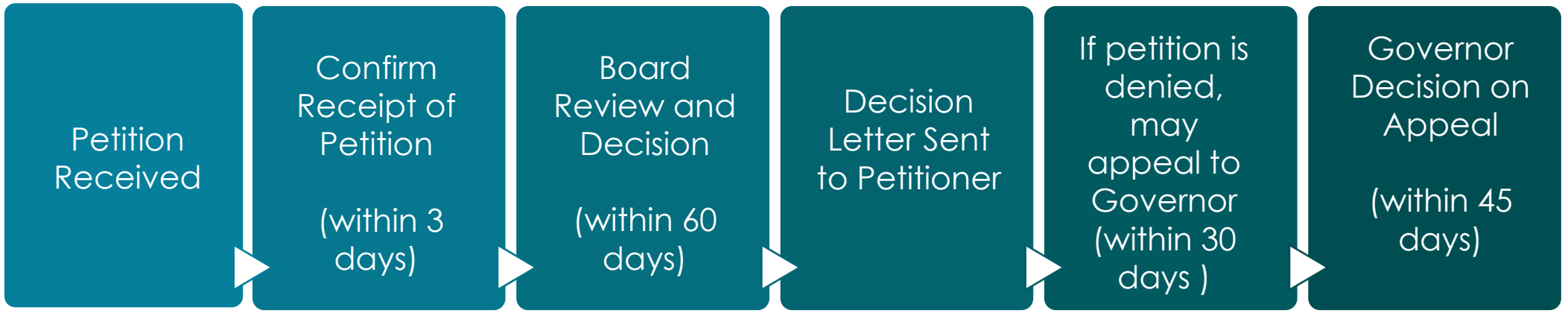
Rulemaking Petition, Group A Public Water Supplies, Drinking Water Materials and Additives, WAC 246-290-220

March 13, 2024

Background

Under the Administrative Procedures Act (RCW 34.05.330), any person may petition a state agency to adopt, repeal, or amend any rule within its authority.

Overview of the Board's Petition Process:



Board Authority

- RCW 43.20.050 requires the Board to adopt rules for group A public water systems necessary to assure safe and reliable drinking water and to protect the public health.
- Chapter 246-290 WAC establishes the standards for these water systems.
- **WAC 246-290-220**

Requires testing and certification for conformance with NSF/ANSI Standards 60 and 61 for:

- Treatment chemicals added to public drinking water supplies; and
- Public water system components in substantial contact with potable water such as water pipes, tank coatings or liners, and treatment system media.

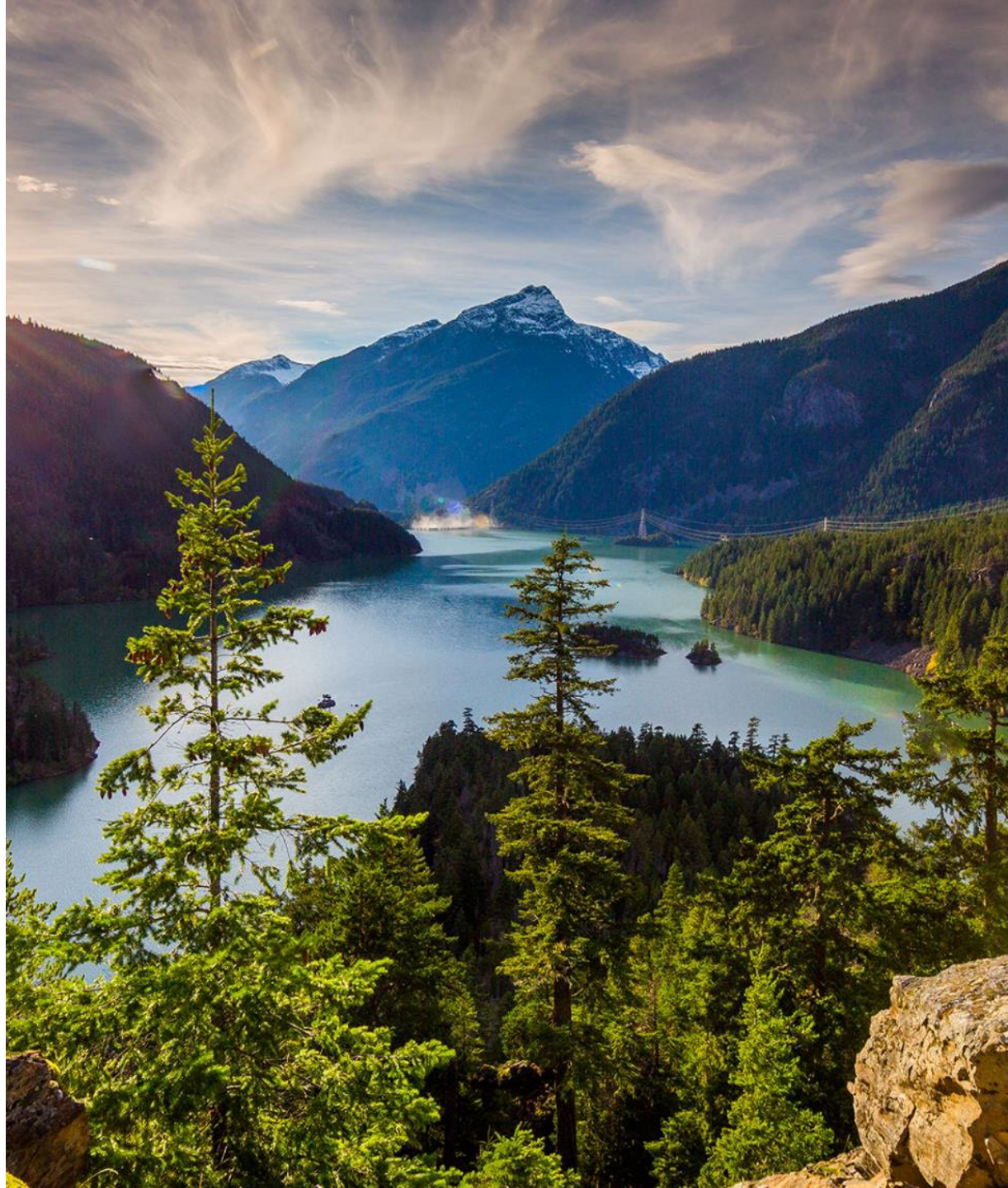
Petition Request

- On February 12, 2024, the Board received a petition for rulemaking to amend WAC 246-290-220.
- The request:
    - Amend WAC 246-290-220 to include:
        - (8) For the safety of the developing fetus, infant, and child, the board no longer endorses the addition of fluoride to public water and recommends reducing fluoride exposure for pregnant mothers, infants, and children under 6 years of age.

          (a) Pregnant mothers and women planning to become pregnant (within 10 years) should limit fluoride ingestion by usually drinking water and liquids with less than 0.2 mg/L of fluoride, and do not swallow toothpaste;

          (b) Caregivers of infants should use water as low in fluoride as practical, less than 0.2 mg/L, for making infant formula, juice, and drinking, and do not use fluoridated toothpaste.15

          (c) Carefully supervise children when they are using fluoridated dental products, such as toothpaste, to assure they are not swallowing the toothpaste and are able to spit, rinse and spit, and again rinse and spit without first swallowing. Read and follow the toothpaste label.



Petitioner's Rationale

- The petitioner claims that the Board is in violation of RCW 43.20.050 and other laws, to assure safe drinking water.
- This petition is focused on a minimum label to protect the development of the most vulnerable, i.e. fetus, infant, and child.
- One of the goals of the petition is to start to "educate the public for their safety."
- The intent of the petitioner's request for amending the rule, as stated in the petition is "to start protecting fetuses, infants, and children from the most significant risks and harm of fluoride exposure."

Water Fluoridation

- The Department of Health supports community water fluoridation as a sound, population-based public health measure. Community water fluoridation is a proven public health prevention measure that benefits both children and adults, regardless of age, race, gender, or income. The department encourages communities to begin and maintain optimal fluoride levels for health benefits in drinking water systems.
- Organizations that recommend the Fluoridation of Public Water Systems and Recognize the Public Benefits:
    - The Washington State Board of Health (WSBOH)
    - The American Water Works Association (AWWA)
    - World Health Organization (WHO),
    - American Medical Association (AMA),
    - Canadian Medical Association (CMA),
    - Centers for Disease Control (CDC),
    - American Dental Association (ADA),
    - Canadian Dental Association (CDA)

Child & Maternal Health

- Maintaining good oral health is essential to maintain a healthy pregnancy. [9]
- The American Academy of Pediatrics has published educational materials focused on optimal fluoride for children that says, "It is safe to use fluoridated water to mix the formula if your baby is younger than 6 months old, but there is a small risk of "fluorosis."
- The CDC also provides information on the safety of fluoridated water for use in infant formula. [10]
- There may be an increased chance of mild fluorosis when mixing infant formula with fluoridated water. [10]

Resources

1. Recommended Strategies to Improve the Oral Health of Washington Residents | SBOH
2. 2023DOHFluorideSupportStatement.pdf (wa.gov)
3. Oral Health Equity Assessment (wa.gov)
4. Fluoride in Water | What You Need To Know About Fluoride (ilikemyteeth.org)
5. Fluoridation (ca.gov)
6. Task Force Recommends Fluoride to Prevent Dental Caries | AAFP
7. Promoting Oral Health through Water Fluoridation | FDI (fdiworlddental.org):
8. Fluoridation of Public Water Supplies | American Water Works Association (awwa.org)
9. AAP continues to recommend fluoride following new study on maternal intake and child IQ | AAP News | American Academy of Pediatrics
10. Infant Formula | FAQs | Community Water Fluoridation | Division of Oral Health | CDC

WASHINGTON STATE BOARD OF HEALTH

2024 Meeting Schedule

Approved by the Board November 8, 2023
Updates approved by the Board January 10, 2024 (to hold April meeting)
Location updates discussed January 10 and March 13, 2024

	Meeting Date	Location
Board	**Wednesday January 10, 2024**	**Hybrid:** • **Physical Location; Washington State Department of Health, 111 Israel Road S.E., Tumwater, WA 98501, Building: Town Center 2, Rooms 166 & 167** • **Virtual Meeting via ZOOM Webinar; hyperlink provided on website and agenda. Public Attendees can pre-register and access the meeting online.**
Board	**Wednesday March 13, 2024**	**Hybrid:** • **Physical Location; Swinomish Casino and Lodge, 12885 Casino Dr, Anacortes, WA 98221, WA Walton Conference Room** • **Virtual Meeting via ZOOM Webinar; hyperlink provided on website and agenda. Public Attendees can pre-register and access the meeting online.**
Board	**Wednesday April 10, 2024**	**Hybrid:** • **Physical Location; Spokane Public Library, 906 W. Main Ave, Spokane, WA, 99201 Rooms: Central Events A & B** • **Virtual Meeting via ZOOM Webinar; hyperlink provided on website and agenda. Public Attendees can pre-register and access the meeting online.**
Board	**Wednesday June 12, 2024**	**Hybrid:** • **Physical Location; Vancouver, WA (location TBD)** • **Virtual Meeting via ZOOM Webinar; hyperlink provided on website and agenda. Public Attendees can pre-register and access the meeting online.** *(note: WA State Association of Local Public Health Officials (WSALPHO) Annual meeting is in Spokane, June 4-6, 2024)*
Board	**Wednesday July 10, 2024**	**Hold date – meet only if necessary**

Board	**Wednesday August 14, 2024**	**Hybrid:** • **Physical Location; To Be Determined (TBD), Walla Walla, Tri-Cities or Ellensburg** • **Virtual Meeting via ZOOM Webinar; hyperlink provided on website and agenda. Public Attendees can pre-register and access the meeting online.**
Board	**Tuesday October 8, 2024**	**Hybrid:** • **Physical Location; Yakima (meeting space TBD)** • **Virtual Meeting via ZOOM Webinar; hyperlink provided on website and agenda. Public Attendees can pre-register and access the meeting online.** *(note: WA State Public Health Association (WSPHA) Annual conference is in Yakima, October 9-11, 2024. The WSALPHO Environmental Public Health Directors meeting is Oct 1-4 in Leavenworth)*
Board	**Wednesday November 13, 2024**	**Hybrid:** • **Physical Location; Tumwater, WA** • **Virtual Meeting via ZOOM Webinar; hyperlink provided on website and agenda. Public Attendees can pre-register and access the meeting online.**

Start time is 9:30 a.m. unless otherwise specified. Time and locations subject to change as needed. See the Board of Health Web site and the Health Disparities Council Web site for the most current information.

Last updated 01/10/2024