WASHINGTON STATE BOARD OF HEALTH

Notice of Public Meeting

Wednesday, August 7, 2024, 9:30 a.m. – 4:00 p.m.
Physical meeting location:
Pacific Tower
Panoramic Center
1200 12th Avenue South, Suite 810
Seattle, WA 98144
Virtual meeting: ZOOM Webinar
(hyperlink provided below)
Language interpretation available

Final Agenda

Time	Agenda Item	Speaker
9:30 a.m.	Call to Order & Introductions	Kelly Oshiro, Board Vice Chair
9:35 a.m.	1. Approval of Agenda – Possible Action	Kelly Oshiro, Board Vice Chair
9:40 a.m.	2. Approval of June 12, 2024, Minutes –Possible Action	Kelly Oshiro, Board Vice Chair
9:45 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.
10:05 a.m.	4. Announcements and Board Business	Michelle Davis, Board Executive Director
10:25 a.m.	5. Seattle & King County Local Health Update: Public Health Approach to Overdose Prevention	Brad Finegood, Seattle & King County Public Health Ashley Bell, Board Staff
10:45 a.m.	6. Yakima Water Recreation Variance Request, Chapters 246-260 & 262 WAC	Kate Dean, Board Member Andrew Kamali, Board Staff Dave DeLong, Department Staff
11:15 a.m.	Break	
11:30 a.m.	7. Cheney Water Recreation Variance Requests, Chapters 246-260 & 262 WAC	Kate Dean, Board Member Andrew Kamali, Board Staff Dave DeLong, Department Staff

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Time	Agenda Item	Speaker
12:00 p.m.	8. Rulemaking Petition – WAC 246-272A-0240, Holding Tank Sewage Systems – On-Site Sewage – Possible Action	Kate Dean, Board Member Shay Bauman, Board Staff Jeremy Simmons, Department Staff
12:15 p.m.	Lunch	
1:00 p.m.	9. Health Impact Reviews – Fiscal Year 2024 Update	Lindsay Herendeen, Board Staff Cait Lang-Perez, Board Staff Miranda Calmjoy, Board Staff
1:30 p.m.	10. Rulemaking Petition – Chapter 246-650 WAC, Newborn Screening, Request to Add Wilson's Disease – Possible Action	Kelly Oshiro, Board Vice Chair Kelly Kramer, Board Staff John Thompson, Department Staff
1:50 p.m.	11. School Rule Review Project Update	Kate Dean, Board Member Andrew Kamali, Board Staff
2:10 p.m.	Break	
2:25 p.m.	12. Pro-Equity Anti-Racism (PEAR) Plan Briefing	Paj Nandi, Board Member Ashley Bell, Board Staff
3:10 p.m.	13. Rules Briefing – Addition of Ornithine Transcarbamylase Deficiency (OTCD), Guanidinoacetate methyltransferase (GAMT) deficiency, and Arginase 1 Deficiency (ARG1-D), Chapter 246-650 WAC, Newborn Screening	Kelly Oshiro, Board Vice Chair Kelly Kramer, Board Staff John Thompson, Department Staff

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Time	Agenda Item	Speaker
3:40 p.m.	14. Board Member Comments and Updates	
4:00 p.m.	Adjournment	

- **To access the meeting online and to register:**
  https://us02web.zoom.us/webinar/register/WN_Zr68w8-KTU2AcaKkY87nAg
- **You can also dial-in using your phone for listen-only mode:**
  Call in: +1 (253) 215-8782 (not toll-free)
  Webinar ID: 861 3961 5310
  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.

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Information About Giving Verbal Public Comment at Hybrid Meetings:

- Individuals may give verbal public comments at the meeting, in-person or virtually, during the public comment period.
- The amount of time allotted to each person will depend on the number of speakers present (typically 1 to 3 minutes per person). We will first call on those who have signed up in advance.
- Sign up **by 12:00 Noon the day before a meeting** to participate in the public comment period:
  - Email the Board or
  - Register through the **Zoom webinar link. The Zoom webinar link is in the meeting agenda located on the** Meeting Information webpage**.**
  - If you are **attending the meeting in person** and did not sign up in advance, you may write your name on the sign-in sheet to provide comments if time allows.

Information About Giving Written Public Comment:

- Please visit the Board's Public Comment webpage for details.

WASHINGTON STATE BOARD OF HEALTH

Draft Minutes of the State Board of Health
June 12, 2024
Hybrid Meeting
ASL (or CART) and Spanish interpretation available
Heathman Lodge
7801 NE Greenwood Drive
Vancouver, WA 98662
Meeting Rooms: Chinook & Klickitat
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, MD
Mindy Flores, MHCM
Paj Nandi, MPH
Dimyana Abdelmalek, MD, MPH
Michael Ellsworth, JD, MPA, 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
Heather Carawan, Communications Consultant
Molly Dinardo, Health Policy Advisor
Shay Bauman, Health Policy Advisor
Jo-Ann Huynh, Administrative Assistant
Andrew Kamali, Health Policy Advisor
Lilia Lopez, Assistant Attorney General
Hannah Haag, Community Engagement Coordinator
Ashley Bell, Equity & Engagement Manager

Guests and other participants:

Kelly Cooper, Department of Health
Dr. Alan Melnick, Clark County Public Health
Mike Means, Department of Health
Meghan Cichy, Department of Health
Katherine Graff, Department of Health
Dani Toepelt, Department of Health

Patty Hayes, Board Chair, called the public meeting to order at 9:30 a.m. and read from a prepared statement (on file). Michelle Davis, Board Executive Director, introduced the public comment process, Board Members gave introductions, and Executive Director Davis gave the land acknowledgement.

1. **APPROVAL OF AGENDA**
   Motion: Approve June 12, 2024, agenda
   Motion/Second: Member Dean/Member Nandi. Approved unanimously

2. **ADOPTION OF APRIL 10, 2024, MEETING MINUTES**
   Motion: Approve the April 10, 2024, minutes
   Motion/Second: Member Abdelmalek/Member Kutz. 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, highlighted concerns over deaths linked to COVID-19 vaccinations in Washington. G. Braude said the Department of Health (Department) balances risks between disease and vaccine, but asserts benefits outweigh risks. G. Braude raised the question if it is worth sacrificing a few for the benefit of the whole.

   Bob Runnels, Informed Choice WA (ICW), presented an article on excess mortality despite COVID-19 containment measures and vaccinations. B. Runnels urged government leaders to consider these statistics and take a comprehensive approach before enforcing unrestricted masking and vaccinations.

   M. Johnson, yielded their time to others.

   Karl Kanthak, Clark County, discussed measles outbreaks and restrictive 2019 legislation. K. Kanthak noted issues with vaccination documentation, and exemptions and commented on immunization data inaccuracy and unresponsiveness from the Department.

   Bill Osmunson, Dentist, criticized fluoride in water supplies. B. Osmunson requested a forum and rule change regarding fluoridation.

   Natalie Chavez highlighted concerns from a court case regarding the Centers for Disease Control (CDC) monitoring of COVID-19 vaccine adverse reports. N. Chavez mentioned websites and documentaries alleging vaccine risks, citing significant increases in cardiac and pulmonary deaths in King County.

   Lisa Templeton, ICW, discussed bird flu and COVID-19 testing, and questioned the response to minor health threats compared to COVID-19. L. Templeton expressed concern regarding developing vaccines for less consequential diseases.

   Stacy Torrence supported community water fluoridation in King County, citing benefits for dental health and fluoride's safety and efficacy.

4. BOARD ANNOUNCEMENTS AND OTHER BUSINESS

Michelle Davis, Board Executive Director welcomed Board Members, summarized recent activities, and invited questions from Board Members regarding current initiatives. Executive Director Davis highlighted the Board's workload, and described ongoing projects in environmental health, health policy, the Pro-equity anti-racism (PEAR Plan), and State Health Report. Executive Director Davis noted the Health Impact Review team is actively working on interim projects and preparing for upcoming presentations. Executive Director Davis talked about the need for additional staffing to handle the workload effectively.

Kelly Oshiro, Board Vice Chair asked about the workload, projects, and staff time. Executive Director Davis mentioned the need to update many aspects of notifiable conditions including the COVID reporting. Executive Director Davis talked about the mentioned updates for conditions, lab reporting, and more. Executive Director Davis talked about the Technical Advisory Committee needs including the time, resources, technical considerations, and subject matter experts. Steve Kutz, Board Member, said this work is complex and can take a significant amount of time.

Kate Dean, Board Member acknowledged the commendable work of the staff and recognized the increasing workload of the Board. Member Dean expressed commitment to supporting the staff in fulfilling their responsibilities, particularly in response to legislative demands.

Executive Director Davis provided updates on subcommittee meetings, new staff biographies and position changes, project recruitments, and changes to upcoming Board meetings.

5. DEPARTMENT OF HEALTH UPDATE (TENTATIVE AGENCY REQUEST LEGISLATION)

Michael Ellsworth, Department of Health, Secretary's Designee and Kelly Cooper, Department of Health (Department), provided an update from the Department (on file).

Patty Hayes, Board Chair asked about assumptions around data sharing. Chair Hayes noted that the Board would like to be a thought partner.

Kelly responded that there is a governance board and they are working with WATech to work out a plan. The Department is also working with Arizona State University to develop a big-picture overview of all the laws impacting public health.

Kate Dean, Board Member asked what the hemoglobin testing will include.

Kelly shared that there are multiple interests, including the Woman Infants and Children (WIC) program, the Food and Drug Administration (FDA), and Medicaid. Kelly also shared that the FDA does not necessarily provide funding for lead testing. The

Department is working to address the barriers. Kelly said they want the draft legislation to be broad enough to include lead testing.

Member Dean asked Executive Director Davis about how the Board and Department work together when developing their decision packages.

Executive Director Davis stated that in the past, with requests not related to Foundational Public Health Services (FPHS), the Board worked closely with the Department's fiscal team. Executive Director Davis spoke about the funding related to FPHS and the discussion about maintenance level funds.

Paj Nandi, Board Member asked how the state is out of compliance with the Drinking Water Act. Kelly responded with information about the farm exemption that exists and how making the change will be a big lift.

Steve Kutz, Board Member expressed support to Executive Director Davis. Member Kutz noted how exceptional and rich the materials and information are that staff brings forward, and that the Board needs more funding to support that work. Member Kutz discussed the complications with WIC and the shift from the public to the private sector.

6. CLARK COUNTY PUBLIC HEALTH

Dr. Alan Melnick, Clark County Public Health described partnerships with Skamania, Wahkiakum, and other jurisdictions. Clark County provides support to Skamania County through Foundational Public Health Service (FPHS) funds for communicable disease (CD) control. Dr. Melnick noted Clark County's support for Skamania during a significant CD outbreak. FPHS was also used to help Skamania County with its community health assessment (CHA). Clark County is also a hub for health care acquired infections and does that work across jurisdictions, including settings like adult family homes.

Dr. Melnick shared that Clark County has supported Skamania County with training and mentoring new staff on food safety standards. Clark County has provided nurse family partnership services with Clallam County for some time.

Dr. Melnick talked about Clark County's work in coordination around incident management, including providing courses and coordination and updates to regional response plans.

Dr. Melnick described Clark County's community engagement work within FPHS funding, such as their work with Fourth Plain Forward, which is a community from different cultures and life experiences. They work with culturally specific doulas and provide linguistically appropriate food permit safety classes. Another initiative, Raising Clark County, is a pregnant and parenting initiative, that informs policies to inform and provide support for infants and young children. Clark County has conducted listening sessions and more.

Dr. Melnick also spoke about environmental justice and climate change. Clark County Department of Planning partners with Clark County Public Health on a comprehensive growth plan. FPHS supports a coalition of Community-Based Organizations for

engagement and has completed contracts with community partners most impacted by climate change. They use community compensation, which includes Community Health Assessments (CHA) and County Health Improvement Plan (CHIP).

Dr. Melnick shared they are on the Board of Accountable Communities of Health (ACH) and members of the Southwest Washington Equity Coalition. Cowlitz Tribal manager, Michael Watkins is on Clark County's Public Health Advisory Council. They just completed the CHA and CHIP and are launching their 5-year strategic plan. They use a MAPP 2.0 framework and have a community steering committee from priority populations. Through a facilitated process, they identified ways to support community health initiatives. In 2020, the Local Board of Health passed a resolution that acknowledged racism as a public health crisis, and they are using FPHS funding to advance that work. They received support for a racial equity director that will be part of the leadership, but before they do that, they will take a deep dive and do an assessment of the organization, policies and procedures, and how they work with community partners. Today they released an RFP for a racial equity consultant to help do that work.

Mike Ellsworth, Department of Health, Secretary's Designee asked if the recently completed CHIP be posted on the website. Dr. Melnick responded yes. Member Ellsworth then asked about MAPP 2.0. Dr. Melnick responded that it is localizing action for policy partnership. It's the state of the art in doing CHA and the CHIP.

Patty Hayes, Board Chair appreciated hearing about the role of a larger jurisdiction and the role that they play in Southwest Washington.

Dimyana Abdelmalek, Board Member asked what Dr. Melnick's thoughts on regional approaches and local health jurisdiction partnerships for public health concerns. Member Abdelmalek asked how it has impacted work and if there are areas the Board can support. Dr. Melnick responded that relationships are great and that they work out very well. Historically we had good work with health officers to provide coverage for others. Local interaction is great, even if we are not providing staffing. We are sharing policies and procedures, and not reinventing the wheel. Elected official changes can have positive and negative impacts.

Member Abdelmalek then asked if there are supports that we should be mindful of as we look at these issues. Dr. Melnick responded that a key factor in terms of writing rules is to consider how work gets done at the local level. Dr. Melnick stated public health shouldn't be a political endeavor and should be based on science. There's a lot of variability around the state. Dr. Melnick noted the main challenge of FPHS is that funding is not indexed, as the cost of living goes up. If funding stays flat, staff may be laid off or positions are unfilled.

The Board took a break at 11:22 a.m. and reconvened at 11:30 a.m.

7. RULES BRIEFING – CHAPTER 246-290 WAC, GROUP A PUBLIC WATER SUPPLIES, IMPLEMENTING THE EPA'S PUBLISHED PFAS STANDARDS – POSSIBLE ACTION

Kate Dean, Board Member introduced the briefing and provided context on Environmental Protection Agency's (EPA) April 2024 adoption of national standards around per- and polyfluoroalkyl substance (PFAS) in drinking water.

Mike Means, Department of Health (Department) presented about Washington state standards for PFAS in drinking water and how the Board's current rules compare to the federal regulations (on file).

Shay Bauman, Board staff presented rulemaking considerations and recommendations related to the new standards (on file).

Steve Kutz, Board Member asked whether activated charcoal or reverse osmosis filters might remove PFAS. Mike said that granular activated carbon in activated charcoal filters is one of the primary treatment methods for PFAS. Mike added that the Department has informational materials about how to treat individual water systems.

Kelly Oshiro, Board Member asked how long emergency rulemaking around this item might recur. Shay said that this emergency would continue to come up in front of the Board until 2026.

Member Kutz asked whether the Board would have the option to keep a state standard that was more stringent than the corresponding federal standard. Shay said yes.

Dimyana Abdelmalek, Board Member asked what the impacts might be if the Board adopted the EPA's standards as state standards until the federal regulations take effect. Shay said these impacts would be analyzed throughout the rulemaking process through mechanisms such as a small business economic impact statement and other analyses.

Mindy Flores, Board Member asked whether the EPA's regulations may change before the federal regulation effect date. Shay said that would be up to the EPA and that there are uncertainties with the potential change in government administrations. Shay and Mike said that the Board does have the authority to adopt more stringent standards than the federal government as appropriate.

Member Kutz asked what scientific body informs the EPA's decision-making process and whether this process is subject to political influence. Mike said that the EPA's Office of Research and Development is robust and produces scientifically sound work. Mike said that rules must be additionally reviewed by the White House's Office of Management and Budget, at which point a rule and its implementation may be politically influenced. Member Kutz asked whether the most recent PFAS rulemaking process followed science, and Mike said yes.

Mike Ellsworth, Department of Health, Secretary's Designee asked what the benefit of emergency rulemaking would be. Lilia Lopez, Assistant Attorney General (AAG), said that the Board's emergency rule would adopt the EPA's standards as state standards until the federal effective date. Member Kutz asked whether this would put the state in charge of enforcement as well. Mike said yes and clarified the mechanisms outlined in Recommendation 2.

Michelle Davis, Board Executive Director said another way to think about this issue might be to consider what would happen if the Board did not adopt this emergency rule. Shay said that if the Board did nothing, the delayed federal standards would take effect over the Board's rules, and the protections over the state standards would lapse until the new federal standards take effect. Executive Director Davis said that additionally, people in Washington State would stop getting notifications about PFAS contaminations in their drinking water since the federal requirements around reporting don't take effect until 2029.

Paj Nandi, Board Member asked whether an equity analysis has been done regarding the impacts of PFAS on communities of color and low-income communities. Mike said that the Department has explored an equity analysis. Mike noted that detections of PFAS in Washington State have been primarily associated with firefighting foam use, though there are exceptions. Mike said that communities of color and communities at risk are primarily served by large water systems, which have the greatest capacity to support safe and reliable water. Mike noted however that PFAS is a ubiquitous concern, impacting both public water systems and individual wells. Mike said that the Department can leverage federal funds to support any system that needs it, but that the need is greater than available funding. Mike said the Department is working with the Department of Ecology (Ecology) on a decision package to consider additional funding sources.

Kelly Oshiro, Vice Chair said that it would be beneficial to have additional briefings about the effects of PFAS on children, as well as the Department and Ecology's work on PFAS.

Member Kutz asked whether there are rules regulating other sources of PFAS, such as fast-food wrappers. Shay replied that actions are being taken around the state around PFAS sources such as fast-food wrappers and cosmetics.

Patty Hayes, Board Chair recommended that Board Members gather their questions, and we develop a process for forwarding these to the Department of Ecology for additional briefings.

Motion: The Board directs staff to do the following:

- File a CR-103E to initiate rulemaking for WAC 246-290-315, to clearly maintain the SALs and associated requirements until the federal standards are effective;
- File a CR-102 to adopt the federal standards and associated effective dates into chapter 246-290 WAC through an exception rulemaking process;
- File a CR-101 to permanently fix the rule language in 246-290-315 and to explore adopting the MCLs as SALs until the MCLs become effective; and
- File a CR-102 to update references in chapter 246-390 WAC through an exception rulemaking process

Motion/Second: Member Abdelmalek/Member Ellsworth. Approved unanimously

8. UPDATE - DELEGATED RULEMAKING – ENGROSSED SECOND SUBSTITUTE HOUSE BILL (E2SHB) 1181, CLIMATE RESILIENCE IN WATER SYSTEM PLANS, GROUP A PUBLIC WATER SUPPLIES, CHAPTER 246-290-100 WAC

Mike Means, Department of Health (Department) updated the Board on delegated rulemaking related to Engrossed Second Substitute House Bill (E2SHB) 1181 (on file). Mike also introduced Brad Burnam, Department of Health as a resource for questions during the meeting.

Steve Kutz, Board Member asked how often plans are updated. Mike responded that plans are updated every 10 years.

Kate Dean, Board Member asked for a quick overview of new requirements. Mike responded that the climate resilience element is the new requirement. Mike said there is an emergency response plan, and this new element is more specific to include climate resilience in those plans. Brad added that there are three elements: extreme weather events, protecting critical assets, and reports about costs and benefits. Brad discussed partnering with the University of Washington's Climate Impact Group to provide guidance and develop tools that will help water systems meet new requirements.

Member Dean stated that Jefferson County was delayed in updating their coordinated water plan due to COVID-19 as were others. Member Dean expressed appreciation for the new guidance.

Mindy Flores, Board Member asked if there has been any research on the impacts of new requirements around water affordability. Mike said it wasn't directly evaluated under the rulemaking process but that there was a small business impact evaluation. Mike said the plan is to keep water affordable for the long term, so requiring utilities to have a plan in place for things like additional water salinity should reduce the long-term water cost increases.

Member Kutz said it is easy to go over the established amounts of water, and then costs increase quickly. Mike responded that this is a very complicated picture across the state. Individual water systems set their rate schedules. Mike said addressing small water system issues in this state is an ongoing conversation.

The Board took a break at 12:37 p.m. and reconvened at 1:32 p.m.

9. RULES HEARING – ABBREVIATED RULEMAKING, HANDLING OF HUMAN REMAINS CHAPTER 246-500 WAC IMPLEMENTING CHANGES FROM SUBSTITUTE HOUSE BILL (SHB) 1974

Patty Hayes, Board Chair introduced the rules hearing and announced procedures for public testimony.

Shay Bauman, Board staff then delivered a presentation about proposed amendments, a summary of comments received, and recommended Board actions for Chapter 246-500 WAC (on file).

Kate Dean, Board Member asked about the process through which unclaimed remains are reduced before burial. Shay replied that this is up to the entity in possession of the remains, but typically the process is cremation.

Steve Kutz, Board Member asked whether unidentified remains are disposed of according to a different regulation. Shay said this was correct.

There were no comments during the public testimony portion of this item.

Motion: The Board adopts the proposed amendments to chapter 246-500 WAC, Handling of Human Remains, as published in WSR 24-10-094, and directs staff to file a CR-103, Order of Adoption, and establish an effective date for the rules.

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

10. 2024 STATE HEALTH REPORT – POSSIBLE ACTION

Patty Hayes, Board Chair commended staff on the quality of this work and the efforts to engage the community. Chair Hayes looks forward to integrating this approach into our regular work and recommended that the Health Promotion Committee discuss and debrief in the fall.

Mindy Flores, Board Member explained how community input has been integrated into the draft State Health Report (SHR). Member Flores encouraged Board Members to provide feedback before the SHR goes to the governor.

Molly Dinardo, Board staff thanked the Board for the flexibility in how the report was approached this year. The feedback received so far has been positive. Molly thanked Member Flores for support throughout this process.

Hannah Haag, Board staff shared a presentation about the first-time approach of convening community panels as part of our process (on file). Board staff are proud of keeping a strong focus on relationships and asking community partners to help create an inclusive and comfortable space for discussion. Many panelists are interested in continuing to partner with the Board to make a difference in public health in Washington. Hannah shared that we heard from diverse voices and acknowledged there were geographic gaps we were unable to cover. Trusted messengers helped us get voices from across the state. Looking at the topics in an interconnected way allowed us to inform the recommendations. Hannah described connecting with panelists after partnering in this work and shared some of their feedback that's important for the Board to consider before moving forward.

Chair Hayes referenced a community panelist's question about whether the right people are being represented in the conversation. Steve Kutz, Board Member appreciated staff who brought community voices forward and looks forward to continuing this approach going forward. Member Flores commended staff members dedicated to maintaining

focus on community members and shared that community members have also reached out to Member Flores with gratitude for being able to share what they're doing in their communities. Member Flores gave kudos to Board Members for asking questions and staying engaged in the conversation.

Kate Dean, Board Member asked if we are getting a lot of voices in identifying who should be at the table and what themes we are addressing. Hannah appreciated this question and said community voices should not just be a check box but should help us shape the work.

Dimyana Abdelmalek, Board Member expressed the importance of being able to listen and participate in the panels; it was extremely helpful to hear lived experiences and to hear how communities are tackling specific key issues. Member Abdelmalek explained that the value is bi-directional and that they carry this knowledge back to their own work.

Member Kutz plans to follow up with some of the panelists on their innovative ideas. Member Kutz asked how we can carve out more time for interaction so that people don't feel rushed through these conversations.

Chair Hayes said that this is a commitment on the Board's part to continue to interact. There is value in having lunch together after the meetings. Chair Hayes asked how the Board can continue to integrate this work every time we create the State Health Report. Lived experience adds a layer to our recommendations. Chair Hayes said one of our highest spaces is to build upon what we've just done in partnership with community. We were asked by the Legislature to do this. We have a rich opportunity to formalize and continue this work.

Kelly Oshiro, Board Vice Chair appreciated having unique voices not traditionally heard in public health.

Mike Ellsworth, Department of Health, Secretary's Designee expressed value in hearing from and following up with community members who shared that they'd had trouble accessing data. Member Ellsworth said this should be a model in our state.

Member Dean appreciates the time invested by staff to meet multiple times with community members and to get input on what's a useful process. Member Dean hopes this will become a best practice.

Member Flores heard from community members that they appreciated interacting with each individual expert. This opportunity expands their network, gets information to them more quickly, and may result in better outcomes for their communities. Member Flores echoed what Dr. Melnick said earlier; "when I am voting on rules, it's important to reflect on how this plays out at the local level. This makes my decisions more sound."

Molly highlighted recommendations which are similar to those in the last report. Molly referenced page 234 of the meeting packet, which shows progress made on recommendations from the last report. Molly shared feedback that Recommendation 1 on Data Disaggregation should be further plain talked and expanded to focus on health equity.

Member Dean asked how we got input. Molly shared that we asked agency partners as well as community partners, Board, and staff.

Molly shared additional questions and feedback on the various recommendations.

Chair Hayes asked about an answer to one of the questions about the Workgroup on Language Access. Molly is waiting for answers from the Department of Health (Department) about whether the workgroup tackles translation as well as interpretation.

Member Kutz commented that we collected information that is useful beyond the Department. Some of the things that came up have also been raised by our Governors Interagency on Health Disparities Council (Council). Member Kutz said the Council should be handing out the report to their networks.

Molly highlighted the feedback on sharing lessons learned from the pandemic and shared a newer recommendation regarding environmental justice. Molly ended the presentation by referencing page 227 of the meeting packet, which includes a draft of the community responsiveness summary.

Motion: The Board directs staff to finalize the 2024 State Health Report (SHR) based on the Board's input today, in consultation with the Chair and Board sponsor and to send the report to the Governor by July 1, 2024. When the Governor's Office receives the SHR, staff are directed to send a copy to community representatives and groups who contributed to it, the Legislature, and appropriate state agencies.

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

Member Oshiro echoed that the report should be distributed widely.

11. SCHOOL ENVIRONMENTAL HEALTH AND SAFETY- EXTEND EFFECTIVE DATE OF CHAPTER 246-366A WAC – POSSIBLE ACTION

Patty Hayes, Board Chair shared that this is the ninth time the Board will have to extend the implementation of Chapter 246-366A WAC.

Andrew Kamali, Board staff referenced page 243 in the meeting packet, which shows the 2004 – 2009 recommendations. Andrew explained the Board will need to act to extend the effective date.

Motion: The Board directs staff to amend the effective date of new sections of chapter 246-366 WAC and new chapter 246-366A WAC, as filed in WSR 23-16-005, by filing a new CR-103, Order of Adoption, to delay the effective date of the new rules to September 1, 2025.

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

Dimyana Abdelmalek, Board Member expressed excitement for the next phase of reviewing these recommendations.

12. SCHOOL RULES REVIEW PROJECT

Patty Hayes, Board Chair introduced the school rule review project and provided historical background on the topic. Chair Hayes stated the current rules on School Environmental Health and Safety are outdated. Chair Hayes said that during the 2024 legislative session, the Legislature passed a proviso, including funding and staff support to review the rules. Chair Hayes stated that the Board will collaborate with schools and organizations to complete this work and that the timeline is advantageous. The work includes an Environmental Justice (EJ) Assessment and extensive partnerships across the state to create equitable rules and recommendations to the Legislature.

Andrew Kamali, Board staff provided an informational briefing on the school rule review project. The project will hire several staff, including a policy advisor, communications consultant, administrative assistant, and community engagement staff who will start work on June 16. Andrew presented an overview of this work, including background, Technical Advisory Committee (TAC) details, and project timeline (on file).

Steve Kutz, Board Member commented that the project timeline does not allow space for changes.

Kate Dean, Board Member asked whether the rule review would begin by working with the 2009 rules or starting the process over. Andrew responded that Board staff will review the 2009 rules, as there was a lot of research conducted then; however, the intent is to write new rules, as science has been updated and there is information to consider that came from the COVID-19 pandemic.

Member Dean stated that local health jurisdictions (LHJs) have varying capacity to monitor school health and safety, and asked if we anticipate existing structures to remain in place.

Chair Hayes stated there is innovation through the Foundational Public Health Services (FPHS) Steering Committee and through the Board's representation, and that interlocal arrangements are a great place for coordination. Chair Hayes stated that health officers are thinking about what approaches will work for collaboration across the state versus what will work for defaulting to the state.

Dimyana Abdelmalek, Board Member agreed with Chair Hayes and provided an example from Thurston County, which conducts school inspections. Member Abdelmalek stated there is varying capacity across jurisdictions and fees can be a burden for schools. Member Abdelmalek also asked how TAC members are selected and how the Board can support this work.

Michelle Davis, Board Executive Director stated the importance of highlighting ways schools are already doing some of this work. For example, with regular food inspections the local health jurisdiction (LHJ) follow up, and preoccupancy inspections. Executive

Director Davis stated that there is variation across the state and that Foundational Public Health Services funds, have enabled LHJs to establish certain programs. However, there are still some funding issues. Executive Director Davis stated that schools and local health collaborated during the COVID-19 pandemic, which may hopefully move this work forward. Executive Director Davis stated that the intention of this work is to create environments that are safe and healthy for kids, and believes all parties involved in this work want that shared goal. Executive Director Davis also reminded the Board that the minimum health and safety rules are what will be established, that schools can implement beyond those standards, and that this project is a huge challenge and a lot of work.

Andrew responded stating that the Board will work to develop minimum and implementable standards. Regarding TAC members, organizations were asked to self-select, where representatives need to have experience in school buildings and infrastructure. Andrew also stated that the Board is working to be as transparent as possible through this process and will develop educational materials for distribution. TAC meetings are open to the public and comments can also be given at Board meetings. There will be an informal comment period and listening sessions with community. In addition, a new agency rulemaking webpage is on sboh.wa.gov with information about this project.

Member Kutz noted the need for a strong partnership with the Office of Superintendent of Public Instruction (OSPI) through this project and asked if a representative from OSPI has been identified yet.

Andrew said that representatives from OSPI have been identified, and include an external affairs/legislative staff person, as well as the head of facilities. This is a great relationship and a partnership that is continuing to grow.

Member Kutz mentioned a recent national report was published on the age of schools and structure across the U.S., and the costs associated with repair. Member Kutz recommended including this information in the report about this project. Member Kutz asked how Tribes would be involved in this work, given there are Tribal schools where the state does not have jurisdiction, but often have the same issues as public schools.

Andrew responded that 2.5% of public-school students are members of Indigenous Tribes, and that Tribes and Tribal Compact Schools may look to state rules for guidance. Andrew also stated that including Tribes in the EJ conversations will be important. Andrew noted working with the Board's Equity and Engagement Manager to write a Dear Tribal Leader letter on this topic.

Member Kutz offered to help staff make connections with Tribes, including through the Bureau of Indian Affairs Office in Portland and possibly the Affiliated Tribes of Northwest Indians. Member Kutz also stated that Dear Tribal Leader letters are received often and may be overlooked.

Paj Nandi, Board Member, asked how "overburdened communities" are defined and how they are involved in this work.

Andrew responded that the definition is pulled from the Healthy Environment for All Act and there may be overlap in identifying overburdened communities as we engage communities across the state, including diverse Parent Teacher Associations and the Washington School for the Deaf and Washington School for the Blind.

Andrew introduced some of the Department of Health staff who will be working on this project with the Board.

13. REQUEST FOR DELEGATED RULEMAKING AUTHORITY, MINOR ADMINISTRATIVE UPDATES TO IMMUNIZATION RULES, WAC 246-105-040 AND 060 – POSSIBLE ACTION

Patty Hayes, Board Chair introduced the topic and relevant rules (WAC 246-105-040 and 060). Chair Hayes said the Department of Health (Department) is asking the Board to delegate rulemaking authority for minor changes to the rules. Chair Hayes mentioned the state law and Board policy that authorize and outline procedures for rulemaking delegation (on file).

Molly Dinardo, Board staff asked Department staff, Megan Cichy and Katherine Graff, to introduce themselves. Megan Cichy, Senior Policy Analyst for Prevention and Community Health presented the Department's request for delegation of rulemaking authority (on file). Megan said that the Department wants to make administrative updates that would not change the listed diseases for which immunity is required for school attendance.

Chair Hayes said the question before the Board is delegated rulemaking under the Board's established criteria. Kate Dean, Board Member thanked the presenters and said the presentation was thorough. Michelle Davis, Board Executive Director requested that the Department provide the Board a rulemaking update in the future.

Motion: The Board delegates to the Washington Department of Health rulemaking authority to make minor changes to WAC 246-105-040 to update the immunization schedule to the most recent version and WAC 246-105-060 to remove the school immunization status reporting date.

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

14. RULES UPDATE – SANITARY CONTROL OF SHELLFISH, CHAPTER 246-282 WAC

Patty Hayes, Board Chair introduced this item. Chair Hayes said the Board held an informal comment period with industry partners on this rule and shared the Board's thanks for the responses received. Chair Hayes restated the Board's commitment to collaborating with partners and noted that the timeline for this rulemaking is being pushed out to allow for further engagement.

Dani Toepelt, Department of Health gave a presentation about the rule's background, recommended changes, summary of informal comments, and next steps (on file).

Kate Dean, Board Member stated regret that there was pushback on adding Vibrio *vulnificus* (Vv) to the Vibrio *parahaemolyticus* (Vp) control plan. Member Dean asked how California handled its case of Vv and whether the national standards may trigger any responses that the Board can use to draft its rule and garner industry support. Dani said that Vv and Vp control plans are often the same but is not sure whether this was the case for California. Dani said that the Food and Drug Administration's national guide has very strict requirements and helps develop state standards.

Member Dean asked whether the industry is compelled by the fact that national standards are more stringent. Dani said that there has been discussion about the plan's existence leading to unfavorable speculation. Dani spoke about further opportunities to engage the industry and clarify the rule.

Member Dean and Dani then discussed the Department of Health (Department) process of monitoring Vibrio. Dani said that the Department only monitors in the summer months. Member Dean then asked whether the absence of bar graph data for May, June, July, and September of 2020 (slide 10) meant that Vibrio was monitored for but not detected. Dani confirmed this was the case. Member Dean and Dani discussed the increase in prevalence of Vibrio over the past few years.

Paj Nandi, Board Member asked if trends indicate the Board will need to pass another emergency rule this summer. Dani said that the Department is not planning to file one for June as the next series of low-tide days is not predicted to have significant temperatures.

Steve Kutz, Board Member shared concerns that certain areas of Washington had temperatures of almost 80 degrees this week. Member Kutz and Dani then spoke about the Department's discussions with the industry about Vibrio in different growing areas.

Kelly Oshiro, Vice Chair asked when the Department will have Vibrio data from this summer. Dani said they will try to have it by November 1, though some illnesses are recorded two to four weeks later. Vice Chair Oshiro and Dani then discussed the Department's data collection timeline to industry harvesting timelines.

Chair Hayes asked about the timeline for rulemaking. Shay said that Board staff will meet again with industry and Tribal partners throughout the summer and early fall to hear impacts and conduct analyses.

Member Kutz and Dani then discussed the Department's guidelines for consumers around cooking and consuming shellfish.

15. POSSIBLE SCHEDULE CHANGE, JULY AND AUGUST BOARD MEETINGS – POSSIBLE ACTION

Michelle Davis, Board Executive Director talked about the upcoming meetings in 2024 and suggested canceling the July meeting and moving the August Board meeting to August 7, 2024.

Motion: The Board approves the proposed schedule changes for the July and August Board meetings.

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

16. PETITION FOR RULEMAKING – CHAPTER 246-260-131 WAC, OPERATION OF WATER RECREATION FACILITIES – POSSIBLE ACTION

Patty Hayes, Board Chair briefly introduced the agenda item. Chair Hayes asked Andrew Kamali, Board staff, Dave DeLong and Ashlie Laydon, Department staff, to present more information on the petition.

Andrew Kamali, Board staff said that the Board received a petition to amend WAC 246-260-131(6)(b)(i) and (ii) on May 8, 2024. Andrew shared background information on the law that guides petitions for rulemaking, and the Board's policy for responding to petitions. Andrew described the petition, which requested the Board amend this specific section of the rule to remove the allowance for the substitution of a swim or dive coach or scuba instructor in place of a lifeguard (on file).

Dave DeLong, Department staff shared about the Board and Department's current rulemaking process to update the water recreation rules (on file) and asked Board Members to direct the petitioner to participate in the current rulemaking process to help make needed changes.

Ashlie Laydon, Department staff provided a timeline of the rulemaking process and stated that they hope to begin work on the section of the rule that addresses lifeguards sometime this fall.

Steve Kutz, Board Member shared the concern of the petitioner. Member Kutz commented that if a person is coaching a swim team or a scuba diving class, it is dangerous and unrealistic for them to also serve in a lifeguarding capacity.

Kelly Oshiro, Board Vice Chair inquired about whether there is a lifeguard workforce shortage and if this contributes to the issue.

Andrew responded that the petitioner runs an aquatic facility in the Seattle area and their concern is that coaches and instructors do not necessarily have lifeguard training. Andrew stated that the petitioner shared the risks that come with not having a trained lifeguard on site and that drownings have occurred at facilities because of a lifeguard not being on duty. Andrew said Dave alluded to workforce issues in the presentation, and that this would be a consideration they would have to investigate and address as the rulemaking process proceeds.

Member Kutz made a motion to decline the petition and added to the motion that the petitioner be invited to participate in the rulemaking process to support changes to this section of the rule.

Patty Hayes, Board Chair asked a clarifying question about whether declining the petition while including Member Kutz's addition to the motion will make it clear to the petitioner that their concern is being addressed, and that the Board isn't denying the petition because they don't think it is a valid request.

Andrew said staff will make it clear in the response letter to the petitioner that their concern is valid and that the Board will consider the issue regardless of the petition outcome. The issue of having trained lifeguards on duty is addressed in the Model Aquatic Health Code (MAHC), which is the document being used in the current rule revision. Andrew also said they will continue to engage with the petitioner and invite them to participate in the rulemaking.

Motion: The Board declines the petition to initiate rulemaking to amend WAC 246-260-131(6)(b)(i) and (ii), for the reasons articulated by the Board, and directs staff to notify the requestor of the Board's decision and requests that the petitioner be invited to participate in the rulemaking process as it's currently underway.

Motion/Second: Member Kutz/ Member Dean. Member Abdelmalek abstained.

17. BOARD MEMBER COMMENTS

Dimyana Abdelmalek, Board Member announced that the National Tuberculosis Controllers Association has created new recommendations for shortening the isolation period, following a more patient- and risk-centered approach.

ADJOURNMENT

Patty Hayes, Board Chair adjourned the meeting at 4:27 p.m.

WASHINGTON STATE BOARD OF HEALTH

_____

Patty Hayes, Chair

WASHINGTON STATE BOARD OF HEALTH

Placeholder for Public Comment

Added three business days prior to meeting

WASHINGTON STATE BOARD OF HEALTH

School Environmental Health and Safety Rule Project 2024 - 2025

Andrew Kamali, MPH
School Rule Project Manager

Andrew Kamali joined the Washington State Board of Health (Board) on May 1, 2023. He initially supported policy work and rulemaking related to environmental public health and disease prevention. In June 2024, Andrew was hired as the Project Manager for the School Rule Project.

Before joining the Board, Andrew worked at The Water Collaborative of Greater New Orleans as a policy fellow, where he worked to develop equitable policy recommendations, study PFAS contamination, and support community workshops on storm water fee development in the city of New Orleans. Before that, Andrew worked in a variety of research positions including studying the effects of indoor allergens on childhood respiratory diseases and novel drug treatment development for traumatic brain injury.

Andrew received his Bachelor of Arts degree in International Development studies from the University of California, Los Angeles, and his Master of Public Health in Health Policy from Tulane University.

WASHINGTON STATE BOARD OF HEALTH

School Environmental Health and Safety Rule Project 2024 - 2025

Mary Baechler, MS, CN
Community Engagement Coordinator

Mary Baechler joined the Washington State Board of Health (Board) on June 16, 2024. Mary focuses on developing, implementing, and evaluating community outreach strategies for the School Rule Project and Technical Advisory Committee (TAC) meetings.

Before joining the Board, Mary worked for Seattle & King County Public Health as a Disease Research and Intervention Specialist on the Covid team. In this role, she conducted COVID-19 outbreak investigations and provided support for homeless service providers, schools, and long-term care facilities. Her previous experience includes working as the Planner in Economic Development for Yakama Nation. Additional experience includes working on water testing for nitrates, coliform, and lead in the Yakima Valley. Mary also worked as a CDC Senior Field Representative in eastern Washington, recruiting physicians, clinics, community health centers, and hospitals for the CDC National Ambulatory Medical Care Survey and National Hospital Ambulatory Medical Care Survey. Additionally, she worked as a community organizer, focusing on voter outreach in Yakima's diverse communities.

Mary received her Bachelor of Science degree in General Studies with a focus on Zoology and animal intelligence from Washington State University, and her Master's in Nutrition from Northeastern University. Mary is licensed as a Certified Nutritionist in Washington State.

WASHINGTON STATE BOARD OF HEALTH

School Environmental Health and Safety Rule Project 2024 - 2025

Marcus DeHart
Communications Consultant

Marcus DeHart joined the Washington State Board of Health (Board) as a Communications Consultant for the School Rule Project on June 17, 2024.

Before joining the Board, Marcus worked for Amazon as a Sr. Program Manager. In this role, Marcus supported a global editorial team through the development of processes, tools, and quality assurance. He used his expertise in communications to develop editorial workflows, collaborate with developers to build the team's content management system, develop training and documentation for tools and processes, and establish content quality control metrics and improvement plans. Before Amazon, Marcus' career encompassed many forms of communications ranging from writing, design, illustration, video, to animation, and a brief stint as the radio voice for Olympia's First Friday events.

Marcus grew up in Washington and graduated from Western Washington University in 1989, where he received his Bachelor of Arts in English with an emphasis on education. He lives in Olympia, Washington with his wife of 33 years and two adult daughters. During the pandemic, he took up pottery, and now partners with his wife's business to sell their creations.

WASHINGTON STATE BOARD OF HEALTH

School Environmental Health and Safety Rule Project 2024 - 2025

Nina Helpling, BSBM
Policy Advisor

Nina Helpling joined the Washington State Board of Health (Board) on June 17, 2024, as a Policy Advisor supporting the School Rule Project.

Before joining the Board, Nina worked for nine and a half years for the Washington State Department of Health (Department). During her time with the Department, she worked for three and a half years as a rules coordinator for the Environmental Public Health division to update and create Washington Administrative Code for topics such as temporary worker housing, U.S. Nuclear Regulatory Commission updates, Drinking Water State Revolving Fund updates, and private detention facilities. Before writing rules, she worked in the Office of Drinking Water as a drinking water laboratory liaison, fluoride program manager, and public disclosure data analyst. For the five years before working in state government, Nina was the operations manager and analyst at a local environmental laboratory in Olympia, Washington.

Nina earned her Associate of Science degree with an emphasis in Engineering from South Puget Sound Community College and her Bachelor of Science in Business Management from Western Governor's University.

WASHINGTON STATE BOARD OF HEALTH

School Environmental Health and Safety Rule Project 2024 - 2025

Crystal Ogle
Administrative Assistant

Crystal is excited to support the School Rule Project with the Washington State Board of Health (Board). The work the Board does is not only very important, but extremely interesting.

Crystal enjoys the opportunity to learn new things and meet new people. By training and education, Crystal is a Licensed Midwife in Washington State who periodically takes a leave from practice to work on other projects. Crystal's strong organizational and planning skills will be put to use on the Newborn Screening Project as well as the School Rule Project.

Crystal has a Bachelor of Science in Midwifery from the Midwives College of Utah and has a background in Social Justice work, Board leadership, and Secondary Education. Crystal lives on Whidbey Island with her family and enjoys her time spent with her children, grandchildren, amazing husband, and a growing herd of goats and chickens.

WASHINGTON STATE BOARD OF HEALTH

Newborn Screening Technical Advisory Committee (TAC)

Kelly Kramer
Policy Advisor

Kelly Kramer joined the Washington State Board of Health as a Policy Advisor on July 16, 2024. Kelly supports the rule review for the Newborn Screening Technical Advisory Committee (TAC).

Before joining the Board, Kelly worked for over three years for the Washington State Department of Health (Department) within the Office of Newborn Screening. During her time at the Department, she worked as a Disorder Follow-up Consultant. Kelly reported abnormal newborn screening results and provided guidance and next steps to providers who had patients with abnormal results. Later, Kelly worked as Team Lead and Hawai'i Liaison where she reported Hawai'i newborn screening results and provided assistance to Hawai'i State Newborn Screening.

Kelly received her Associate of Arts Degree from Yakima Valley Community College, and Bachelor of Arts in Public Health from the University of Washington (UW). She then went back to UW to get her Master of Public Health with a concentration in Public Health Genetics.

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: June 24, 2024
TIME: 12:54 PM

WSR 24-14-016

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: Testing of drinking water contaminates - State action levels (SALs) and state maximum contaminant levels (MCLs) in WAC 246-290-315.

The State Board of Health (board) has authority under RCW 43.20.050 to adopt rules for group A public water systems that are necessary to assure safe and reliable public drinking water and to protect the public health. Chapter 246-290 WAC, Group A Public Water Supplies, establishes standards and requirements for these water systems. The Department of Health (department) administers the rules.

To ensure safe drinking water, water must be tested for contaminants. The board establishes SALs and MCLs to ensure contaminate levels are below a certain threshold. The board sets criteria for the adoption of SALs and MCLs in WAC 246-290-315, and includes criteria that would apply upon federal adoption of MCLs. WAC 246-290-315(8) states that upon federal adoption of a MCL, the MCL will supersede a less stringent SAL and associated requirements, including monitoring and public notice.

The EPA published new federal standards for per- and polyfluoroalkyl substances (PFAS) on April 10, 2024, with an adoption date of June 25, 2024. These new standards include MCLs. This affects the board's rule and triggers the provision in WAC 246-290-315(8). The federal standards, however, have delayed effective dates for criteria and public health protections that are currently in place for Washington. According to the Washington state rules associated with the SALs, public water systems must notify customers of detections of PFAS above the SAL within 30 days of that detection. This is necessary to allow people the opportunity to protect themselves by using bottled water, securing a filter, or taking other measures. 30-day public notification is not effective for MCLs in the federal standard until April 2029. Without this amendment to WAC 246-290-315, customers served by group A public water systems will no longer be notified of dangerous levels of PFAS in their drinking water, which is a significant reduction in protections.

The board adopted an emergency rule on June 12, 2024, to amend WAC 246-290-315 such that the criteria would apply on the effective date of an MCL as set in the federal standard, not the adoption date, in order to maintain vital public health protections for drinking water safety. Along with the emergency rulemaking, the board initiated a permanent rulemaking to amend the rule language to align with the emergency provision and explore other protections.

Citation of rules affected by this order:

New:
Repealed:
Amended: WAC 246-290-315
Suspended:

Statutory authority for adoption: RCW 43.20.050(2)(a)

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 federal adoption date of the standards is June 25, 2024, at which point the MCLs and relative protections will supersede the SALs. Because of the delayed effective date, currently active public health protections will end on that date. The Board finds that emergency adoption of this rule is necessary to preserve public health.

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: June 24, 2024	**Signature:** 
Name: Michelle Davis, MPA	
Title: Executive Director, Washington State Board of Health	

AMENDATORY SECTION (Amending WSR 21-23-097, filed 11/17/21, effective 1/1/22)

WAC 246-290-315 State action levels (SALs) and state maximum contaminant levels (MCLs). (1) The department shall consider the following criteria to select a contaminant for developing a SAL:

(a) Drinking water contributes to human exposure to the contaminant.

(b) The contaminant is known or likely to occur in public water systems at levels of public health concern. Sources of occurrence information include, but are not limited to:

(i) Washington state department of agriculture;

(ii) Washington state department of ecology; and

(iii) Monitoring results reported in accordance with 40 C.F.R. 141.35.

(c) The contaminant has a possible adverse effect on the health of persons exposed based on peer-reviewed scientific literature or government publications, such as:

(i) An EPA health assessment such as an Integrated Risk Information System assessment;

(ii) Agency for Toxic Substances and Disease Registry toxicological profiles;

(iii) State government science assessment; and

(iv) EPA guidelines for exposure assessment such as the EPA exposure factors handbook.

(d) A certified drinking water lab can accurately and precisely measure the concentration of the contaminant in drinking water at and below the level of public health concern using EPA-approved analytical methods.

(2) After consideration of the criteria in subsection (1) of this section, the department may develop a SAL based on the following:

(a) Evaluation of available peer-reviewed scientific literature and government publications on fate, transport, exposure, toxicity and health impacts of the contaminant and relevant metabolites;

(b) An assessment based on the most sensitive adverse effect deemed relevant to humans and considering susceptibility and unique exposures of the most sensitive subgroup such as pregnant women, fetuses, young children, or overburdened and underserved communities; and

(c) Technical limitations to achieving the SAL such as insufficient analytical detection limit achievable at certified drinking water laboratories.

(3) The state board of health shall consider the department's findings under subsections (1) and (2) of this section when considering adopting a SAL under this chapter.

(4) Contaminants with a SAL.

(a) If a SAL under Table 9 of this section is exceeded, the purveyor shall take follow-up action as required under WAC 246-290-320. For contaminants where the SAL exceedance is determined based upon an RAA, the RAA will be calculated consistent with other organic contaminants per WAC 246-290-320(6) or other inorganic contaminants per WAC 246-290-320(3).

TABLE 9
STATE ACTION LEVELS

Contaminant or Group of Contaminants	SAL	SAL Exceedance Based On:
Per- and polyfluoroalkyl substances (PFAS)		
PFOA	10 ng/L	Confirmed detection
PFOS	15 ng/L	Confirmed detection
PFHxS	65 ng/L	Confirmed detection
PFNA	9 ng/L	Confirmed detection
PFBS	345 ng/L	Confirmed detection

(b) If a system fails to collect and submit a confirmation sample to a certified lab within ten business days of notification of the sample results, or as required by the department, the results of the original sample will be used to determine compliance with the SAL.

(5) The department shall consider the following when developing a state MCL:

(a) The criteria in subsection (1) of this section;

(b) Whether regulating the contaminant presents a meaningful opportunity to reduce exposures of public health concern for persons served by public water systems;

(c) The need for an enforceable limit to achieve uniform public health protection in Group A public water systems; and

(d) The need for an enforceable limit to support source water investigation and clean-up of a contaminant in drinking water supplies by responsible parties.

(6) In addition to the requirements in subsection (5) of this section, the department shall:

(a) Meet the requirements of subsection (2) of this section;

(b) Comply with the requirements in RCW 70A.130.010 to establish standards for chemical contaminants in drinking water;

(c) Consider the best available treatment technologies and affordability taking into consideration the costs to small water systems; and

(d) Determine that the probable benefits of the rule are greater than its probable costs, taking into account both the qualitative and quantitative benefits and costs.

(7) The state board of health shall consider the department's findings under subsections (5) and (6) of this section and follow the requirements under chapters 34.05 and 19.85 RCW when adopting a state MCL under this chapter.

(8) ((~~Upon federal adoption of an MCL~~)) When a federal MCL takes effect, the federal MCL will supersede a SAL or a less stringent state MCL, and the associated requirements, including for monitoring and public notice. If the federally adopted MCL is less stringent than a SAL or state MCL, the board may take one of the following actions:

(a) Adopt the federal MCL; or

(b) Adopt a state MCL, at least as stringent as the federal MCL, using the process in subsections (6) and (7) of this section.

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: June 28, 2024
TIME: 10:42 AM

WSR 24-14-089

Agency: State Board of health

Effective date of rule:

Permanent Rules

☐ 31 days after filing.

☒ Other (specify) 09/01/2025___ **(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: Restrictions imposed by the 2009 legislature on the implementation of new or amended school facility rules are retained in the 2023-2025 supplemental state operating budget, prohibiting implementation of the rules through June 2025.

Purpose: This filing delays the effective date of new sections of chapter 246-366 WAC, Primary and Secondary Schools, and new chapter 246-366A WAC, Environmental Health and Safety Standards for Primary and Secondary Schools, one year due to legislative direction in the supplemental state operating budget (Engrossed Substitute Senate Bill 5187) prohibiting implementation until the legislature acts to formally fund implementation. The rules provide minimum environmental health and safety standards for schools.

New sections of chapter 246-366 WAC, Primary and Secondary Schools, and new chapter 246-366A WAC, Environmental Health and Safety Standards for Primary and Secondary Schools, were adopted by the State Board of Health (Board) on August 12, 2009, filed as WSR 09-14-136. The Board filed a Rule-Making Order (CR-103), WSR 10-01-174, on December 22, 2009 setting the effective date of the rules as July 1, 2010. However, in advance of the Board's actions, the 2009 Legislature adopted a proviso in the state operating budget (Engrossed Substitute House Bill 1244) suspending implementation of the rules until the Legislature acts to formally fund implementation. The proviso has been included in all subsequent state operating budgets, including the 2023-2025 supplemental state operating budget (ESSB 5187). In response, the Board has taken the following series of actions to delay implementation of the rules:

- Voted on March 10, 2010 to file an amended Rule-Making Order, filed as WSR 10-12-018 on May 21, 2010, to delay the effective date to July 1, 2011;
- Voted on April 13, 2011 to file an amended Rule-Making Order, filed as WSR 11-10-080 on May 3, 2011, to delay the effective date to July 1, 2013;
- Voted on March 13, 2013 to file an amended Rule-Making Order, filed as WSR 13-09-040 on April 11, 2013, to delay the effective date to July 1, 2015;
- Voted on March 11, 2015 to file an amended Rule-Making Order, filed as WSR 15-09-070 on April 15, 2015, to delay the effective date to July 1, 2017;
- Voted on June 14, 2017 to file an amended Rule-Making Order, filed as WSR 17-14-055 on June 28, 2017, to delay the effective date to August 1, 2019;
- Voted on June 12, 2019 to file an amended Rule-Making Order, filed as WSR 19-14-107 on July 2, 2019, to delay the effective date to August 1, 2021;
- Voted on June 9, 2021 to file an amended Rule-Making Order, filed as WSR 21-14-056 on July 1, 2021, to delay the effective date to August 1, 2022; and
- Voted on June 8, 2022 to file an amended Rule-Making Order, filed as WSR 22-14-021 on June 24, 2021, to delay the effective date to August 1, 2023.

- Voted on June 14, 2023 to file an amended Rule-Making order, filed as WSR 23-16-005 on July 19, 2023, to delay the effective date to August 1, 2024.

Action by the Board in June 2024 extends the effective date of the new rules to September 1, 2025. The Board will continue to monitor the state budget and budget proviso suspending implementation of the new rules in the coming legislative sessions in 2025.

Citation of rules affected by this order:
New: None
Repealed: None
Amended: None
Suspended: None

Statutory authority for adoption: RCW 43.20.050

Other authority:

PERMANENT RULE (Including Expedited Rule Making)
Adopted under notice filed as WSR 09-14-136 on 07/01/2009 (date).
Describe any changes other than editing from proposed to adopted version: See WSR 10-01-174

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

Name: Andrew Kamali
Address: PO Box 47990 Olympia WA 98504-7990
Phone: (360) 584-6737
Fax: N/A
TTY: 711
Email: andrew.kamali@doh.wa.gov
Web site: www.sboh.wa.gov
Other: N/A

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	0	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:

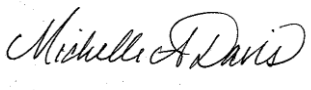
Negotiated rule making:	New	0	Amended	0	Repealed	0
Pilot rule making:	New	0	Amended	0	Repealed	0
Other alternative rule making:	New	0	Amended	0	Repealed	0

Date Adopted: 6/24/2024

Name: Michelle A. Davis

Title: Executive Director, Washington State Board of Health

Signature:



PREPROPOSAL STATEMENT OF INQUIRY

CR-101 (October 2017)
(Implements RCW 34.05.310)
Do **NOT** use for expedited rule making

CODE REVISER USE ONLY

OFFICE OF THE CODE REVISER
STATE OF WASHINGTON
FILED

DATE: June 20, 2024
TIME: 8:27 AM

WSR 24-13-117

Agency: Washington State Board of Health

Subject of possible rule making: Primary and Secondary School Environmental Health and Safety regulations. The State Board of Health (Board) is considering creating a new chapter (chapter 246-370 WAC) of draft rules for school environmental health and safety. The Board will consider establishing minimum statewide health and safety standards for schools, formalizing school environmental health and safety inspection procedures, and repealing chapters 246-366 and 246-366A WAC.

Statutes authorizing the agency to adopt rules on this subject: RCW 43.20.050 and Engrossed Substitute Senate Bill 5950 (Chapter 376, Laws of 2024), Section 222, subsection 159.

Reasons why rules on this subject may be needed and what they might accomplish: During the 2024 legislative session, the Legislature directed the Board to review the current rules and draft new proposed school environmental health and safety rules in collaboration with a technical advisory committee and other state agencies. The Board has the authority under RCW 43.20.050 to adopt rules controlling public health for schools. The current school environmental health and safety rules are outdated and need modernization. The intended goal of this rule-making is to replace the existing rules in chapters 246-366 and 246-366A WAC and develop minimum environmental health and safety standards alongside an implementation plan that helps to prioritize pieces of the rule by section or subject matter to improve the health and safety of students.

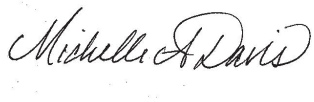
Identify other federal and state agencies that regulate this subject and the process coordinating the rule with these agencies: The Board will lead a technical advisory committee with representatives from the Department of Health, Office of the Superintendent of Public Instruction, school districts, local health jurisdictions, Washington Association of School Administrators, Washington State School Directors' Association, Washington Association of Maintenance and Operations Administrators, and Washington Association of School Business Officials. The Board will also coordinate with the Department of Labor and Industries, the State Building Code Council, the Department of Commerce, and the State Board of Education.

Process for developing new rule (check all that apply):

- ☐ Negotiated rule making
- ☐ Pilot rule making
- ☐ Agency study
- ☒ Other (describe) The Board will use a collaborative rule-making approach, via a technical advisory committee, in developing the proposed rules

Interested parties can participate in the decision to adopt the new rule and formulation of the proposed rule before publication by contacting:

	(If necessary)
Name: Andrew Kamali	Name:
Address: PO Box 47990, Olympia, WA 98504-7790	Address:
Phone:360-584-6737	Phone:
Fax: 360-236-4088	Fax:
TTY: 711	TTY:
Email: andrew.kamali@sboh.wa.gov	Email:
Web site:	Web site:
Other:	Other:

Additional comments: To be added to the listserv for notifications regarding this rule-making, email schoolehs@sboh.wa.gov with the subject line "School Rules Review Email Notification." The Board in collaboration with the Department of Health will complete an environmental justice assessment for this rule-making.	
Date: 6/20/2024 **Name:** Michelle A. Davis, MPA **Title:** Executive Director, State Board of Health	**Signature:** 

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: July 23, 2024
TIME: 2:49 PM

WSR 24-15-129

Agency: State Board of Health

Effective date of rule:

Permanent Rules

☒ 31 days after filing.

☐ Other (specify) _____ **(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: Chapter 246-500 WAC – Handling of Human Remains. The State Board of Health adopted changes to WAC 245-500-050, WAC 246-500-053, and WAC 246-500-055 to align with changes in statute. These rules establish the requirements for remains reduced through cremation, alkaline hydrolysis, and natural organic reduction. Under these sections, the local registrar or the Department of Health may issue a burial-transfer permit for the disposition of cremated remains, remains reduced through alkaline hydrolysis, or remains reduced through natural organic reduction which have been in the lawful possession of any person, firm, corporation, or association for a holding period established by statute. The holding period is established in RCW 68.50.230, which was recently amended from 90 days to 45 days. The amendment also added counties to the list of entities that may lawfully dispose of human remains after the holding period. The adopted changes align the rule with the changes in statute.

Citation of rules affected by this order:

New: None
Repealed: None
Amended: WAC 246-500-050, WAC 246-500-053, and WAC 246-500-055
Suspended:

Statutory authority for adoption: RCW 43.20.050(2)(f)

Other authority:

PERMANENT RULE (Including Expedited Rule Making)

Adopted under notice filed as WSR 24-10-094 on April 30, 2024.

Describe any changes other than editing from proposed to adopted version:

There have been no changes to the proposed rule language filed under WSR 24-10-094.

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

Name:
Address:
Phone:
Fax:
TTY:
Email:
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	0	Amended	0	Repealed	0
Federal rules or standards:	New	0	Amended	0	Repealed	0
Recently enacted state statutes:	New	0	Amended	3	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:

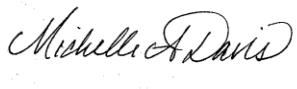
	New	0	Amended	0	Repealed	0

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	3	Repealed	0

Date Adopted: June 12, 2024	**Signature:**
Name: Michelle A. Davis	
Title: Executive Director, State Board of Health	

AMENDATORY SECTION (Amending WSR 21-01-039, filed 12/7/20, effective 1/7/21)

WAC 246-500-050 Human remains reduced through cremation. (1) Other than the provisions in this section and WAC 246-500-010, this chapter does not apply to human remains after cremation.

(2) A local registrar, in cooperation with the Washington state funeral and cemetery board, may issue a burial-transit permit for disposition of cremated human remains. The permit for the disposition of cremated remains may be used in connection with the transportation of cremated remains by common carrier or other means.

(3) The local registrar or the department of health may issue a burial-transit permit for the disposition of cremated human remains which have been in the lawful possession of any person, firm, corporation, county, or association for a period of ((~~ninety~~)) 45 days or more. This permit will specify that the disposition of cremated remains must be consistent with Washington state laws and rules.

AMENDATORY SECTION (Amending WSR 21-01-039, filed 12/7/20, effective 1/7/21)

WAC 246-500-053 Human remains reduced through alkaline hydrolysis. (1) Other than the provisions in this section and WAC 246-500-010, this chapter does not apply to human remains after alkaline hydrolysis.

(2) A hydrolysis facility must:

(a) Operate a high-temperature purpose built vessel, that reaches a minimum temperature of ((~~two hundred fifty~~)) 250 degrees Fahrenheit for a minimum of ((~~thirty~~)) 30 minutes during the reduction process; or

(b) Operate a purpose built vessel, for which third-party validation testing is provided demonstrating the reduction process destroys prions, and achieves sterilization in both the water and airspace, according to the manufacturer's specifications. The testing criteria must include a matrix-assisted laser desorption/ionization time of flight (MALDI-TOF) mass spectrometry peptide sizing analysis and a ((~~6~~)) six spore log reduction or greater in the level of *Bacillus* spores. An operator shall retain this documentation on-site and be able to provide it upon request to state or local health officials.

(3) A local registrar, in cooperation with the Washington state funeral and cemetery board, may issue a burial-transit permit for disposition of human remains reduced through alkaline hydrolysis. The permit for the disposition of remains reduced through alkaline hydrolysis may be used in connection with the transportation of remains reduced through alkaline hydrolysis by common carrier or other means.

(4) The local registrar or the department of health may issue a burial-transit permit for the disposition of human remains reduced through alkaline hydrolysis which have been in the lawful possession of any person, firm, corporation, county, or association for a period of ((~~ninety~~)) 45 days or more. This permit will specify that the disposition of remains reduced through alkaline hydrolysis must be consistent with Washington state laws and rules.

AMENDATORY SECTION (Amending WSR 23-09-027, filed 4/12/23, effective 5/13/23)

WAC 246-500-055 Human remains reduced through natural organic reduction. (1) Other than the provisions of this section and WAC 246-500-010, this chapter does not apply to human remains after natural organic reduction.

(2) A natural organic reduction facility operator shall:

(a) Collect material samples for analysis that are representative of each instance of natural organic reduction using a sampling method such as described in the U.S. Composting Council 2002 Test Methods for the Examination of Composting and Compost, Method 02.01-A through E;

(b) Analyze each instance of reduced human remains for physical contaminants. Reduced remains must have less than 0.01 mg/kg dry weight of physical contaminants which include, but are not limited to, intact bone, dental fillings, and medical implants;

(c) Analyze, using a third-party laboratory, the reduction facility's reduced human remains according to the following schedule:

(i) The reduction facility's initial 20 instances of reduced human remains for the parameters identified in Table 500-A, and any additional instances of human remains necessary to achieve 20 reductions meeting the limits identified in Table 500-A;

(ii) Following 20 reductions meeting limits outlined in Table 500-A, analyze, at minimum, 25 percent of a facility's monthly instances of reduced human remains for the parameters identified in Table 500-A until 80 total instances have met the requirements in Table 500-A;

(iii) The local health jurisdiction may require tests for additional parameters under (b) and (c) of this subsection;

(d) Not release any human remains that exceed the limits identified in Table 500-A;

(e) Prepare, maintain, and provide upon request by the local health jurisdiction, an annual report each calendar year. The annual report must detail the facility's activities during the previous calendar year and must include the following information:

(i) Name and address of the facility;

(ii) Calendar year covered by the report;

(iii) Annual quantity of reduced human remains;

(iv) Results of any laboratory analyses of reduced human remains; and

(v) Any additional information required by the local health jurisdiction; and

(f) Test for arsenic, cadmium, lead, mercury, and selenium, and either fecal coliform or salmonella in reduced human remains to meet the testing parameters and limits identified in Table 500-A.

Table 500-A
Testing Parameters

Metals and other testing parameters	Limit (mg/kg dry weight), unless otherwise specified
Fecal coliform	< 1,000 Most probable number per gram of total solids (dry weight)
or	

Salmonella	< 3 Most probable number per 4 grams of total solids (dry weight)
and	
Arsenic	≤ 20 ppm
Cadmium	≤ 10 ppm
Lead	≤ 150 ppm
Mercury	≤ 8 ppm
Selenium	≤ 18 ppm

(3) A local registrar, in cooperation with the Washington state funeral and cemetery board, may issue a burial-transit permit for disposition of human remains reduced through natural organic reduction. The permit for the disposition of remains reduced through natural organic reduction may be used in connection with the transportation of remains reduced through natural organic reduction by common carrier or other means.

(4) The local registrar or the department of health may issue a burial-transit permit for the disposition of human remains reduced through natural organic reduction which have been in the lawful possession of any person, firm, corporation, county, or association for a period of ((~~90~~)) 45 days or more. This permit will specify that the disposition of remains reduced through natural organic reduction must be consistent with Washington state laws and rules.

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

June 24, 2024

David Belanger

Sent Via Email

Dear Mr. Belanger:

This letter provides formal notice that the Washington State Board of Health (Board) denied your petition for rulemaking, submitted on May 8, 2024, at its regular business meeting on June 12, 2024, for the reasons described below.

The petition asked the Board to revise WAC 246-260-131, Operation of Water Recreation Facilities, subsections (6)(b)(i) and(ii), by removing an allowance for the substitution of a swim or dive coach or scuba diver instructor in place of a lifeguard.

Prior to the meeting, Board members were provided with all materials that were submitted relating to the petition. Board staff provided background information about the scope and intent of the existing rule and staff from the Department of Health discussed its current recommendations. The Board and the Department recognize the substitution of lifeguards with coaches as an area for review.

The Board opted to decline the petition because the water recreation rules are currently open for rulemaking. As a piece of the rulemaking, the Board is considering the Center for Disease Control's Model Aquatic Health Code (MAHC) which does not currently recognize the substitution of coaches for lifeguards. Members stated that this issue will already be considered in the coming months, thus the petition is not necessary to bring it under consideration for rulemaking.

RCW 34.05.330(3) allows a person to appeal a petition's denial to the Governor within 30 days of the denial.

Sincerely,

Patty Hayes

Patty Hayes, MPH
Chair

STATE OF WASHINGTON
OFFICE OF GOVERNOR JAY INSLEE

July 3, 2024

William Osmunson, DDS, MPH
Washington Action for Safe Water
Via email: bill@teachingsmiles.com

Re: APA Appeal –Washington Administrative Code (WAC) 246-290-220

Dear Dr. Osmunson:

On May 22, 2024, the Governor's Office received the appeal you filed in response to the Washington State Board of Health's (Board) decision to deny your petition to amend WAC 246-390-220. You petitioned the Board to add new language recommending specific parameters for the ingestion of fluoride from drinking water and toothpaste for pregnant mothers, infants, and children. Under RCW 34.05.330(3), I may consider appeals of an agency's denial of a petition to amend an existing rule.

Specifically, you requested that the Board add a subsection to WAC 246-390-220 that would establish a "minimum label to protect the development of the most vulnerable" that would name the potential risks of fluoride ingestion. You stated that the Board has failed to comply with its obligation under RCW 43.20.050(2)(a) to "adopt rules for group A public water systems, as defined in RCW 70.119A.020, necessary to assure safe and reliable public drinking water and to protect the public health." You also stated that the Board considers and publicly cites the benefit of fluoridation in public drinking water but does not fully consider the data that you believe demonstrates the harm caused by fluoride. You believe that the addition of this new subsection, providing specific limits for the ingestion of fluoride in drinking water, is necessary for the Board to fulfill its obligation under state law.

Since 2015, the Board has considered the topic of community water fluoridation seven times, including this most recent petition, as a stand-alone topic or part of broader discussions related to oral health. Each time, the Board examined the topic carefully. Additionally, between 2010 and 2015, the Board received and considered 15 petitions for rulemaking related to community water fluoridation. At its March 13, 2024, meeting, while discussing your petition, Board members clarified that decisions regarding whether or not a water supply is to be fluoridated are made locally, as the state does not require fluoridation in public water systems. Rather than it being the Board's role to set parameters for fluoride ingestion via drinking water, Board members stated that medical and dental providers are better positioned to provide individualized guidance related to fluoride ingestion.

The Board has previously considered evidence of fluoride's possible negative effects, evidence similar to that which is cited in your petition and attached to your current appeal. The Board has repeatedly determined that its current approach regarding fluoridation is sound, and that no additional rules are needed for it to ensure safe and reliable public drinking water.

Under RCW 34.05.330, an individual may appeal an agency's decision within 30 days of its receipt. In this case, your appeal to the Governor was not received until May 22, 2024, even though the Board's decision was dated "March 2024." While the denial date is imprecise, it was issued at least 52 days prior to the receipt of your appeal to the Governor, rendering your appeal more than three weeks late. However, if your appeal were timely filed, I would not be persuaded that the Board erred in denying your petition to amend WAC 246-290-220. For all the reasons stated above, I affirm the Board's decision and deny your petition.

Sincerely,

Jay Inslee
Governor of Washington

WASHINGTON STATE BOARD OF HEALTH

Placeholder for Presentation

Additional materials for Agenda Item 5,
Seattle & King County Local Health Update:
Public Health Approach to Overdose Prevention
will be added soon.

WASHINGTON STATE BOARD OF HEALTH

Date: August 7, 2024

To: Washington State Board of Health Members

From: Kate Dean, Board Member

Subject: Variance Request (Yakima) – Chapter 246-262-010(21) WAC, Definition of Diving Envelope, & 246-262-060(5)(vi) Diving envelope Requirements

Background and Summary:

RCW 70.90.120 authorizes the State Board of Health (Board) to adopt rules governing safety, sanitation, and water quality of water recreation facilities. WAC 246-262-160 sets the process for variance requests. The Board has the sole discretion to approve variance requests, if the Board determines the data and our research provides sufficient evidence that the variance will adequately protect public health and safety. Upon receipt of a request, the Board will strive to make a determination on the variance request within sixty days.

On June 21, 2024, the Board received a variance request from Brooke Hanley requesting the approval of 3 separate pieces of equipment under sections 246-262-010(21) WAC, definition of diving envelope, & 246-262-060(5)(vi) diving envelope requirements. The equipment includes a NinjaCross Obstacle Course, AquaZip'n Rope Swing, and a climbing wall.

Today, Board and Department of Health staff will introduce the variance requests for consideration by the Board. Due to the large size of supporting documentation, staff need additional time to complete the review and to determine compliance with the health and safety standards as outlined in Chapter 246-262 WAC.

Staff recommends that the Board hold a special meeting in late August to allow for additional time to review the requests and to provide a timely response to the requester.

Recommended Board Actions

This is an information briefing.

Staff

Andrew Kamali

PO Box 47990 • Olympia, WA 98504-7990
360-236-4110 • wsboh@sboh.wa.gov • sboh.wa.gov

Washington State Board of Health
Policy & Procedure

Policy Number:	**2018-001**
Subject:	**Handling Variances, Exemptions, and Waivers in State Board of Health Rules**
Approved Date:	**August 8, 2018**

Background

The State Board of Health (Board) has broad authority to adopt rules on a number of public health and safety topics. These rules may include provisions regarding variances, exemptions, or waivers allowed under the rules, which may be granted by the Washington Department of Health (Department), local health jurisdictions, or the Board.

Variances, exemptions, and waivers are different types of exceptions that support flexible and reasonable application of Board rules depending on the particular situation. The terms are not defined in the regulations referenced below, but the general dictionary definitions of these words can be used to understand the distinctions between them:

- **Variance** means a modified means of meeting a rule requirement.
- **Exemption** means relief from a rule requirement.
- **Waiver** means the setting aside of a rule requirement.

As outlined in Table 1 of this policy, one or more of these exception provisions are used in twelve Board rules. In addition, state rules on reclaimed water administered by the Washington Department of Ecology reference Board waiver authority in chapter 246-290 WAC, *Group A Public Water Supplies*, for approval of direct potable reuse of reclaimed water.

In most cases, authority to grant exceptions is assigned to the Department, local board of health, or local health officer. Only three rules directly involve the Board. Two rules assign decision-making authority to the Board and a third provides the Board with optional approval authority:

1) WAC 246-262-160: Authorizes the Board to act on variance requests to requirements of chapter 246-262 WAC, *Recreational Water Contact Facilities*.
2) WAC 246-290-060: Authorizes the Board to act on requests for variances, exemptions, or waivers to requirements of chapter 246-290 WAC, *Group A Public Water Supplies*.
3) WAC 246-260-201: Authorizes the Department or local health officer to act on variance requests to requirements of chapter 246-260 WAC, *Water Recreation Facilities*. However, the Board may require that variance requests be submitted for Board review and approval.

Policy Statement

Variances, exemptions, and waivers are valuable tools in Board rules. The Board plays a limited role directly granting such exceptions in implementing the rules. Where required in rules, the Board will consider requests for variances, exemptions, and waivers under the procedure outlined below.

New or revised Board rules can help refine the Board's limited role granting these exceptions and help align provisions for variances, exemptions, and waivers across Board rules. The following should be taken into consideration as Board rules containing these provisions are next updated:

- Variances, exemptions, and waivers should be clearly defined and correctly applied in all Board rules.
- Approval authority for variances, exemptions, and waivers should rest with the health agency where it best protects public health and safety, ensures accountability, and is most easily administered.
- Unless it provides needed flexibility, rules granting variances, exemptions, or waivers should avoid listing multiple or optional approval authorities and should instead authorize one agency.
- For ease of administration, rules authorizing local health jurisdictions to approve variances, exemptions, or waivers should identify local health officers rather than local boards of health as the approval authority.
- Provisions in chapter 246-260 WAC and chapter 246-262 WAC should be aligned—or combined if the rules are consolidated—and should assign approval authority to either the Department or local health officer.
- Where meaningful, annual reporting to the Board on activity related to variances, exemptions, and waivers can be required. If required, such reporting should occur consistently.

Board Procedure

Where required in rule, the Board will consider requests for variances, exemptions, and waivers. As noted previously, two rules require Board action: chapter 246-262 WAC, *Recreational Water Contact Facilities*, and chapter 246-290 WAC, *Group A Public Water Supplies*. Chapter 246-262 WAC lacks any process requirements, so the following procedures apply in full. In contrast, WAC 246-290-060 and Policy J.28 of the Department's Office of Drinking Water outline a few process requirements that should be applied to dovetail with Board process requirements starting at the point of application to the Department. Variance and exemption requests under WAC 246-290-060 must be considered in accordance with 40 CFR s. 141.4 (variances and exemptions to National Primary Drinking Water Regulations).

Submittal of Requests

Requests should be addressed to the Board Chair and signed by an authorized agent of the owner/operator of the facility or utility (not a third-party agent). With applications to the Department of Health under WAC 246-290-060, the Board Chair should be copied. The request should include and describe the following:

- name and address of the facility or utility, name of the owner/operator, and name and information for the lead contact;
- rule citation authorizing Board action;
- the specific rule or rules for which a variance, exemption, or waiver is sought;
- the situation, need, and justification for the request;
- supporting documentation and technical analysis developed or used to assess the request and meet the intent of the regulation to ensure health and safety;
- steps taken to mitigate concerns or risks; and
- commitment to carry out conditions or follow–up actions that may be applied to the request.

Receipt and Notification

Upon receipt of a request, Board staff, in consultation with the Executive Director, will respond to the requester within five business days acknowledging receipt of the request. The Executive Director or staff will notify Board members that a request has been received and will be brought to the Board for consideration at the next regularly scheduled Board meeting. The Board will strive to complete its

work and respond to a request within 60 days. If no regular meeting is scheduled within 60 days of receipt, or if the agenda for the regular meeting cannot accommodate review of the request, or if staff need more time to complete its review, the request may be addressed at the following Board meeting. The Executive Director or staff will notify the requester of dates and times that the Board is scheduled to meet and consider the request. As part of its initial review, the Board will determine whether a request falls within its authority to review. If the Board determines that a request falls outside the scope of its authority, staff will notify the requester of this and close the request.

Review and Board Action

The Board may identify a sponsoring Board member and will direct staff to review the request on the basis of relevant laws, industry standards, health and safety guidelines, and other relevant material. Board staff will coordinate and consult with the Department and other subject matter experts as appropriate in reviewing the request.

The sponsor and Board staff assigned to review the request will present their findings and recommendation to the Board. The Board may ask a Department representative to provide a recommendation or technical analysis to help inform Board discussions. The Board may invite the requester to present the request and respond to questions from the Board at its meeting.

Following review, the Board may grant the request, grant the request with conditions, deny the request, or ask for additional information before acting on the request. The Board may grant a variance, exemption, or waiver from rule requirements if it meets the substantive requirements of the rule allowing a variance, exemption, or waiver. Variances and exemptions granted to public water systems must be conditioned on a compliance schedule in accordance with WAC 246-290-060(6). The decision will be made by the Board in public meeting. Once the Board has made its decision, Board staff will follow up with a written notice to the requester. If the Board denies a request, the notice will contain information about how the requester may appeal the decision.

Water Recreation Variance Request, Aquatic Center at MLK Jr. Park

Chapter 246-262 WAC

Andrew Kamali, Policy Advisor - August 7, 2024

WASHINGTON STATE BOARD OF HEALTH

David DeLong,

Water Recreation Program Lead

Aquatic Center at MLK Jr Park Variances

- Purpose
- Review variance requirements
- Cheney Variance Request(s)
  - Aqua Climb Climbing Wall
  - Aqua Zip’N Rope Swing
  - Ninja Cross Obstacle Course

Background

WAC 246-262-160

Variance.

The board may grant a variance from requirements of chapter 246-262 WAC if, in the sole discretion of the board, data and/or research provides sufficient evidence that the recreational water contact Facility (attraction, device, equipment, procedure, etc.), will adequately protect public health and safety, as well as water quality.

Background

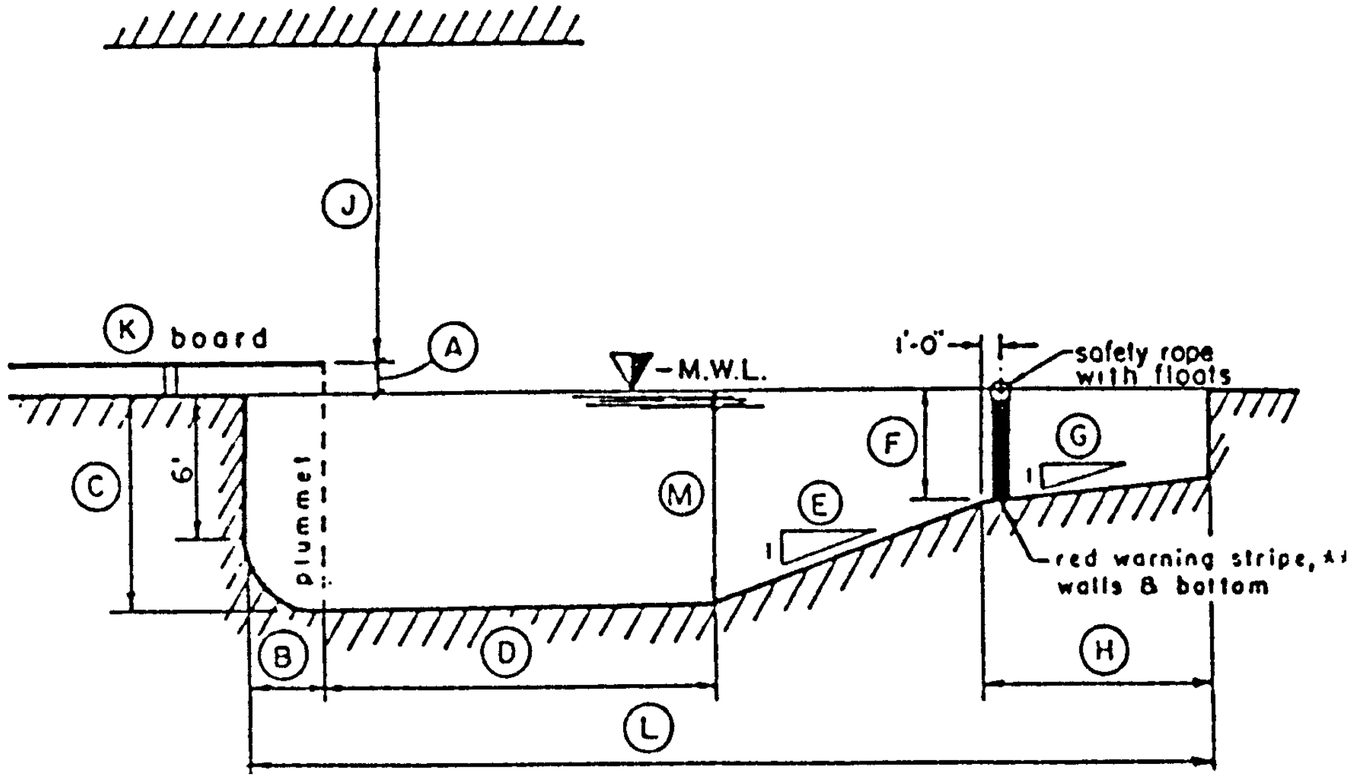
- **WAC 246-262-010(21)** "Diving envelope" means the minimum dimensions of an area within the pool necessary to provide entry from a diving board, platform, or attraction segment where users enter above pool water level.

- **WAC 246-262-060(5)(vi)** *describes diving envelope requirements*

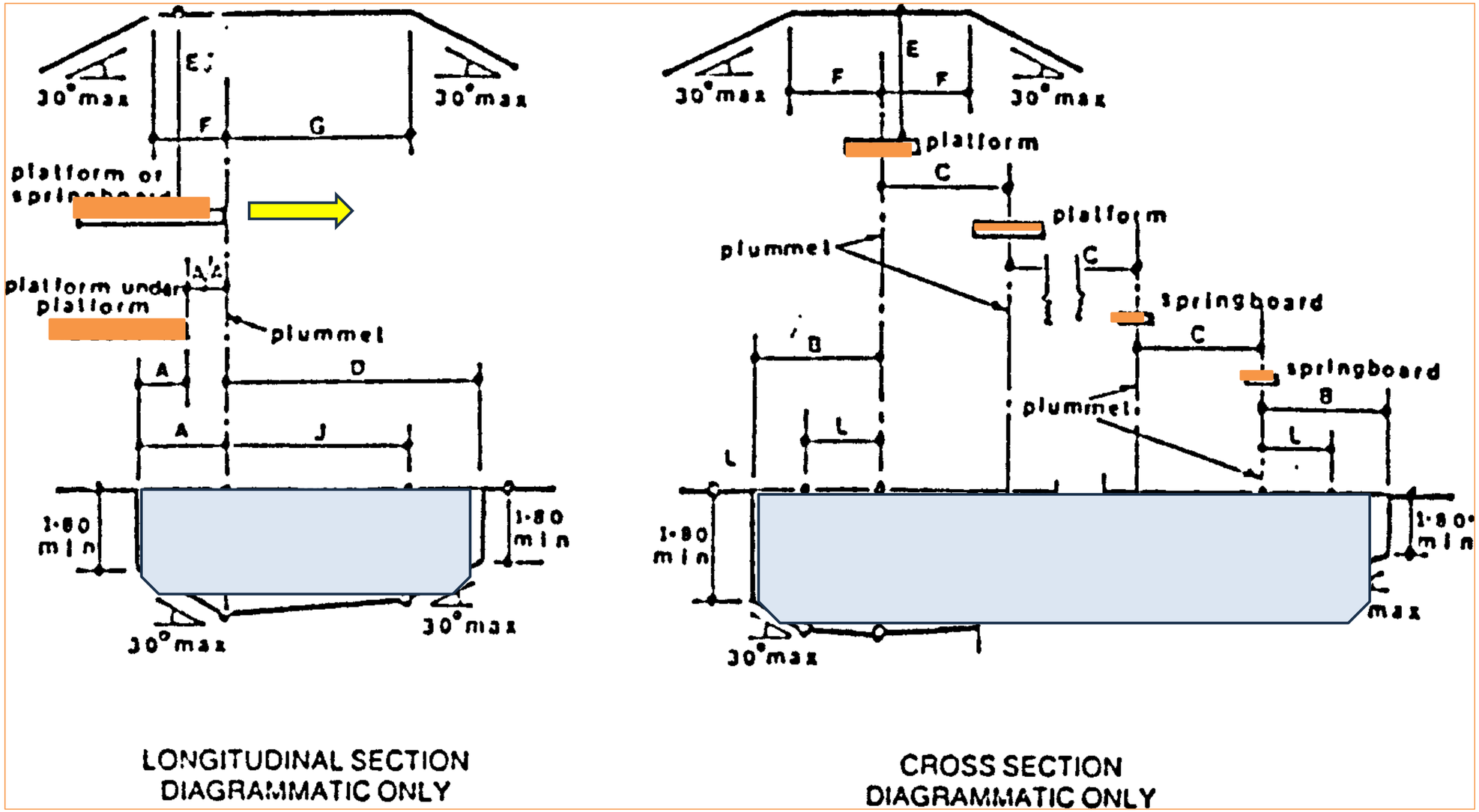
Diving Envelopes

Non-competitive Diving

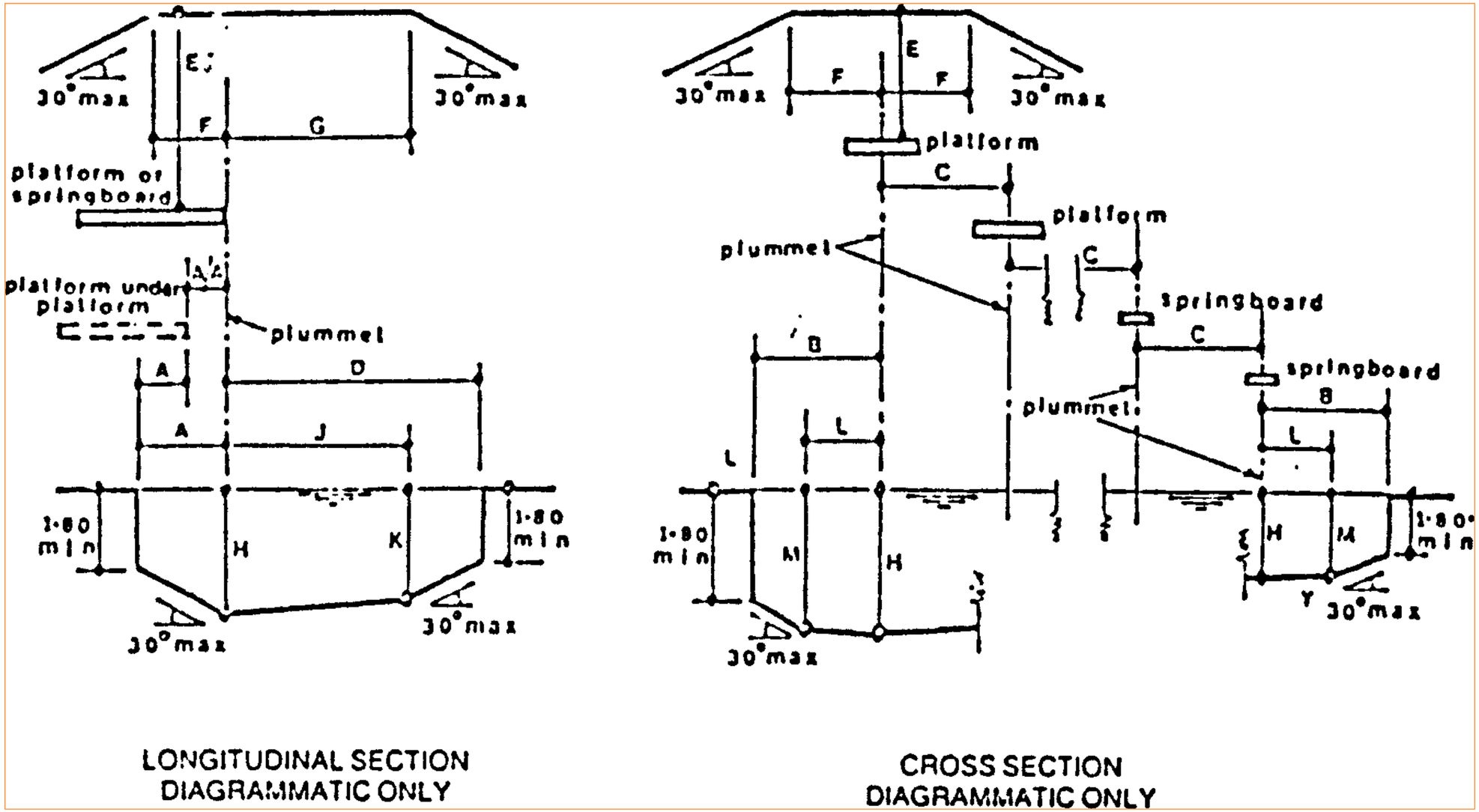
- Council for National Cooperation in Aquatics Standards
- 20-inch board height
- Depth at plummet 9-10 feet
- Length 16 feet



Competitive Diving Envelopes

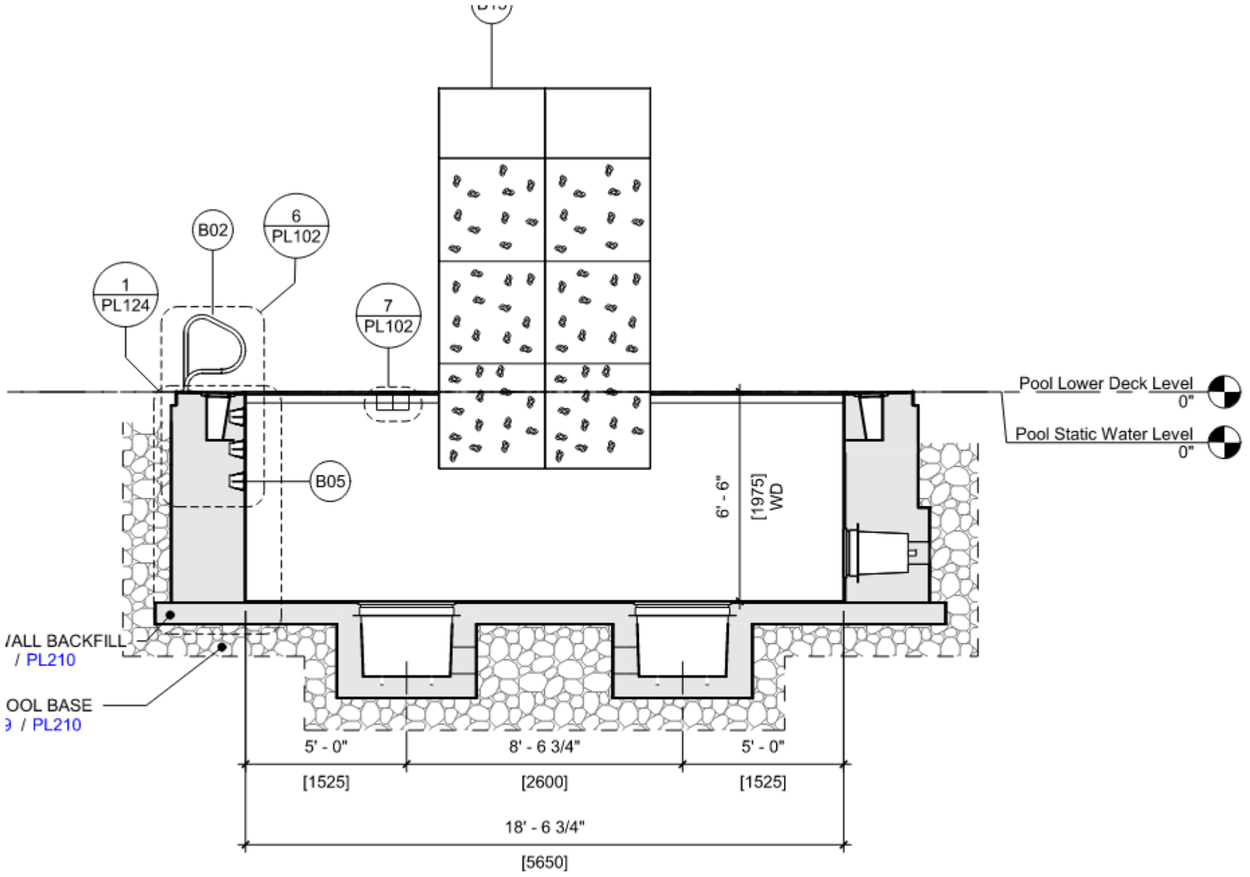


Competitive Diving Envelopes



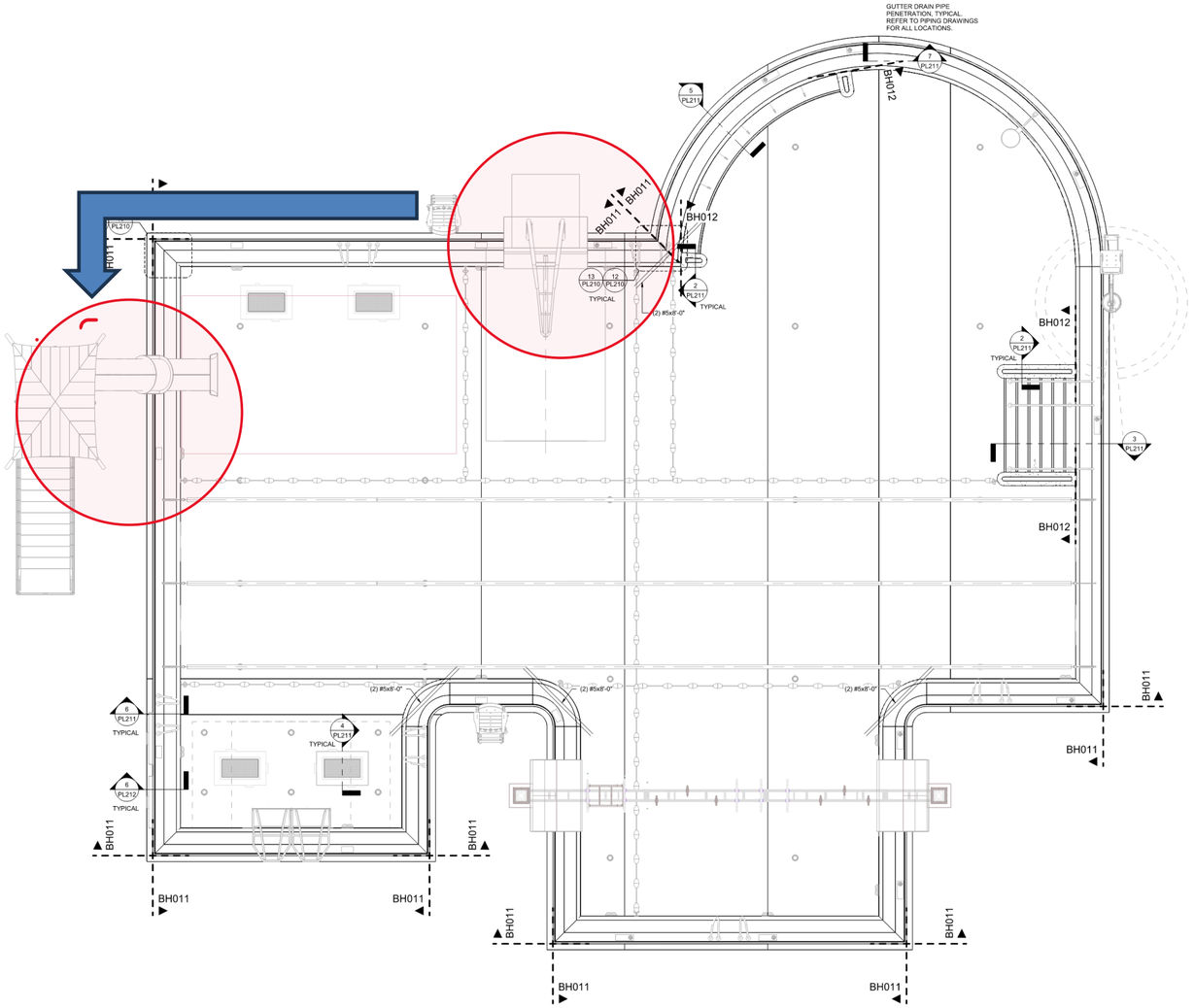
MLK Jr Park
Climbing Wall

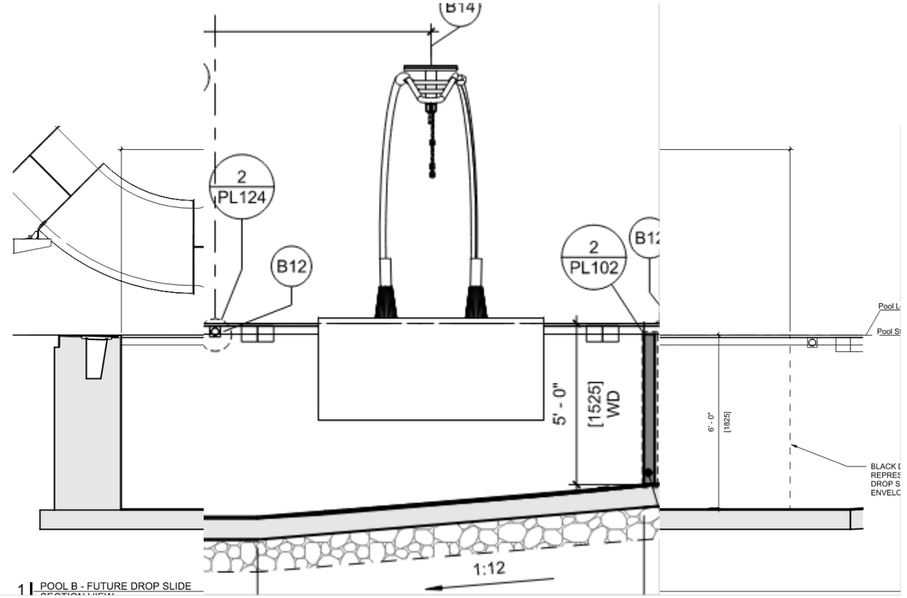
MLK Jr Park – Climbing Wall



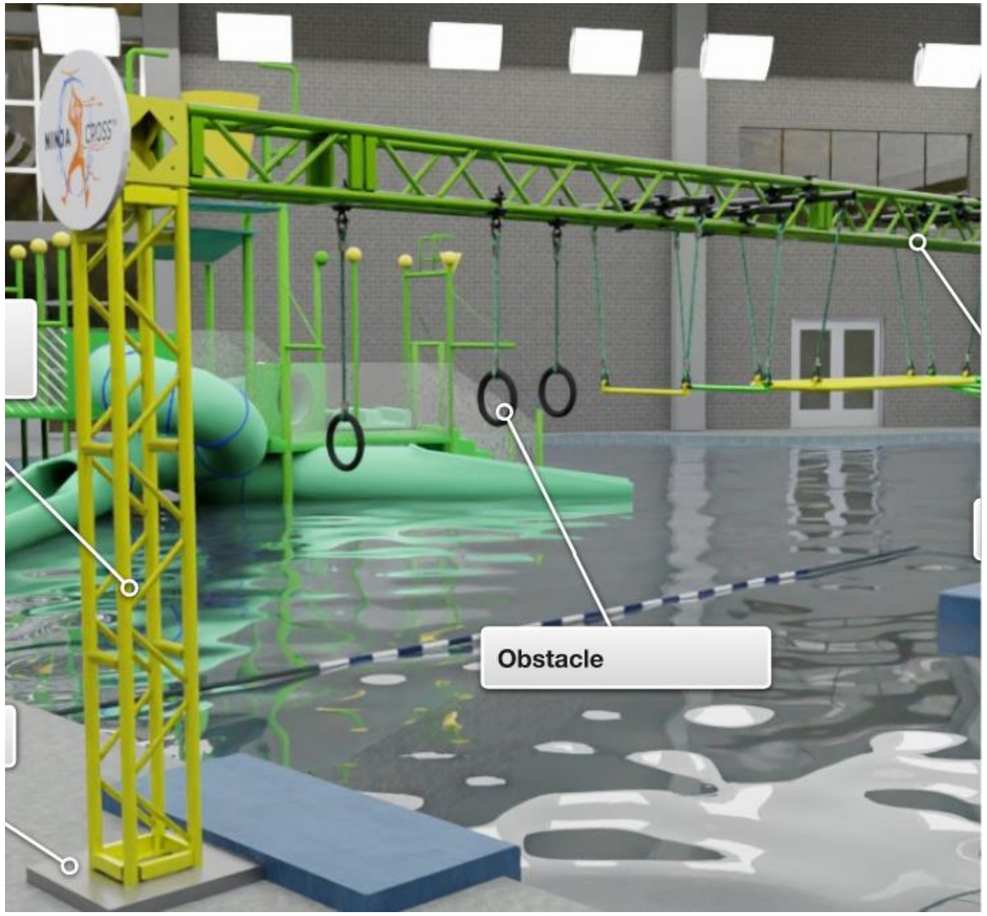
MLK Jr Park Pool
Aqua Zip

MLK Jr Park Pool – Aqua Zip

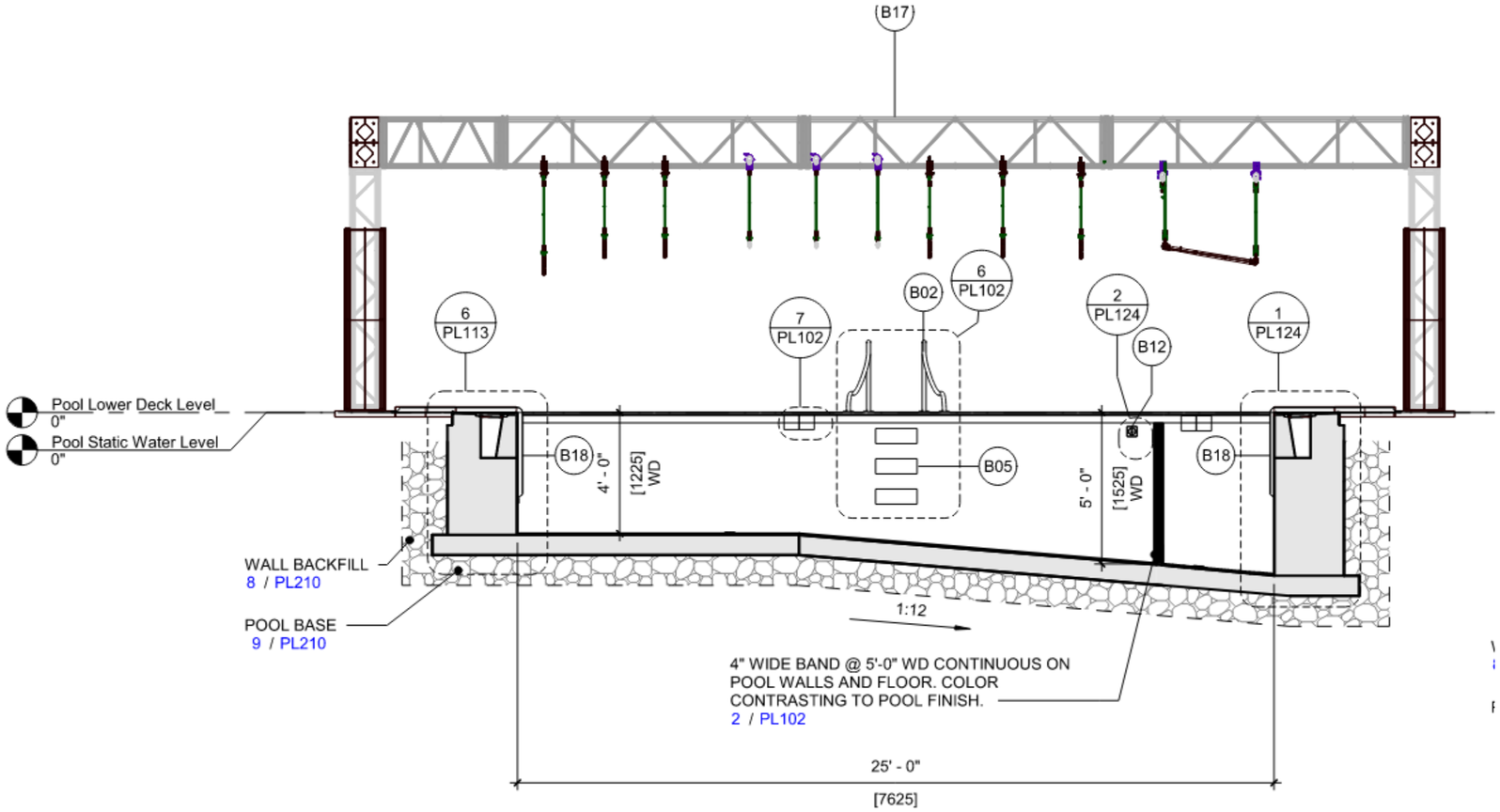




MLK Jr Park Pool
Ninja Cross



MLK Jr Park – Ninja Cross



2 | POOL B - ACTIVITY POOL

MLK Jr Park Variances Status

- Department of Health and State Board of Health staff are still reviewing the data, arguments, and mitigations proposed by the facility and are not prepared to give an evaluation of or recommendation to the Board at this time.

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.

NAC
ARCHITECTURE

Patty Hayes, Board Chair
Washington State Board of Health
PO Box 47990
Olympia, WA 98504-7990

AQUATIC CENTER at MLK JR. PARK, Yakima

Variance Letter Date: 2024.06.20

STATE IDENTIFICATION: State ID Facility #: F0476 Project #:2024003

Facility Information:
Aquatic Center at MLK Jr. Park (New outdoor pool facility with 5,300sf pool building and two leisure pools)
Plan Submittal: Drawing Plans have been submitted for review.

Aquatic Center at MLK Jr. Park, City of Yakima

Owner Contact:	Ken Wilkinson	Phone: 509-576-6416
Owner Address:	129 N 2nd street	Yakima, WA 98901
Facility Address:	610 S 9th Street	Yakima, WA 98901
Owner Representative:	Brooke Hanley (NAC Architecture) 509-838-8240	

Variance Request Contact:
NAC Architecture: Brooke Hanley Phone: 509-838-8240 Email: bhanley@nacarchitecture.com

Variance Request Citation:
WAC 246-262-160 states *the board may grant a variance from requirements of chapter **246-262** WAC if, in the sole discretion of the board, data and/or research provides sufficient evidence that the RWCF (attraction, device, equipment, procedure, etc.), will adequately protect public health and safety, as well as water quality.*

Variance Request: Code language related to Diving Envelope (WAC 246-262-010(21) & WAC 246-262-060(5)(vi)) for the **NinjaCross Obstacle Course** attraction.

Items noted in review letter include:

- **NinjaCross Obstacle Course** attraction receiving pool shall conform to the CNCA or FINA standards (depth application and setbacks)

In the Department of Health review response letter issued by Justin Law dated May 22, 2024, Justin requests NAC Architecture (NAC) and WaterTechnology, Inc. (WTI) to address important concerns regarding public safety related to the receiving pool for the proposed **NinjaCross Obstacle Course** attraction in Pool B. The concern is to address the minimum depth of the pool to be compliant with the WAC 246-262-010(21) & WAC 246-262-060(5)(c)(vi) regarding diving envelopes for features where users enter the water from above the water surface.

On behalf of the City of Yakima, WA; NAC & WTI respectfully requests your consideration of the current pool depth design at the NinjaCross for the future Aquatic Center at MLK Jr. Park. To support this request we provide the attached information, engineering exhibits, and following commentary:

- The review letter states that the "diving envelope" from WAC 246-262-010(21) applies to **all attractions** where users enter above pool water level and therefore requires the CNCA (enter less than 20" above the water surface) or FINA (enter 20" or greater above the water surface) water depths. We submit that the attached independent engineering calculations for the **NinjaCross Obstacle Course** will demonstrate that the manufacturer's required water depths and the designed water depths provided at the Yakima Aquatic Center are sufficient to protect the safety of the users allowed to participate in this attraction. Calculations were completed for users ranging in height from 51" tall up to 72" tall, and weight ranging from 58lbs to 275lbs. The minimum user height is 48" and the maximum weight is 275lbs. The manufacturer's minimum depth requirement is 3'-6" feet depending on the obstacles purchased for the system. The current Yakima receiving pool water depth starts at 4'-0" at one end and slopes down to a depth of 5'-4" at the other end. Please review the attached engineering calculations in support of using the manufacturer's depth requirements in lieu of the CNCA or FINA diving envelope dimensions. See page 11 for a graphic section depicting an average user height compared and their position in or above the water using each obstacle. In the event that someone does drop from a height of 20" above the water, which is not anticipated for this attraction, the heaviest user would contact the pool floor feet-first with a force equivalent to contacting the ground after a 3.4" high jump on pavement. Quote from review letter, "The participant is expected to contact the pool bottom in a manner that is consistent with any shallow pool activities." The current design at the Yakima receiving pool exceeds these calculation assumptions by providing deeper water than the minimum required and will be lifeguarded to prevent people from incorrectly using the obstacles.

- WAC 246-262-060(5)(c)(vi) appears to apply specifically to "diving envelopes in pools or areas of pools designated for diving activities". The applicant submits that diving activities are generally defined as plunging into the water headfirst. Diving headfirst into water results in the need for deeper water to avoid a head & neck collision with the bottom of the pool which is different than a feet-first or tucked entry plunge where the body is significantly slowed in the first 2 feet of water. The **NinjaCross Obstacle Course** safety guidelines (provided in the exhibits) will note that users are required to enter the water in a feet-first manner. Diving from the unit is prohibited. The engineering calculations completed also assumes a feet-first plummet into the water. As users traverse the obstacles, they will generally have their feet dragging in the water and would not drop from a height above the water that is any different from stepping into the pool from the deck edge, see page 11.

- The Model Aquatic Health Code also addresses the complexity of “other aquatic features” like this and would suggest that the manufacturer recommendations for design and operation would be adequate to install the feature.

  4.12.10[A] Other Aquatic Features Other AQUATIC FEATURES not otherwise addressed in the CODE, including but not limited to climbing walls, inflatables, and play structures, shall not be installed unless designed and operated in accordance with all manufacturer’s installation and operations recommendations.

- ‘A-frame’ signs with all written safety guidelines will be publicly displayed near the NinjaCross (see page 100 for example) to meet the criteria of WAC 246-262-070(10).

- Safety padding rated for falls from 6ft or less are provided around the base of the truss structure and down the face of the pool wall to prevent injuries at the corner of the gutter.

- This pool will be lifeguarded at all times while in operation and the lifeguard staff will be the first line of defense to screen bathers to make sure they are experienced swimmers, instruct swimmers on proper use of the attraction, and direct proper swimmer circulation to and from the activity within the pool to avoid congestion or collisions. The **NinjaCross** will have a dedicated lifeguard to closely supervise the safety of swimmers when the attraction is open for use.

- Injury statistics requested by the review letter are not available from the manufacturer or another source at this time, but many aquatic centers across the country are replacing their lily pad crossing activities with the NinjaCross obstacle course because it has been deemed safer than having the lily pads anchored to the floor and permanently obscuring the view of the water below the pads from lifeguard supervision. The NinjaCross obstacles do not have those same supervision issues.

- The **NinjaCross** has also been designed and engineered to meet the following standards: Where applicable, NinjaCross follows guidelines from the MAHC (model aquatic health code). As for ASTM, NinjaCross has registered their products as fitness/sporting goods equipment which fall under ASTM F2461-18 Section 1.3.8 Exclusions "1.3.8 Sports equipment, fitness equipment, and diving equipment." This system’s patents and trademarks are registered under Sporting Goods & Fitness equipment and is not classified as an Amusement Ride.

- The City of Yakima specifically requested a pool design that would have a variety of intriguing activities for their patrons but would not need water deeper than 6-7ft. Pools deeper than 6-7ft come with their own safety risks and lifeguarding challenges. Shallow water is easier to supervise and guard. Rescues are much more likely to be needed in deep water where a bather in trouble cannot push off the bottom of the pool to bob back above the surface quickly until the lifeguard can assist them. Yakima is dedicated to making this facility fun while also as safe as possible for their community members and patrons.

- NAC submits that the design as described above and substantiated in the attached documentation meets the intent of providing a safe receiving pool for the **NinjaCross Obstacle Course** feature. NAC, WTI, and the City of Yakima respectfully requests a variance accordingly. If the State Board of Health has any follow-up conditions or actions required of the owner/operator, we are committed to implementing them.

NAC Architecture (NAC) has teamed with Water Technology (WTI) on numerous aquatic projects and so we have a history of producing these projects successfully. WTI has been designing Aquatic venues for over 40 years. WTI is widely known in the industry as one of the leading aquatic design firms in North America. As one of the industry's leaders, WTI has represented the waterpark industry during CPSC meetings on review of VGB rules and has also been involved in reviewing/editing sections of the MAHC. They are also represented in the Washington DOH committee to update the existing administrative code to adopt a more comprehensive aquatic code like the MAHC. The NAC and WTI commitment to safe aquatic facilities is proven. The design of the receiving pool at the **NinjaCross Obstacle Course** for the Yakima Aquatic Center will not put the health and safety of the public at risk. The City of Yakima, having operated a public pool for many years is experienced and committed to the safety and the welfare of their patrons.

On behalf of the City of Yakima, NAC Architecture would like to thank you for your consideration of this Variance Request. Please feel free to contact me with any questions you may have regarding this request.

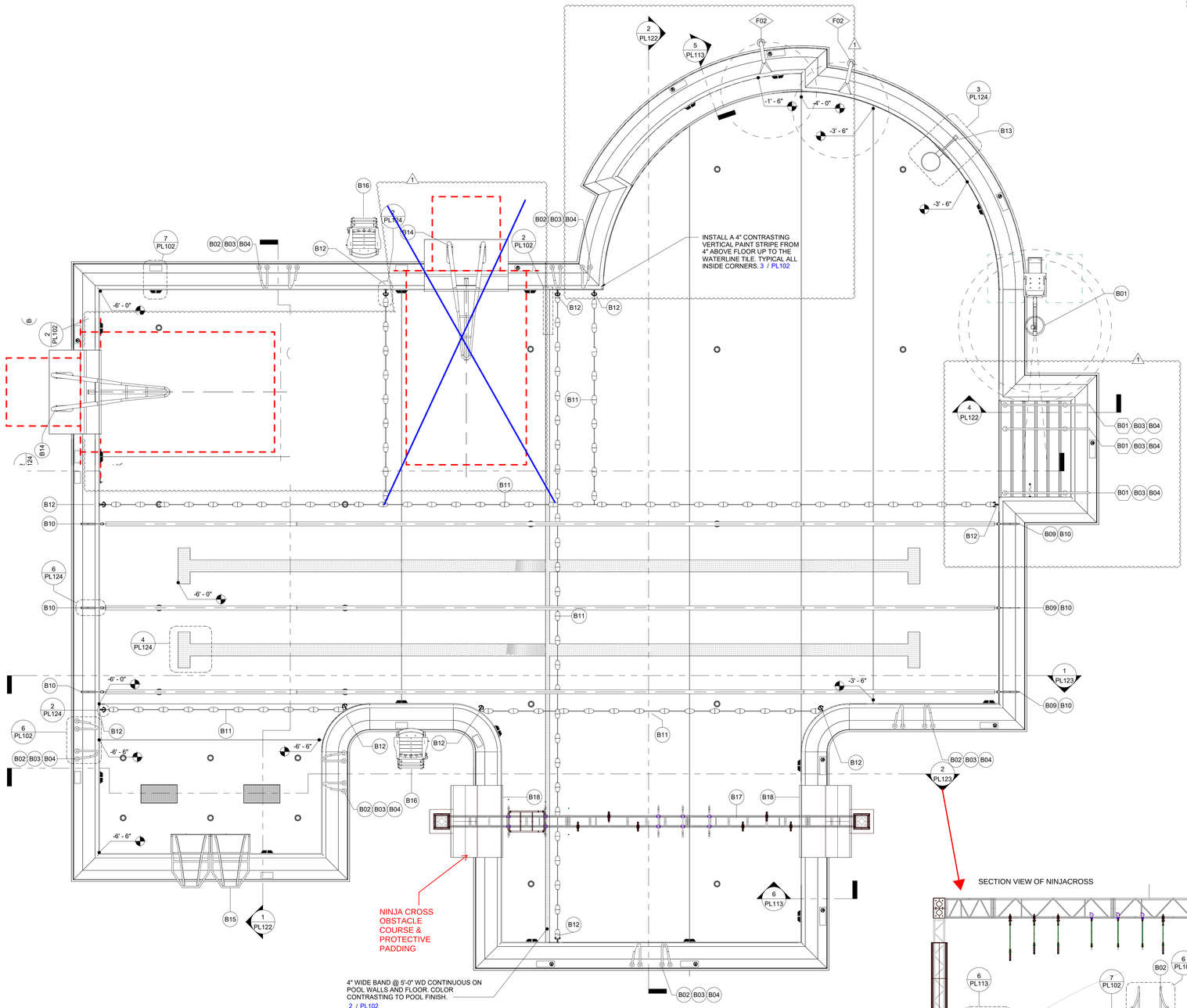
Thank you,

Brooke Hanley

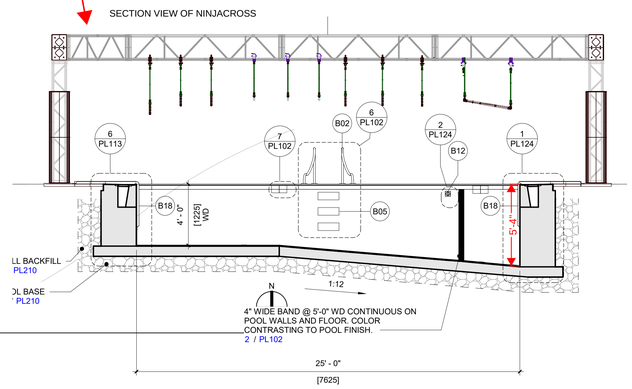
Brooke Hanley, AIA, Principal Architect, NAC Architecture

Attachments:

- NinjaCross Safety Information and Fall Zone Engineering, including a floor plan and section of the receiving pool for the Yakima Aquatic Center.



1 POOL B - ACTIVITY PLAN
PLAN VIEW
1/8" = 1'-0"



POOL B-ACTIVITY DATA

DESCRIPTION	QTY	UNITS		
POOL PERIMETER	314'-0"	FEET		
WATER SURFACE AREA	3,832	SQUARE FEET		
POOL WATER TEMPERATURE	84	°F		
POOL VOLUME	136,514	GALLONS		
SURGE TANK OPERATING VOLUME	7,415	GALLONS		
TOTAL VOLUME OF WATER	147,268	GALLONS		
CIRCULATION RATE	1,033	GPM		
TURNOVER/VOLUME/FLOW	60 MIN.	19,330 GAL.	322	GPM
TURNOVER/VOLUME/FLOW	180 MIN.	127,938 GAL.	711	GPM
FILTRATION RATE	12.66	GPM/FT²		
BACKWASH FLOW	306	GPM		
SURGE FACTOR	1.06	GAL/SQFT		
AVAILABLE SURGE CAPACITY IN SURGE TANK	4075	GALLONS		

SCHEDULE - BASIS OF DESIGN - POOL B

POOL ID	EQUIPMENT ID	EQUIPMENT	QTY	MANUFACTURER	DESCRIPTION
B	01	POOL LIFT	1	SR SMITH, AQUA CREEK, OR EQUAL	STANDARD ANCHORED, ROTATIONAL POOL LIFT, WITH 400 LB MINIMUM LIFTING CAPACITY. MUST MEET ALL APPLICABLE ADA REQUIREMENTS, WHILE MAINTAINING REQUIRED DECK CLEARANCE. PACKAGE TO INCLUDE ARMRESTS, ANCHOR, LIFT COVER, BATTERY CHARGER, AND CADDY
B	02	GRAB RAILS (PAIRS)	6	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	PRETZEL BEND STYLE, 1.50" OD x .120 WALL THICKNESS, 500 GRIT FINISH MIN.
B	03	ESCUTCHEON PLATE	34	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	STAINLESS STEEL ROUND ESCUTCHEON FOR 1.50" O.D. RAILS
B	04	WEDGE ANCHOR	34	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CAST BRONZE, 4-1/4" LONG, ACCEPTS 1.500" OD TUBING
B	05	IN-WALL STEPS	18	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	17-1/2" x 6", INJECTION MOLDED PLASTIC, PEBBLE TEXTURE, 1/4" WALL THICKNESS
B	09	LANE DIVIDERS	3	COMPETITOR SWIM PRODUCTS	4" WAVE QUELLING RACING LANE LINE, COLORS BY OWNER / ARCHITECT
B	10	DaiFLEX LANE LINE ANCHOR	6	DALDORADO	12" - NON-CORROSIVE PVC FLIP UP LANE LINE ANCHOR TO BE USED WITH DALDORADO PARALLEL GRATING. INCLUDES FLIP-UP HATCH, BASE UNIT, & SILICON COVERED SS BRAIDED STRAP EXTENSION WITH HOOK. CAN BE USED WITH THE DaiFLEX 8" OR 14" LANE LINE EXTENSION.
B	11	SAFETY ROPE	6	PARAGON AQUATICS	3/4" POLYETHYLENE ROPE WITH 5"x9" HANDI-LOCK FLOAT, VERIFY LENGTH WITH PLANS
B	12	CUP ANCHOR	10	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	4" SQUARE 304L SS ANCHOR AND 304L SS EYE BOLT
B	13	BASKETBALL HOOP	1	SR SMITH	STAINLESS STEEL BASKETBALL HOOP WITH ROCKSOLID ANCHOR
B	14	AQUA ZIPN	1	AQUACLIMB	DECK-MOUNTED OVERHEAD ROPE SWING, WITH SELF-RETRACTING TROLLEY, POWDER-COATED STAINLESS STEEL WITH HIGH TENACITY POLYESTER ROPE. INCLUDES SAFETY PAD-UNIVERSAL, WITH 316 SS HILTI FLUSH MOUNT CONCRETE ANCHORS.
B	15	AQUACLIMB	1	AQUACLIMB	2 WIDE X 3 HIGH AQUATIC CLIMBING WALL
B	16	LIFEGUARD CHAIR	2	TAILWIND, KEIFER, SPECTRUM AQUATICS, SR SMITH OR APPROVED EQUAL	RECYCLED PLASTIC WITH 304 SS HARDWARE, COLOR BY OWNER/ARCHITECT 40" SEAT HEIGHT (OWNER'S SAFETY CONSULTANT TO SPECIFY LOCATION.)
B	17	NINJACROSS	1	NINJACROSS	AQUATIC OBSTACLE COURSE
B	18	SAFETY PAD	3	PLAYTIME	WALL AND DECK SAFETY PAD AT NINJACROSS SYSTEM

SCHEDULE - CUSTOM RAILGOODS - POOL B

POOL ID	EQUIPMENT ID	EQUIPMENT	QTY	MANUFACTURER	DESCRIPTION
B	01	HAND RAIL	3	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CUSTOM FABRICATED, 316L SS, 1.50" OD x .120 WALL THICKNESS, 500 GRIT FINISH MIN.
B	02	HAND RAIL	2	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CUSTOM FABRICATED, 316L SS, 1.50" OD x .120 WALL THICKNESS, 500 GRIT FINISH MIN.

SCHEDULE - WATER FEATURE - POOL B

POOL ID	FEATURE ID	FEATURE	QTY	MANUFACTURER	DESCRIPTION	GPM (ea)	GPM (Total)
~~B~~	~~F01~~	~~DROP SLIDE~~	~~1~~	~~SPLASHTACULAR~~	~~FUTURE SLIDE. PROVIDE PIPING CAPPED ONLY~~	~~500~~	~~500~~
B	F02	WATER SPRAY	2	WATERPLAY	PIPE DELUGE-FAN SPRAY FEATURE	60	120

REVISIONS

REV.NO.	DESCRIPTION	DATE
1	CHANGE PROPOSAL	04/16/2024

CONFORMED SET

CITY OF YAKIMA
YAKIMA POOL
YAKIMA WA

WTI
WATER TECHNOLOGY, INC.
World Leaders in Aquatic Planning, Design and Engineering
100 Park Avenue | Beaver Dam, WI 53916
t 920.887.7375

NAC ARCHITECTURE
nacarchitecture.com
1203 WEST RIVERSIDE AVENUE
SPOKANE WA 99201
P:509.838.8240

NAC NO 111-22082
ISSUE DATE: 4/16/24
PROJECT NUMBER: 22314
DRAWN BY: T.ED
CHECKED BY: ACC

7893 REGISTERED ARCHITECT
MATTHEW W. FREEBY
STATE OF WASHINGTON
4/16/2024

POOL B - ACTIVITY POOL PLAN

PL120

June 12, 2024

Stephen Wagner
Director of Design & Development
NinjaCross™ Systems
stevew@ninjacrosssystems.com

Re: NinjaCross™ Drop Zone Assessment
Spokane Regional Health District
Project #2024-03-129

Stephen,

As requested, Eclipse Engineering has completed the drop zone assessment while using the NinjaCross™ System for the above noted jurisdiction. We utilized data from the CDC to determine the 10th, 50th and 90th percentile for male and female children aged 10, 12, and 14 years old. Using these participants in addition to the maximum user weight for the system, we analyzed a variety of drop orientations into a pool depth of 3'6" from 20" above the surface of the pool, which is comparable to jumping into the water from the pool deck.

While considering the drop orientations from the available system obstacles, we concluded that a drop into the water while using the NinjaCross™ System per its intended use and safety standards would not present a life safety hazard from impacting the water's surface or contacting the pool floor. When a participant who is using the system per design drops from an obstacle, their acceleration stops when they contact the water's surface, and their velocity is significantly reduced within the first 24", thus allowing the participant to contact the pool floor without a sudden impact. The participant is expected to contact the pool bottom in a manner consistent with any shallow pool activities.

Please note that accidents and injuries can happen in any situation regardless of prevention measures put in place. It is the responsibility of the facility, staff, and local governing agencies to follow the operation and maintenance manuals of the NinjaCross™ system to ensure proper use. Eclipse Engineering does not guarantee the health and safety of any participant of a NinjaCross™ system or the facility itself.

Please contact us with any questions.

Sincerely,
Eclipse Engineering, PC

Wade Ambach, P.E.
Project Manager
wambach@eclipse-engineering.com

Digitally signed by Rolf Henry Armstrong
DN: E=rarmstrong@eeimt.com, CN=Rolf Henry Armstrong, O="Eclipse Engineering, P.C.", L=Bend, S=Oregon, C=US
Date: 2024.06.14 01:41:26-07'00'

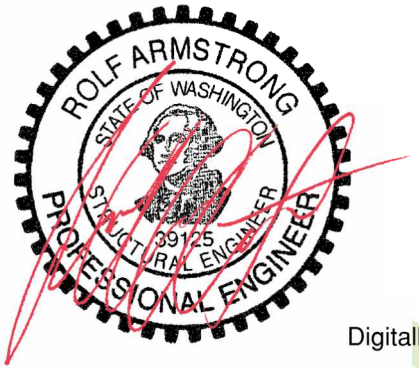
ROLF ARMSTRONG STATE OF WASHINGTON 19125 STRUCTURAL ENGINEER PROFESSIONAL ENGINEER

Rolf Armstrong, P.E., S.E.
CFO, Principal Engineer
rarmstrong@eclipse-engineering.com

Attachment: Safe Drop Zone Graphic

STRUCTURAL CALCULATIONS

NinjaCross – Drop Zone Assessment



Digitally signed by Rolf Henry Armstrong
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Date: 2024.06.14 01:40:52-07'00'

Prepared For:

NinjaCross Systems
Kyle W. Rieger, CPO
kyle@ninjacrosssystems.com

TABLE OF CONTENTS

Assumptions

A. DENSITY OF PERSON IS 980 KG/M^3.

B. COEFFICIENT OF DRAG OF PERSON DROPPING THROUGH WATER IS 1.0.

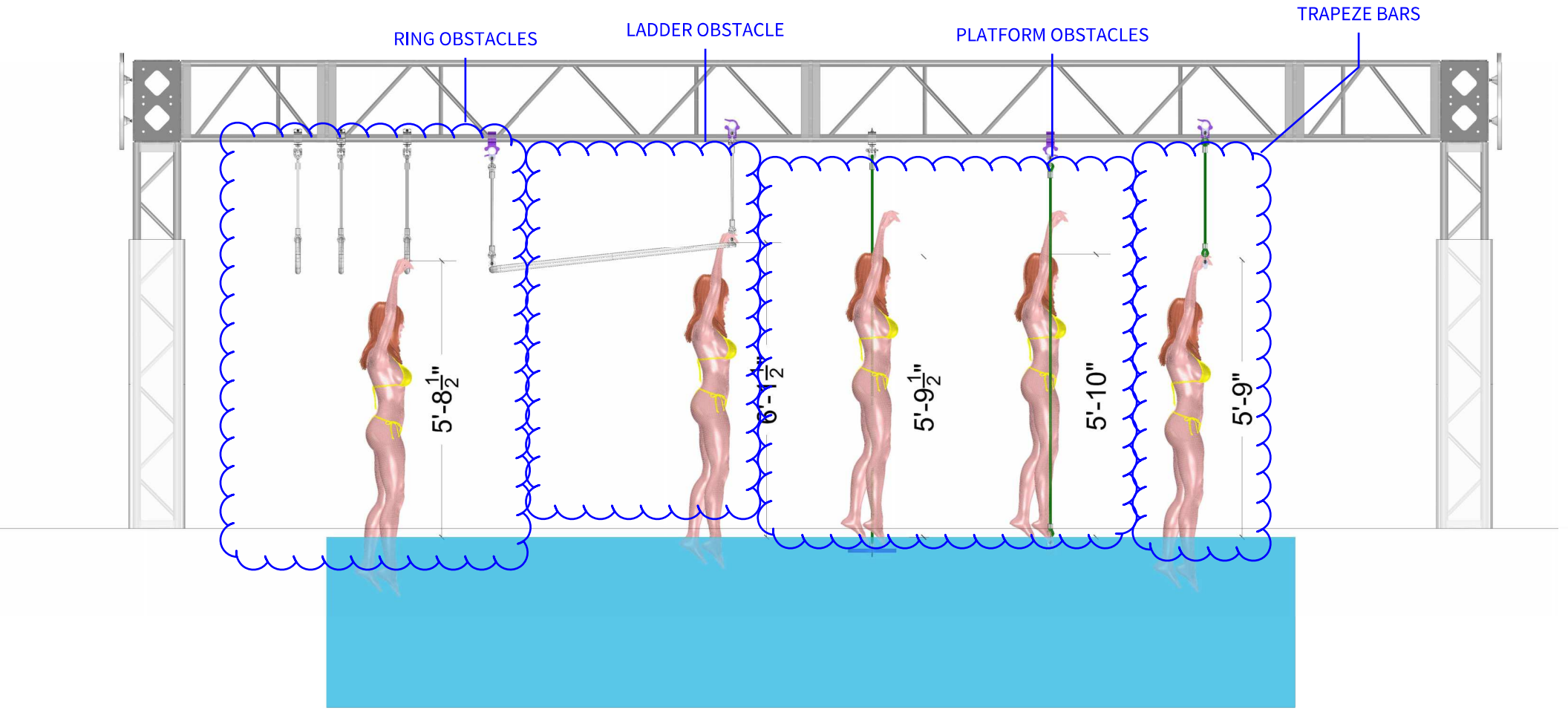
C. PERSON REMAINS STILL THROUGHOUT THE DROP UNTIL MAKING CONTACT WITH THE POOL FLOOR (IF APPLICABLE).

D. THE POOL DEPTH IS 3'-6".

E. PERSON DROPS WITH THEIR FEET 20 INCHES ABOVE THE TOP OF THE WATER.

F. PERSON DROPS FROM REST.

OBSTACLES AND USER CONDITIONS CONSIDERED IN EEPC FALL ZONE REVIEW
MINI NINJA SYSTEM



2 to 20 years: Girls
Stature-for-age and Weight-for-age percentiles

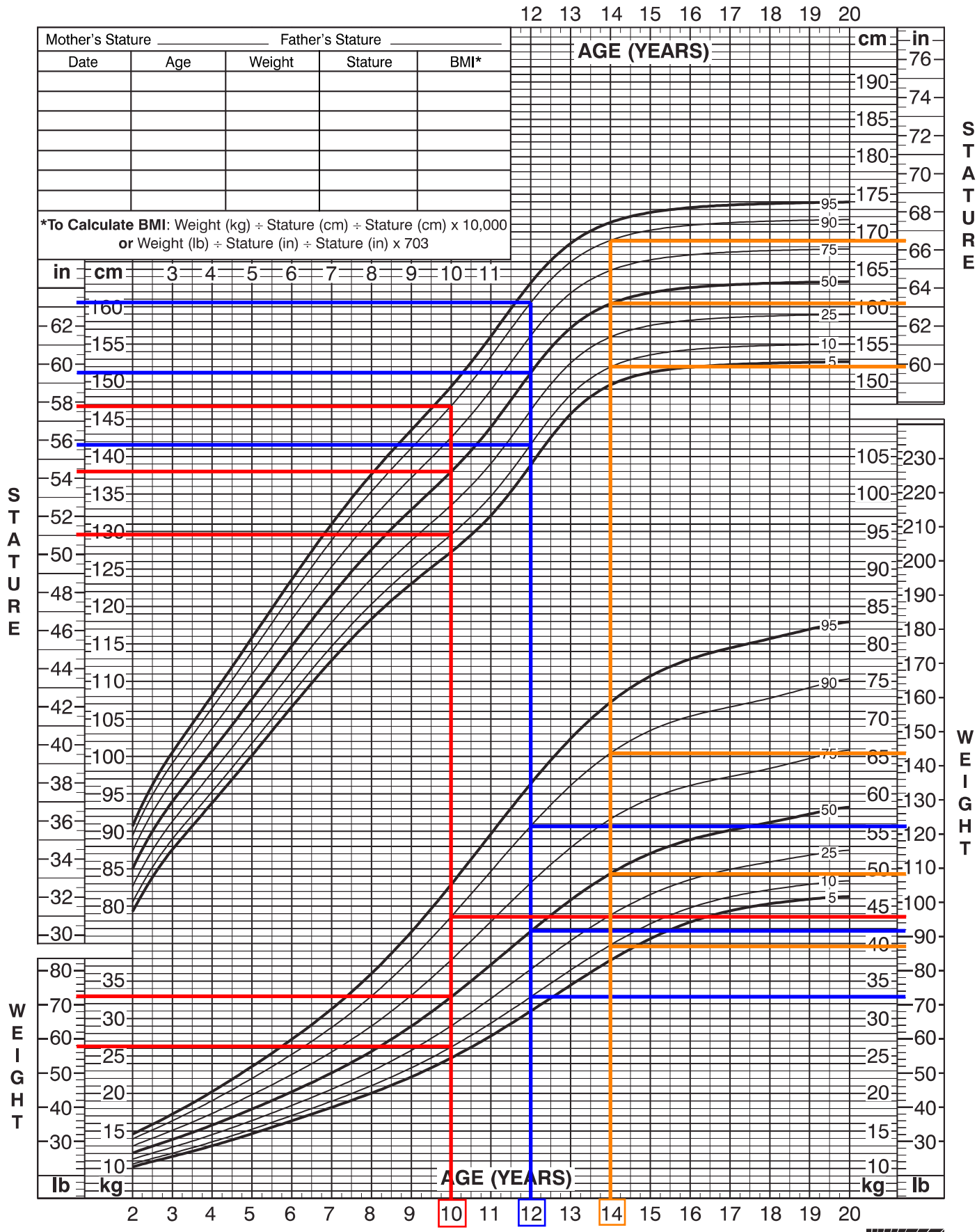
NAME _____

RECORD # _____

Mother's Stature _____ Father's Stature _____

Date	Age	Weight	Stature	BMI*

***To Calculate BMI**: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 11/21/00).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

2 to 20 years: Boys
Stature-for-age and Weight-for-age percentiles

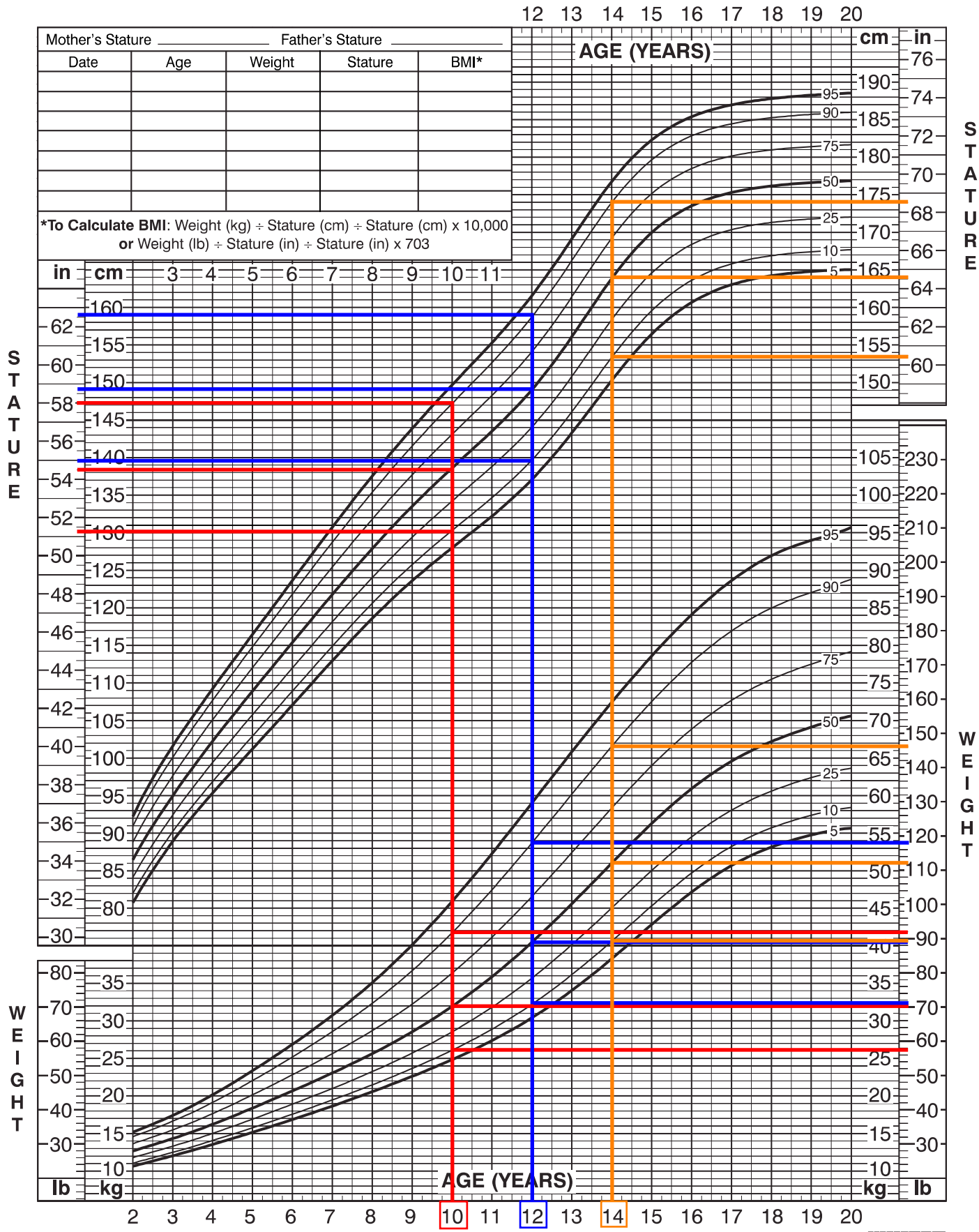
NAME _____

RECORD # _____

Mother's Stature _____ Father's Stature _____

Date	Age	Weight	Stature	BMI*

***To Calculate BMI**: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 11/21/00).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

Summary Data

Girls Stature & Weight for Age per CDC			
Age	Percentile	Weight (lb)	Height (in)
10	10	58	51
	50	72	54.5
	90	96	57.75
12	10	72	55.75
	50	92	59.5
	90	122	63.25
14	10	87	59.75
	50	108	63.25
	90	144	66.5

Boys Stature & Weight for Age per CDC			
Age	Percentile	Weight (lb)	Height (in)
10	10	58	51.25
	50	70	54.5
	90	92	58
12	10	71	55
	50	89	58.75
	90	118	62.75
14	10	89	60.5
	50	112	64.5
	90	146	68.5

NinjaCross System Design Participant	
Weight (lb)	Height (in)
275.0	72.0

NinjaCross System Design Participant Results				
	Vertical Drop	Diagonal Drop	Tucked Knee Drop	Horizontal Drop
Velocity at Pool Bottom	2.9 mph	2.9 mph	1.8 mph	0.0 mph
Effective Height of Drop	3.4 in	3.4 in	1.3 in	0.0 in

THE MAXIMUM VELOCITY AT WHICH THE PERSON HITS THE POOL FLOOR IS THAT WITH WHICH A PERSON HITS THE GROUND FROM A 3.4 INCH HEIGHT FALL.

$$mgh = \frac{1}{2}mv^2$$

Effective Height Above Ground $h = \frac{v^2}{2g}$

Please note that OSHA does not consider drops less than 4'-0" to require fall protection

Excerpt from https://www.osha.gov/fall-protection:
"OSHA requires that fall protection be provided at elevations of four feet in general industry workplaces."

Female Participant Results

Age	Percentile		Vertical Drop	Diagonal Drop	Tucked Knee Drop	Horizontal Drop
10	10	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	1.3 mph	0.9 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.7 in	0.3 in	0.0 in	0.0 in
12	10	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	1.3 mph	0.7 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.7 in	0.2 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	2.5 mph	2.2 mph	0.0 mph	0.0 mph
		Effective Height of Drop	2.4 in	2.0 in	0.0 in	0.0 in
14	10	Velocity at Pool Bottom	0.9 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.3 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	2.0 mph	1.8 mph	0.0 mph	0.0 mph
		Effective Height of Drop	1.6 in	1.3 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	2.9 mph	2.9 mph	0.0 mph	0.0 mph
		Effective Height of Drop	3.4 in	3.4 in	0.0 in	0.0 in

Male Participant Results

Age	Percentile		Vertical Drop	Diagonal Drop	Tucked Knee Drop	Horizontal Drop
10	10	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	1.1 mph	0.4 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.5 in	0.1 in	0.0 in	0.0 in
12	10	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	1.1 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.5 in	0.0 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	2.2 mph	2.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	2.0 in	1.6 in	0.0 in	0.0 in
14	10	Velocity at Pool Bottom	1.1 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.5 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	2.2 mph	1.8 mph	0.0 mph	0.0 mph
		Effective Height of Drop	2.0 in	1.3 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	3.1 mph	2.9 mph	0.0 mph	0.0 mph
		Effective Height of Drop	3.9 in	3.4 in	0.0 in	0.0 in

NinjaCross System
Design Participant Calculations

Drops Vertically into the Pool

Height of COM	h = 1.42	m	
Mass of Person	m = 124.72	kg =	275 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.19	m^2 =	2 ft^2
Length of Person	L = 1.83	m =	6 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	624.2	10.0	-634.2	0.29	0.9
0.200	4.7	432.1	18.2	-450.3	0.52	1.7
0.300	4.1	314.2	25.1	-339.3	0.72	2.4
0.400	3.5	236.3	31.0	-267.3	0.89	2.9
0.500	3.1	182.0	36.2	-218.1	1.04	3.4
0.600	2.7	142.4	40.8	-183.1	1.17	3.8
0.700	2.4	112.6	44.8	-157.4	1.28	4.2
0.800	2.2	89.6	48.4	-138.0	1.39	4.5
0.900	1.9	71.5	51.6	-123.1	1.48	4.9
1.000	1.7	56.9	54.5	-111.4	1.56	5.1
1.100	1.5	45.1	57.1	-102.2	1.63	5.4
1.200	1.4	35.4	59.4	-94.8	1.70	5.6
1.300	1.2	27.5	61.4	-88.9	1.76	5.8
1.400	1.0	20.9	63.2	-84.1	1.81	5.9
1.500	0.9	15.5	64.7	-80.2	1.85	6.1
1.600	0.8	11.1	66.0	-77.1	1.89	6.2
1.700	0.6	7.5	67.1	-74.6	1.92	6.3
1.800	0.5	4.7	68.0	-72.7	1.95	6.4
1.900	0.4	2.6	68.6	-71.2	1.96	6.4
1.980	0.3	1.4	69.0	-70.4	1.98	6.5
2.000	0.2	1.1	69.1	-70.3	1.98	6.5
2.100	0.1	0.3	69.4	-69.7	1.99	6.5

Drops Diagonally into the Pool

Height of COM	h = 1.15	m	
Mass of Person	m = 124.72	kg =	275 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.19	m^2 =	2 ft^2
Length of Person	L = 1.29	m =	4.24264069 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	623.6	14.1	-637.7	0.29	0.9
0.200	4.7	430.1	25.7	-455.8	0.52	1.7
0.300	4.0	310.8	35.4	-346.2	0.72	2.3
0.400	3.5	231.6	43.7	-275.3	0.88	2.9
0.500	3.0	176.1	51.0	-227.1	1.03	3.4
0.600	2.7	135.5	57.3	-192.8	1.16	3.8
0.700	2.3	104.9	62.9	-167.8	1.27	4.2
0.800	2.1	81.3	67.7	-149.0	1.37	4.5
0.900	1.8	62.7	72.1	-134.7	1.46	4.8
1.000	1.6	47.8	75.8	-123.6	1.53	5.0
1.100	1.4	35.9	79.1	-115.0	1.60	5.3
1.200	1.2	26.3	81.9	-108.2	1.66	5.4
1.300	1.0	18.6	84.3	-102.9	1.71	5.6
1.400	0.8	12.5	86.3	-98.8	1.75	5.7
1.500	0.6	7.7	87.9	-95.6	1.78	5.8
1.600	0.5	4.2	89.1	-93.3	1.80	5.9
1.700	0.3	1.8	90.0	-91.8	1.82	6.0
1.800	0.1	0.4	90.5	-90.9	1.83	6.0
1.900						
1.980						
2.000						
2.100						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.97	m	
Mass of Person	m = 124.72	kg =	275 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.28	m^2 =	3 ft^2
Length of Person	L = 0.91	m =	3 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.2	783.8	19.1	-802.9	0.27	0.9
0.200	4.0	475.9	33.5	-509.4	0.48	1.6
0.300	3.3	313.5	45.0	-358.5	0.64	2.1
0.400	2.7	216.6	54.5	-271.1	0.78	2.6
0.500	2.3	153.9	62.4	-216.3	0.89	2.9
0.600	2.0	110.7	69.1	-179.9	0.99	3.2
0.700	1.7	79.8	74.8	-154.7	1.07	3.5
0.800	1.4	57.0	79.7	-136.7	1.14	3.7
0.900	1.2	39.9	83.7	-123.6	1.20	3.9
1.000	1.0	26.9	87.1	-114.0	1.25	4.1
1.100	0.8	17.2	89.8	-107.0	1.29	4.2
1.200	0.6	9.9	91.9	-101.9	1.32	4.3
1.300	0.4	4.9	93.5	-98.4	1.34	4.4
1.400	0.2	1.7	94.5	-96.1	1.35	4.4
1.500	0.1	0.1	95.0	-95.1	1.36	4.5
1.600						
1.700						
1.800						
1.900						
1.980						
2.000						
2.100						

Drops Horizontally into the Pool

Height of COM	h = 0.81	m	
Mass of Person	m = 124.72	kg =	275 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.56	m^2 =	6 ft^2
Length of Person	L = 0.61	m =	2 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.0	985.0	25.4	-1010.4	0.24	0.8
0.200	2.8	463.1	41.3	-504.4	0.39	1.3
0.300	2.1	259.2	52.8	-312.0	0.50	1.7
0.400	1.6	157.4	61.6	-219.1	0.59	1.9
0.500	1.3	99.1	68.6	-167.7	0.65	2.1
0.600	1.0	62.6	74.1	-136.7	0.71	2.3
0.700	0.8	38.6	78.5	-117.0	0.75	2.5
0.800	0.6	22.4	81.8	-104.2	0.78	2.6
0.900	0.4	11.5	84.3	-95.8	0.80	2.6
1.000	0.3	4.6	86.0	-90.6	0.82	2.7
1.100	0.1	0.9	87.0	-87.9	0.83	2.7
1.200						
1.300						
1.400						
1.500						
1.600						
1.700						
1.800						
1.900						
1.980						
2.000						
2.100						

10-year-old Girl Calculations

Drops Vertically into the Pool

Height of COM	h = 1.16	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.30	m =	4.25 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.4	195.3	12.5	-207.8	0.25	0.8
0.200	3.1	96.7	20.8	-117.5	0.42	1.4
0.300	2.3	53.1	26.9	-79.9	0.54	1.8
0.370	1.9	35.5	30.2	-65.6	0.61	2.0
0.400	1.7	29.8	31.4	-61.2	0.64	2.1
0.500	1.3	16.1	34.7	-50.8	0.70	2.3
0.570	1.0	9.8	36.5	-46.3	0.74	2.4
0.600	0.9	7.7	37.1	-44.8	0.75	2.5
0.700	0.5	2.7	38.7	-41.4	0.78	2.6
0.730	0.4	1.8	39.0	-40.8	0.79	2.6
0.800	0.2	0.4	39.5	-39.8	0.80	2.6
0.850		0.0	39.6	-39.6	0.80	2.6

Drops Diagonally into the Pool

Height of COM	h = 0.97	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 0.92	m =	3.00520382 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.4	193.8	17.7	-211.5	0.25	0.8
0.200	3.1	93.4	29.3	-122.7	0.42	1.4
0.300	2.2	48.6	37.6	-86.2	0.54	1.8
0.370	1.8	30.5	42.0	-72.5	0.60	2.0
0.400	1.6	24.7	43.6	-68.3	0.62	2.0
0.500	1.0	11.1	47.7	-58.8	0.68	2.2
0.570	0.7	5.2	49.7	-54.9	0.71	2.3
0.600	0.6	3.5	50.3	-53.8	0.72	2.4
0.700	0.1	0.3	51.4	-51.7	0.74	2.4
0.730		0.0	51.5	-51.5	0.74	2.4
0.800						
0.850						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.83	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.20	m^2 =	2.125 ft^2
Length of Person	L = 0.65	m =	2.125 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = Vg(\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.0	202.3	20.7	-223.0	0.21	0.7
0.200	1.8	70.4	31.2	-101.6	0.32	1.0
0.300	1.1	27.8	37.8	-65.6	0.38	1.3
0.370	0.8	13.6	40.8	-54.3	0.41	1.4
0.400	0.6	9.5	41.7	-51.2	0.42	1.4
0.500	0.2	1.6	43.8	-45.4	0.44	1.5
0.570		0.0	44.2	-44.2	0.45	1.5
0.600						
0.700						
0.730						
0.800						
0.850						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.39	m^2 =	4.25 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	1.8	149.5	33.5	-182.9	0.16	0.5
0.200	0.8	32.9	45.7	-78.6	0.22	0.7
0.300	0.3	4.9	51.2	-56.1	0.24	0.8
0.370		0.0	52.3	-52.3	0.25	0.8
0.400						
0.500						
0.570						
0.600						
0.700						
0.730						
0.800						
0.850						

Drops Vertically into the Pool

Height of COM	h = 1.20	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.38	m =	4.54166667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.8	226.4	12.2	-238.6	0.26	0.9
0.200	3.5	123.2	20.8	-143.9	0.45	1.5
0.300	2.7	73.1	27.3	-100.4	0.59	1.9
0.400	2.1	44.9	32.4	-77.2	0.70	2.3
0.500	1.7	27.3	36.3	-63.6	0.79	2.6
0.600	1.3	15.9	39.4	-55.3	0.85	2.8
0.660	1.1	11.0	40.8	-51.8	0.89	2.9
0.700	0.9	8.3	41.7	-50.0	0.90	3.0
0.800	0.6	3.5	43.2	-46.8	0.94	3.1
0.850	0.4	1.9	43.8	-45.7	0.95	3.1
0.900	0.3	0.8	44.2	-45.0	0.96	3.1
0.990		0.0	44.4	-44.4	0.96	3.2

Drops Diagonally into the Pool

Height of COM	h = 1.00	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 0.98	m =	3.2114433 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = Vg(p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.8	225.2	17.2	-242.4	0.26	0.9
0.200	3.5	120.1	29.3	-149.4	0.45	1.5
0.300	2.6	68.7	38.3	-107.0	0.59	1.9
0.400	2.0	39.5	45.1	-84.7	0.69	2.3
0.500	1.5	21.6	50.3	-71.9	0.77	2.5
0.600	1.0	10.4	54.0	-64.4	0.83	2.7
0.660	0.8	5.9	55.6	-61.5	0.85	2.8
0.700	0.6	3.7	56.4	-60.1	0.86	2.8
0.800	0.2	0.5	57.6	-58.0	0.88	2.9
0.850		0.0	57.7	-57.7	0.88	2.9
0.900						
0.990						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.85	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.21	m^2 =	2.270833 ft^2
Length of Person	L = 0.69	m =	2.27083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.3	257.6	20.3	-277.8	0.22	0.7
0.200	2.1	98.2	31.3	-129.5	0.34	1.1
0.300	1.4	43.8	38.5	-82.3	0.42	1.4
0.400	0.9	19.0	43.3	-62.2	0.47	1.5
0.500	0.5	6.5	46.3	-52.8	0.50	1.6
0.600	0.2	0.9	47.8	-48.7	0.52	1.7
0.660		0.0	48.0	-48.0	0.52	1.7
0.700						
0.800						
0.850						
0.900						
0.990						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.42	m^2 =	4.541667 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.0	204.9	35.8	-240.7	0.17	0.6
0.200	1.0	52.0	50.1	-102.1	0.24	0.8
0.300	0.5	12.0	57.4	-69.4	0.27	0.9
0.400	0.1	0.4	60.0	-60.4	0.29	0.9
0.500						
0.600						
0.660						
0.700						
0.800						
0.850						
0.900						
0.990						

Drops Vertically into the Pool

Height of COM	h = 1.24	m	
Mass of Person	m = 43.54	kg =	96 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.47	m =	4.8125 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.2	266.3	12.0	-278.2	0.27	0.9
0.200	4.1	161.8	21.0	-182.9	0.48	1.6
0.300	3.3	105.0	28.3	-133.2	0.65	2.1
0.400	2.7	70.3	34.1	-104.4	0.78	2.6
0.490	2.3	49.3	38.5	-87.8	0.88	2.9
0.500	2.2	47.4	38.9	-86.3	0.89	2.9
0.600	1.8	31.6	42.9	-74.5	0.98	3.2
0.700	1.4	20.4	46.1	-66.5	1.06	3.5
0.790	1.2	13.1	48.4	-61.5	1.11	3.6
0.800	1.1	12.4	48.6	-61.0	1.12	3.7
0.900	0.8	6.7	50.5	-57.3	1.16	3.8
1.000	0.5	2.9	51.9	-54.8	1.19	3.9
1.100	0.3	0.7	52.7	-53.4	1.21	4.0
1.200		0.0	52.9	-52.9	1.22	4.0

Drops Diagonally into the Pool

Height of COM	h = 1.03	m	
Mass of Person	m = 43.54	kg =	96 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.04	m =	3.40295138 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.2	265.2	16.9	-282.1	0.27	0.9
0.200	4.0	159.1	29.7	-188.8	0.48	1.6
0.300	3.2	100.7	39.7	-140.5	0.65	2.1
0.400	2.6	64.8	47.8	-112.6	0.78	2.5
0.490	2.1	43.2	53.7	-96.8	0.87	2.9
0.500	2.1	41.2	54.2	-95.4	0.88	2.9
0.600	1.6	25.1	59.3	-84.4	0.96	3.2
0.700	1.2	14.0	63.2	-77.2	1.03	3.4
0.790	0.8	7.2	65.8	-73.0	1.07	3.5
0.800	0.8	6.6	66.0	-72.6	1.07	3.5
0.900	0.4	2.1	67.8	-69.9	1.10	3.6
1.000	0.1	0.1	68.5	-68.7	1.11	3.7
1.100						
1.200						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.87	m	
Mass of Person	m = 43.54	kg =	96 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.22	m^2 =	2.40625 ft^2
Length of Person	L = 0.73	m =	2.40625 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.7	345.5	20.4	-365.9	0.23	0.8
0.200	2.5	148.0	32.5	-180.5	0.37	1.2
0.300	1.8	74.2	40.8	-115.0	0.47	1.5
0.400	1.3	38.3	46.7	-85.0	0.54	1.8
0.490	0.9	20.1	50.6	-70.7	0.58	1.9
0.500	0.9	18.6	50.9	-69.6	0.58	1.9
0.600	0.5	7.4	53.8	-61.2	0.62	2.0
0.700	0.2	1.7	55.4	-57.0	0.64	2.1
0.790		0.0	55.8	-55.8	0.64	2.1
0.800						
0.900						
1.000						
1.100						
1.200						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 43.54	kg =	96 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.45	m^2 =	4.8125 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.4	306.2	39.4	-345.6	0.19	0.6
0.200	1.4	91.0	57.0	-148.0	0.27	0.9
0.300	0.8	29.3	67.0	-96.3	0.32	1.0
0.400	0.3	6.0	72.2	-78.2	0.34	1.1
0.490		0.0	73.6	-73.6	0.35	1.2
0.500						
0.600						
0.700						
0.790						
0.800						
0.900						
1.000						
1.100						
1.200						

12-year-old Girl Calculations

Drops Vertically into the Pool

Height of COM	h = 1.22	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.42	m =	4.64583333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.8	226.5	11.9	-238.4	0.26	0.9
0.200	3.5	123.3	20.3	-143.6	0.45	1.5
0.300	2.7	73.4	26.7	-100.1	0.59	1.9
0.400	2.2	45.2	31.7	-76.8	0.70	2.3
0.500	1.7	27.6	35.6	-63.2	0.79	2.6
0.600	1.3	16.2	38.6	-54.8	0.85	2.8
0.660	1.1	11.3	40.0	-51.3	0.89	2.9
0.700	0.9	8.6	40.8	-49.4	0.91	3.0
0.800	0.6	3.7	42.4	-46.2	0.94	3.1
0.860	0.4	1.9	43.0	-44.9	0.95	3.1
0.900	0.3	1.0	43.3	-44.3	0.96	3.2
1.000		0.0	43.7	-43.7	0.97	3.2

Drops Diagonally into the Pool

Height of COM	h = 1.01	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.00	m =	3.28510025 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.8	225.3	16.8	-242.1	0.26	0.9
0.200	3.5	120.3	28.6	-149.0	0.45	1.5
0.300	2.7	69.1	37.4	-106.5	0.59	1.9
0.400	2.0	39.9	44.2	-84.1	0.69	2.3
0.500	1.5	22.1	49.2	-71.3	0.77	2.5
0.600	1.0	10.8	52.9	-63.7	0.83	2.7
0.660	0.8	6.3	54.5	-60.7	0.85	2.8
0.700	0.6	4.0	55.3	-59.3	0.87	2.8
0.800	0.2	0.6	56.5	-57.2	0.89	2.9
0.860		0.0	56.7	-56.8	0.89	2.9
0.900						
1.000						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.86	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.22	m^2 =	2.322917 ft^2
Length of Person	L = 0.71	m =	2.32291667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.3	257.3	19.7	-277.1	0.22	0.7
0.200	2.0	97.6	30.3	-127.9	0.34	1.1
0.300	1.4	43.5	37.2	-80.8	0.41	1.4
0.400	0.9	19.0	41.9	-60.8	0.46	1.5
0.500	0.5	6.6	44.8	-51.4	0.50	1.6
0.600	0.2	1.0	46.3	-47.3	0.51	1.7
0.660		0.0	46.5	-46.5	0.52	1.7
0.700						
0.800						
0.860						
0.900						
1.000						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.43	m^2 =	4.645833 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.0	202.6	35.5	-238.1	0.17	0.6
0.200	1.0	51.0	49.5	-100.5	0.24	0.8
0.300	0.5	11.7	56.6	-68.3	0.27	0.9
0.400	0.0	0.3	59.2	-59.5	0.28	0.9
0.500						
0.600						
0.660						
0.700						
0.800						
0.860						
0.900						
1.000						

Drops Vertically into the Pool

Height of COM	h = 1.26	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.51	m =	4.95833333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.2	260.6	11.5	-272.2	0.27	0.9
0.200	4.0	156.2	20.2	-176.4	0.48	1.6
0.300	3.2	100.3	27.1	-127.4	0.64	2.1
0.400	2.6	66.6	32.7	-99.2	0.77	2.5
0.480	2.2	48.3	36.4	-84.7	0.86	2.8
0.500	2.1	44.6	37.2	-81.8	0.88	2.9
0.600	1.7	29.5	40.9	-70.4	0.97	3.2
0.700	1.4	18.9	43.9	-62.7	1.04	3.4
0.780	1.1	12.6	45.8	-58.4	1.08	3.6
0.800	1.1	11.3	46.2	-57.5	1.09	3.6
0.900	0.8	6.0	48.0	-54.0	1.14	3.7
1.000	0.5	2.5	49.2	-51.7	1.16	3.8
1.100	0.2	0.6	49.9	-50.5	1.18	3.9
1.190		0.0	50.1	-50.1	1.19	3.9

Drops Diagonally into the Pool

Height of COM	h = 1.04	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.07	m =	3.50607112 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	259.6	16.3	-275.9	0.27	0.9
0.200	4.0	153.5	28.5	-182.0	0.48	1.6
0.300	3.1	96.1	38.1	-134.3	0.64	2.1
0.400	2.5	61.3	45.7	-107.1	0.76	2.5
0.480	2.1	42.5	50.7	-93.2	0.85	2.8
0.500	2.0	38.6	51.8	-90.4	0.87	2.8
0.600	1.5	23.3	56.6	-79.8	0.95	3.1
0.700	1.1	12.8	60.2	-72.9	1.01	3.3
0.780	0.8	7.0	62.3	-69.3	1.04	3.4
0.800	0.8	5.8	62.7	-68.6	1.05	3.4
0.900	0.4	1.7	64.3	-66.1	1.08	3.5
1.000	0.0	0.1	65.0	-65.0	1.09	3.6
1.100						
1.190						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.89	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.23	m^2 =	2.479167 ft^2
Length of Person	L = 0.76	m =	2.47916667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.6	332.4	19.4	-351.8	0.23	0.8
0.200	2.4	138.5	30.7	-169.2	0.36	1.2
0.300	1.7	68.2	38.3	-106.5	0.45	1.5
0.400	1.2	34.7	43.7	-78.4	0.52	1.7
0.480	0.9	19.3	46.9	-66.2	0.55	1.8
0.500	0.8	16.5	47.6	-64.0	0.56	1.8
0.600	0.5	6.3	50.1	-56.4	0.59	1.9
0.700	0.2	1.2	51.4	-52.7	0.61	2.0
0.780		0.0	51.8	-51.8	0.61	2.0
0.800						
0.900						
1.000						
1.100						
1.190						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.46	m^2 =	4.958333 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.3	285.1	38.3	-323.4	0.18	0.6
0.200	1.3	81.9	54.9	-136.8	0.26	0.9
0.300	0.7	25.1	64.1	-89.2	0.31	1.0
0.400	0.3	4.3	68.7	-73.1	0.33	1.1
0.480		0.0	69.7	-69.7	0.33	1.1
0.500						
0.600						
0.700						
0.780						
0.800						
0.900						
1.000						
1.100						
1.190						

Drops Vertically into the Pool

Height of COM	h = 1.31	m	
Mass of Person	m = 55.33	kg =	122 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.61	m =	5.27083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.5	297.0	11.2	-308.2	0.28	0.9
0.200	4.5	196.1	20.2	-216.3	0.51	1.7
0.300	3.8	135.9	27.6	-163.5	0.69	2.3
0.400	3.2	96.8	33.8	-130.6	0.85	2.8
0.500	2.7	69.9	39.0	-108.9	0.98	3.2
0.550	2.5	59.5	41.4	-100.8	1.04	3.4
0.600	2.3	50.5	43.5	-94.0	1.09	3.6
0.700	1.9	36.1	47.3	-83.4	1.19	3.9
0.800	1.6	25.3	50.5	-75.8	1.27	4.2
0.900	1.3	17.0	53.1	-70.2	1.34	4.4
0.930	1.2	15.0	53.8	-68.8	1.35	4.4
1.000	1.1	10.8	55.3	-66.1	1.39	4.6
1.100	0.8	6.2	56.9	-63.1	1.43	4.7
1.200	0.5	3.0	58.1	-61.1	1.46	4.8
1.230	0.5	2.3	58.4	-60.6	1.47	4.8
1.300	0.3	1.0	58.9	-59.9	1.48	4.9
1.400	0.1	0.1	59.2	-59.3	1.49	4.9
1.430		0.0	59.2	-59.2	1.49	4.9

Drops Diagonally into the Pool

Height of COM	h = 1.08	m	
Mass of Person	m = 55.33	kg =	122 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.14	m =	3.72704199 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.5	296.1	15.9	-312.0	0.28	0.9
0.200	4.5	193.7	28.5	-222.2	0.51	1.7
0.300	3.7	132.1	38.8	-170.9	0.69	2.3
0.400	3.1	91.7	47.4	-139.2	0.84	2.8
0.500	2.6	63.8	54.6	-118.4	0.97	3.2
0.550	2.3	53.0	57.7	-110.7	1.03	3.4
0.600	2.1	43.7	60.5	-104.3	1.08	3.5
0.700	1.7	29.0	65.4	-94.4	1.16	3.8
0.800	1.4	18.2	69.4	-87.5	1.23	4.0
0.900	1.0	10.3	72.4	-82.7	1.29	4.2
0.930	0.9	8.5	73.2	-81.6	1.30	4.3
1.000	0.7	4.9	74.6	-79.5	1.33	4.4
1.100	0.4	1.6	76.0	-77.5	1.35	4.4
1.200	0.1	0.1	76.6	-76.7	1.36	4.5
1.230		0.0	76.6	-76.6	1.36	4.5
1.300						
1.400						
1.430						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.91	m	
Mass of Person	m = 55.33	kg =	122 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.24	m^2 =	2.635417 ft^2
Length of Person	L = 0.80	m =	2.63541667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.0	433.7	19.3	-453.0	0.24	0.8
0.200	2.8	200.5	31.3	-231.8	0.39	1.3
0.300	2.0	108.0	39.9	-147.9	0.50	1.6
0.400	1.5	61.4	46.3	-107.8	0.58	1.9
0.500	1.2	34.8	51.2	-86.0	0.64	2.1
0.550	1.0	25.7	53.1	-78.8	0.67	2.2
0.600	0.8	18.5	54.8	-73.3	0.69	2.3
0.700	0.6	8.5	57.3	-65.8	0.72	2.4
0.800	0.3	2.7	58.9	-61.6	0.74	2.4
0.900	0.1	0.2	59.6	-59.8	0.75	2.5
0.930		0.0	59.6	-59.6	0.75	2.5
1.000						
1.100						
1.200						
1.230						
1.300						
1.400						
1.430						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 55.33	kg =	122 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.49	m^2 =	5.270833 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.7	411.5	41.6	-453.1	0.20	0.7
0.200	1.6	134.8	61.6	-196.4	0.29	1.0
0.300	1.0	51.0	73.6	-124.6	0.35	1.2
0.400	0.5	16.2	80.8	-97.0	0.39	1.3
0.500	0.2	2.1	84.3	-86.4	0.40	1.3
0.550		0.1	84.8	-84.8	0.40	1.3
0.600						
0.700						
0.800						
0.900						
0.930						
1.000						
1.100						
1.200						
1.230						
1.300						
1.400						
1.430						

14-year-old Girl Calculations

Drops Vertically into the Pool

Height of COM	h = 1.27	m	
Mass of Person	m = 39.46	kg =	87 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.52	m =	4.97916667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	253.1	11.4	-264.5	0.27	0.9
0.200	3.9	148.6	19.9	-168.5	0.47	1.6
0.300	3.1	94.0	26.5	-120.5	0.63	2.1
0.400	2.5	61.5	31.9	-93.4	0.76	2.5
0.460	2.2	48.0	34.6	-82.5	0.82	2.7
0.500	2.0	40.6	36.2	-76.8	0.86	2.8
0.600	1.6	26.4	39.7	-66.1	0.94	3.1
0.700	1.3	16.4	42.5	-58.9	1.01	3.3
0.750	1.1	12.6	43.7	-56.3	1.04	3.4
0.800	1.0	9.5	44.7	-54.1	1.06	3.5
0.900	0.7	4.7	46.2	-50.9	1.10	3.6
0.990	0.4	1.9	47.2	-49.1	1.12	3.7
1.000	0.4	1.7	47.3	-49.0	1.12	3.7
1.100	0.1	0.2	47.8	-48.0	1.14	3.7
1.150		0.0	47.9	-47.9	1.14	3.7

Drops Diagonally into the Pool

Height of COM	h = 1.04	m	
Mass of Person	m = 39.46	kg =	87 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.07	m =	3.52080251 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	252.0	16.1	-268.2	0.27	0.9
0.200	3.9	145.9	28.1	-173.9	0.47	1.5
0.300	3.0	89.9	37.3	-127.1	0.63	2.1
0.400	2.4	56.3	44.6	-100.9	0.75	2.5
0.460	2.1	42.3	48.2	-90.6	0.81	2.7
0.500	1.9	34.8	50.4	-85.1	0.85	2.8
0.600	1.4	20.3	54.8	-75.2	0.92	3.0
0.700	1.0	10.7	58.2	-68.8	0.98	3.2
0.750	0.8	7.2	59.4	-66.6	1.00	3.3
0.800	0.7	4.4	60.5	-64.9	1.02	3.3
0.900	0.3	1.0	61.8	-62.8	1.04	3.4
0.990		0.0	62.1	-62.1	1.04	3.4
1.000						
1.100						
1.150						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.89	m	
Mass of Person	m = 39.46	kg =	87 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.23	m^2 =	2.489583 ft^2
Length of Person	L = 0.76	m =	2.48958333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.5	314.3	19.0	-333.4	0.23	0.7
0.200	2.3	127.6	29.8	-157.4	0.35	1.2
0.300	1.6	61.5	37.1	-98.6	0.44	1.4
0.400	1.1	30.5	42.2	-72.6	0.50	1.6
0.460	0.9	19.3	44.4	-63.8	0.53	1.7
0.500	0.7	13.8	45.7	-59.5	0.54	1.8
0.600	0.4	4.8	47.9	-52.7	0.57	1.9
0.700	0.1	0.6	49.0	-49.7	0.58	1.9
0.750		0.0	49.2	-49.2	0.58	1.9
0.800						
0.900						
0.990						
1.000						
1.100						
1.150						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 39.46	kg =	87 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.46	m^2 =	4.979167 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.2	262.2	37.4	-299.6	0.18	0.6
0.200	1.2	72.8	53.1	-125.9	0.25	0.8
0.300	0.6	21.0	61.7	-82.7	0.29	1.0
0.400	0.2	2.9	65.7	-68.6	0.31	1.0
0.460		0.0	66.4	-66.4	0.32	1.0
0.500						
0.600						
0.700						
0.750						
0.800						
0.900						
0.990						
1.000						
1.100						
1.150						

Drops Vertically into the Pool

Height of COM	h = 1.31	m	
Mass of Person	m = 48.98	kg =	108 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.61	m =	5.27083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.4	281.8	11.1	-292.9	0.28	0.9
0.200	4.3	178.8	19.7	-198.6	0.50	1.6
0.300	3.5	120.2	26.7	-147.0	0.67	2.2
0.400	2.9	83.4	32.5	-115.9	0.82	2.7
0.500	2.5	58.6	37.3	-95.9	0.94	3.1
0.600	2.1	41.1	41.4	-82.5	1.04	3.4
0.700	1.7	28.3	44.8	-73.1	1.13	3.7
0.800	1.4	18.9	47.6	-66.5	1.20	3.9
0.870	1.2	13.8	49.2	-63.0	1.24	4.1
0.900	1.1	11.9	49.8	-61.8	1.25	4.1
1.000	0.8	6.9	51.6	-58.4	1.30	4.3
1.100	0.6	3.3	52.8	-56.2	1.33	4.4
1.150	0.4	2.1	53.3	-55.4	1.34	4.4
1.200	0.3	1.1	53.7	-54.8	1.35	4.4
1.300	0.1	0.1	54.0	-54.1	1.36	4.5
1.330		0.0	54.0	-54.0	1.36	4.5

Drops Diagonally into the Pool

Height of COM	h = 1.08	m	
Mass of Person	m = 48.98	kg =	108 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.14	m =	3.72704199 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.4	280.9	15.7	-296.6	0.28	0.9
0.200	4.3	176.4	27.8	-204.2	0.49	1.6
0.300	3.5	116.3	37.6	-154.0	0.67	2.2
0.400	2.8	78.3	45.6	-123.9	0.81	2.7
0.500	2.3	52.6	52.2	-104.8	0.93	3.0
0.600	1.9	34.6	57.5	-92.1	1.02	3.4
0.700	1.5	21.7	61.8	-83.5	1.10	3.6
0.800	1.1	12.5	65.1	-77.6	1.16	3.8
0.870	0.9	7.8	66.9	-74.7	1.19	3.9
0.900	0.8	6.2	67.6	-73.7	1.20	3.9
1.000	0.5	2.2	69.1	-71.3	1.23	4.0
1.100	0.1	0.2	69.9	-70.2	1.24	4.1
1.150		0.0	70.0	-70.0	1.24	4.1
1.200						
1.300						
1.330						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.91	m	
Mass of Person	m = 48.98	kg =	108 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.24	m^2 =	2.635417 ft^2
Length of Person	L = 0.80	m =	2.63541667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.8	389.3	18.7	-408.0	0.24	0.8
0.200	2.6	170.8	30.0	-200.8	0.38	1.2
0.300	1.8	88.5	37.8	-126.3	0.48	1.6
0.400	1.4	48.3	43.6	-91.9	0.55	1.8
0.500	1.0	25.8	47.8	-73.6	0.60	2.0
0.600	0.7	12.5	50.8	-63.3	0.64	2.1
0.700	0.4	4.7	52.8	-57.5	0.66	2.2
0.800	0.2	0.8	53.9	-54.7	0.68	2.2
0.870		0.0	54.1	-54.1	0.68	2.2
0.900						
1.000						
1.100						
1.150						
1.200						
1.300						
1.330						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 48.98	kg =	108 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.49	m^2 =	5.270833 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.5	349.7	39.8	-389.6	0.19	0.6
0.200	1.4	107.6	57.9	-165.5	0.28	0.9
0.300	0.8	37.3	68.5	-105.8	0.33	1.1
0.400	0.4	9.6	74.4	-84.0	0.35	1.2
0.500	0.0	0.3	76.6	-76.9	0.37	1.2
0.600						
0.700						
0.800						
0.870						
0.900						
1.000						
1.100						
1.150						
1.200						
1.300						
1.330						

Drops Vertically into the Pool

Height of COM	h = 1.35	m	
Mass of Person	m = 65.31	kg =	144 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.69	m =	5.54166667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	316.4	10.9	-327.3	0.29	0.9
0.200	4.8	219.8	19.8	-239.5	0.52	1.7
0.300	4.1	158.7	27.3	-186.0	0.72	2.4
0.400	3.5	117.3	33.7	-151.1	0.89	2.9
0.500	3.0	87.9	39.3	-127.2	1.04	3.4
0.600	2.6	66.1	44.1	-110.2	1.17	3.8
0.700	2.3	49.6	48.3	-97.9	1.28	4.2
0.800	2.0	36.8	51.9	-88.7	1.37	4.5
0.900	1.7	26.8	55.0	-81.8	1.45	4.8
1.000	1.4	18.9	57.6	-76.5	1.52	5.0
1.100	1.1	12.7	59.8	-72.5	1.58	5.2
1.200	0.9	8.0	61.5	-69.5	1.63	5.3
1.300	0.7	4.5	62.9	-67.3	1.66	5.5
1.380	0.5	2.4	63.7	-66.1	1.68	5.5
1.400	0.4	2.0	63.8	-65.8	1.69	5.5
1.500	0.2	0.5	64.4	-65.0	1.70	5.6
1.600		0.0	64.6	-64.6	1.71	5.6

Drops Diagonally into the Pool

Height of COM	h = 1.11	m	
Mass of Person	m = 65.31	kg =	144 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.19	m =	3.91855008 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	315.7	15.3	-331.1	0.29	0.9
0.200	4.8	217.7	27.9	-245.6	0.52	1.7
0.300	4.0	155.1	38.5	-193.6	0.72	2.4
0.400	3.4	112.5	47.4	-159.9	0.89	2.9
0.500	2.9	81.9	55.1	-137.0	1.03	3.4
0.600	2.5	59.3	61.6	-120.9	1.15	3.8
0.700	2.1	42.2	67.1	-109.3	1.25	4.1
0.800	1.7	29.1	71.7	-100.9	1.34	4.4
0.900	1.4	19.2	75.5	-94.7	1.41	4.6
1.000	1.1	11.6	78.5	-90.2	1.47	4.8
1.100	0.8	6.2	80.8	-87.0	1.51	5.0
1.200	0.5	2.5	82.4	-84.9	1.54	5.1
1.300	0.2	0.5	83.2	-83.7	1.56	5.1
1.380		0.0	83.4	-83.4	1.56	5.1
1.400						
1.500						
1.600						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.93	m	
Mass of Person	m = 65.31	kg =	144 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.26	m^2 =	2.770833 ft^2
Length of Person	L = 0.84	m =	2.77083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.2	502.0	18.8	-520.8	0.25	0.8
0.200	3.0	244.5	31.0	-275.5	0.41	1.3
0.300	2.3	137.5	39.9	-177.4	0.53	1.7
0.400	1.7	82.2	46.7	-128.9	0.62	2.0
0.500	1.4	49.8	52.0	-101.8	0.69	2.3
0.600	1.0	29.5	56.1	-85.6	0.74	2.4
0.700	0.8	16.2	59.2	-75.4	0.78	2.6
0.800	0.5	7.7	61.4	-69.1	0.81	2.7
0.900	0.3	2.6	62.8	-65.4	0.83	2.7
1.000	0.1	0.2	63.5	-63.7	0.84	2.8
1.100						
1.200						
1.300						
1.380						
1.400						
1.500						
1.600						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 65.31	kg =	144 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.51	m^2 =	5.541667 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.9	501.1	43.3	-544.4	0.21	0.7
0.200	1.8	175.2	65.2	-240.4	0.31	1.0
0.300	1.1	72.2	78.8	-151.0	0.38	1.2
0.400	0.7	27.5	87.5	-115.0	0.42	1.4
0.500	0.3	6.9	92.4	-99.3	0.44	1.4
0.600	0.0	0.1	94.1	-94.2	0.45	1.5
0.700						
0.800						
0.900						
1.000						
1.100						
1.200						
1.300						
1.380						
1.400						
1.500						
1.600						

10-year-old Boy Calculations

Drops Vertically into the Pool

Height of COM	h = 1.16	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.30	m =	4.27083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.4	195.3	12.4	-207.7	0.25	0.8
0.200	3.1	96.7	20.7	-117.4	0.42	1.4
0.300	2.3	53.1	26.7	-79.9	0.54	1.8
0.370	1.9	35.5	30.0	-65.6	0.61	2.0
0.400	1.7	29.9	31.2	-61.1	0.64	2.1
0.500	1.3	16.1	34.6	-50.7	0.70	2.3
0.570	1.0	9.8	36.3	-46.2	0.74	2.4
0.600	0.9	7.7	37.0	-44.7	0.75	2.5
0.700	0.5	2.8	38.5	-41.3	0.79	2.6
0.730	0.4	1.8	38.8	-40.6	0.79	2.6
0.800	0.2	0.4	39.3	-39.7	0.80	2.6
0.860		0.0	39.4	-39.4	0.80	2.6

Drops Diagonally into the Pool

Height of COM	h = 0.97	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 0.92	m =	3.01993521 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = Vg(p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.4	193.8	17.6	-211.4	0.25	0.8
0.200	3.1	93.4	29.2	-122.6	0.42	1.4
0.300	2.2	48.7	37.4	-86.1	0.54	1.8
0.370	1.8	30.6	41.8	-72.4	0.60	2.0
0.400	1.6	24.8	43.4	-68.2	0.62	2.1
0.500	1.0	11.1	47.5	-58.6	0.68	2.2
0.570	0.7	5.3	49.5	-54.8	0.71	2.3
0.600	0.6	3.5	50.1	-53.6	0.72	2.4
0.700	0.1	0.3	51.2	-51.5	0.74	2.4
0.730		0.0	51.3	-51.3	0.74	2.4
0.800						
0.860						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.83	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.20	m^2 =	2.135417 ft^2
Length of Person	L = 0.65	m =	2.13541667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.0	202.2	20.6	-222.7	0.21	0.7
0.200	1.8	70.2	31.0	-101.3	0.32	1.0
0.300	1.1	27.8	37.5	-65.3	0.38	1.3
0.370	0.8	13.6	40.5	-54.0	0.41	1.4
0.400	0.6	9.5	41.5	-50.9	0.42	1.4
0.500	0.2	1.6	43.5	-45.1	0.44	1.5
0.570		0.0	43.9	-43.9	0.45	1.5
0.600						
0.700						
0.730						
0.800						
0.860						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.40	m^2 =	4.270833 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	1.8	149.1	33.4	-182.4	0.16	0.5
0.200	0.8	32.8	45.5	-78.3	0.22	0.7
0.300	0.3	4.9	51.0	-55.9	0.24	0.8
0.370		0.0	52.1	-52.1	0.25	0.8
0.400						
0.500						
0.570						
0.600						
0.700						
0.730						
0.800						
0.860						

Drops Vertically into the Pool

Height of COM	h = 1.20	m	
Mass of Person	m = 31.75	kg =	70 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.38	m =	4.54166667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.7	222.4	12.1	-234.5	0.26	0.9
0.200	3.5	119.6	20.6	-140.2	0.45	1.5
0.300	2.7	70.4	27.0	-97.4	0.59	1.9
0.400	2.1	42.8	32.0	-74.8	0.69	2.3
0.500	1.6	25.8	35.8	-61.6	0.78	2.5
0.600	1.2	14.7	38.8	-53.5	0.84	2.8
0.650	1.0	10.7	40.0	-50.7	0.87	2.8
0.700	0.9	7.5	41.0	-48.5	0.89	2.9
0.800	0.5	3.0	42.5	-45.5	0.92	3.0
0.840	0.4	1.8	42.9	-44.7	0.93	3.0
0.900	0.2	0.6	43.3	-43.9	0.94	3.1
0.980		0.0	43.5	-43.5	0.94	3.1

Drops Diagonally into the Pool

Height of COM	h = 1.00	m	
Mass of Person	m = 31.75	kg =	70 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 0.98	m =	3.2114433 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = Vg(p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.7	221.2	17.1	-238.3	0.26	0.9
0.200	3.4	116.6	29.0	-145.6	0.44	1.5
0.300	2.6	66.0	37.9	-103.9	0.58	1.9
0.400	2.0	37.5	44.6	-82.1	0.68	2.2
0.500	1.4	20.2	49.6	-69.8	0.76	2.5
0.600	1.0	9.4	53.1	-62.6	0.81	2.7
0.650	0.7	5.8	54.4	-60.2	0.83	2.7
0.700	0.5	3.1	55.4	-58.5	0.85	2.8
0.800	0.1	0.3	56.4	-56.7	0.86	2.8
0.840		0.0	56.5	-56.5	0.87	2.8
0.900						
0.980						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.85	m	
Mass of Person	m = 31.75	kg =	70 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.21	m^2 =	2.270833 ft^2
Length of Person	L = 0.69	m =	2.27083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = Vg(\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.3	249.7	20.1	-269.8	0.22	0.7
0.200	2.0	94.0	30.9	-124.9	0.34	1.1
0.300	1.3	41.4	37.9	-79.3	0.41	1.3
0.400	0.9	17.5	42.6	-60.1	0.46	1.5
0.500	0.5	5.7	45.4	-51.1	0.49	1.6
0.600	0.1	0.6	46.8	-47.4	0.51	1.7
0.650		0.0	46.9	-46.9	0.51	1.7
0.700						
0.800						
0.840						
0.900						
0.980						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 31.75	kg =	70 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.42	m^2 =	4.541667 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.0	196.0	35.4	-231.4	0.17	0.6
0.200	1.0	48.8	49.3	-98.1	0.24	0.8
0.300	0.5	10.7	56.2	-67.0	0.27	0.9
0.400	0.0	0.2	58.7	-58.9	0.28	0.9
0.500						
0.600						
0.650						
0.700						
0.800						
0.840						
0.900						
0.980						

Drops Vertically into the Pool

Height of COM	h = 1.24	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.47	m =	4.83333333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.2	260.6	11.8	-272.4	0.27	0.9
0.200	4.0	156.0	20.8	-176.8	0.48	1.6
0.300	3.2	100.0	27.8	-127.8	0.64	2.1
0.400	2.6	66.3	33.5	-99.7	0.77	2.5
0.480	2.2	48.0	37.3	-85.2	0.86	2.8
0.500	2.1	44.2	38.1	-82.3	0.88	2.9
0.600	1.7	29.1	41.9	-71.0	0.97	3.2
0.700	1.4	18.4	44.9	-63.4	1.04	3.4
0.770	1.1	12.9	46.7	-59.6	1.08	3.5
0.800	1.1	10.9	47.3	-58.2	1.09	3.6
0.900	0.8	5.6	49.1	-54.7	1.13	3.7
1.000	0.5	2.2	50.3	-52.5	1.16	3.8
1.010	0.4	2.0	50.4	-52.4	1.16	3.8
1.100	0.2	0.4	51.0	-51.4	1.17	3.9
1.170		0.0	51.1	-51.1	1.18	3.9

Drops Diagonally into the Pool

Height of COM	h = 1.03	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.04	m =	3.41768278 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	259.5	16.7	-276.3	0.27	0.9
0.200	4.0	153.2	29.3	-182.5	0.48	1.6
0.300	3.1	95.8	39.1	-134.9	0.64	2.1
0.400	2.5	60.9	46.9	-107.7	0.76	2.5
0.480	2.1	42.0	51.9	-93.9	0.85	2.8
0.500	2.0	38.1	53.1	-91.2	0.87	2.8
0.600	1.5	22.7	57.9	-80.6	0.94	3.1
0.700	1.1	12.3	61.6	-73.9	1.00	3.3
0.770	0.8	7.2	63.5	-70.6	1.03	3.4
0.800	0.7	5.4	64.1	-69.6	1.05	3.4
0.900	0.4	1.5	65.7	-67.2	1.07	3.5
1.000	0.0	0.0	66.2	-66.2	1.08	3.5
1.010		0.0	66.2	-66.2	1.08	3.5
1.100						
1.170						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.88	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.22	m^2 =	2.416667 ft^2
Length of Person	L = 0.74	m =	2.41666667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.7	331.9	20.0	-351.9	0.23	0.8
0.200	2.4	139.3	31.8	-171.1	0.37	1.2
0.300	1.7	68.7	39.7	-108.4	0.46	1.5
0.400	1.2	34.8	45.4	-80.2	0.52	1.7
0.480	0.9	19.3	48.7	-68.0	0.56	1.8
0.500	0.8	16.4	49.4	-65.7	0.57	1.9
0.600	0.5	6.1	52.0	-58.1	0.60	2.0
0.700	0.2	1.1	53.3	-54.4	0.61	2.0
0.770		0.0	53.6	-53.6	0.62	2.0
0.800						
0.900						
1.000						
1.010						
1.100						
1.170						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.45	m^2 =	4.833333 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.4	287.9	38.7	-326.6	0.18	0.6
0.200	1.3	83.4	55.6	-139.0	0.27	0.9
0.300	0.7	25.8	65.0	-90.8	0.31	1.0
0.400	0.3	4.5	69.8	-74.4	0.33	1.1
0.480		0.0	70.9	-70.9	0.34	1.1
0.500						
0.600						
0.700						
0.770						
0.800						
0.900						
1.000						
1.010						
1.100						
1.170						

12-year-old Boy Calculations

Drops Vertically into the Pool

Height of COM	h = 1.21	m	
Mass of Person	m = 32.20	kg =	71 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.40	m =	4.58333333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.8	224.5	12.0	-236.5	0.26	0.9
0.200	3.5	121.5	20.5	-142.0	0.45	1.5
0.300	2.7	71.9	26.9	-98.8	0.59	1.9
0.400	2.1	43.9	31.9	-75.8	0.70	2.3
0.410	2.1	41.8	32.3	-74.2	0.71	2.3
0.500	1.7	26.7	35.8	-62.4	0.78	2.6
0.600	1.3	15.4	38.8	-54.2	0.85	2.8
0.650	1.1	11.3	40.0	-51.3	0.87	2.9
0.700	0.9	8.0	41.0	-49.0	0.90	2.9
0.800	0.6	3.3	42.5	-45.9	0.93	3.1
0.850	0.4	1.8	43.0	-44.9	0.94	3.1
0.900	0.3	0.8	43.4	-44.2	0.95	3.1
0.990		0.0	43.6	-43.7	0.95	3.1
1.000						

Drops Diagonally into the Pool

Height of COM	h = 1.00	m	
Mass of Person	m = 32.20	kg =	71 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 0.99	m =	3.24090608 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.7	223.2	17.0	-240.2	0.26	0.9
0.200	3.5	118.4	28.9	-147.3	0.45	1.5
0.300	2.6	67.5	37.8	-105.3	0.58	1.9
0.400	2.0	38.7	44.5	-83.2	0.69	2.3
0.410	1.9	36.5	45.0	-81.6	0.70	2.3
0.500	1.5	21.1	49.5	-70.6	0.77	2.5
0.600	1.0	10.1	53.1	-63.2	0.82	2.7
0.650	0.8	6.3	54.4	-60.8	0.84	2.8
0.700	0.6	3.5	55.4	-59.0	0.86	2.8
0.800	0.2	0.4	56.6	-57.0	0.87	2.9
0.850		0.0	56.7	-56.7	0.88	2.9
0.900						
0.990						
1.000						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.86	m	
Mass of Person	m = 32.20	kg =	71 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.21	m^2 =	2.291667 ft^2
Length of Person	L = 0.70	m =	2.29166667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.3	253.6	20.0	-273.5	0.22	0.7
0.200	2.0	95.8	30.7	-126.6	0.34	1.1
0.300	1.4	42.5	37.7	-80.2	0.41	1.4
0.400	0.9	18.2	42.3	-60.6	0.46	1.5
0.410	0.8	16.6	42.7	-59.3	0.47	1.5
0.500	0.5	6.2	45.2	-51.4	0.49	1.6
0.600	0.2	0.8	46.7	-47.5	0.51	1.7
0.650		0.0	46.9	-46.9	0.51	1.7
0.700						
0.800						
0.850						
0.900						
0.990						
1.000						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 32.20	kg =	71 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.43	m^2 =	4.583333 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.0	199.5	35.5	-235.0	0.17	0.6
0.200	1.0	50.0	49.5	-99.4	0.24	0.8
0.300	0.5	11.2	56.5	-67.7	0.27	0.9
0.400	0.0	0.3	59.0	-59.3	0.28	0.9
0.410		0.1	59.0	-59.1	0.28	0.9
0.500						
0.600						
0.650						
0.700						
0.800						
0.850						
0.900						
0.990						
1.000						

Drops Vertically into the Pool

Height of COM	h = 1.25	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.49	m =	4.89583333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	256.1	11.6	-267.8	0.27	0.9
0.200	3.9	151.6	20.3	-171.9	0.47	1.6
0.300	3.2	96.4	27.2	-123.5	0.63	2.1
0.400	2.6	63.4	32.7	-96.0	0.76	2.5
0.470	2.2	47.5	35.9	-83.4	0.84	2.8
0.500	2.1	42.0	37.1	-79.1	0.87	2.8
0.600	1.7	27.4	40.8	-68.1	0.95	3.1
0.700	1.3	17.1	43.7	-60.8	1.02	3.3
0.760	1.1	12.5	45.1	-57.6	1.05	3.5
0.800	1.0	9.9	45.9	-55.9	1.07	3.5
0.900	0.7	5.0	47.6	-52.6	1.11	3.6
0.990	0.4	2.1	48.6	-50.7	1.14	3.7
1.000	0.4	1.9	48.7	-50.6	1.14	3.7
1.100	0.1	0.3	49.3	-49.5	1.15	3.8
1.160		0.0	49.3	-49.3	1.15	3.8

Drops Diagonally into the Pool

Height of COM	h = 1.04	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.06	m =	3.46187695 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	255.1	16.5	-271.5	0.27	0.9
0.200	3.9	148.8	28.7	-177.5	0.47	1.6
0.300	3.1	92.2	38.2	-130.4	0.63	2.1
0.400	2.4	58.1	45.7	-103.8	0.76	2.5
0.470	2.1	41.7	50.0	-91.8	0.83	2.7
0.500	1.9	36.0	51.7	-87.7	0.85	2.8
0.600	1.5	21.2	56.3	-77.5	0.93	3.1
0.700	1.1	11.2	59.8	-71.0	0.99	3.2
0.760	0.8	7.0	61.3	-68.3	1.01	3.3
0.800	0.7	4.8	62.2	-66.9	1.03	3.4
0.900	0.3	1.2	63.6	-64.7	1.05	3.4
0.990		0.0	64.0	-64.0	1.06	3.5
1.000						
1.100						
1.160						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.88	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.23	m^2 =	2.447917 ft^2
Length of Person	L = 0.75	m =	2.44791667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.6	321.4	19.6	-341.0	0.23	0.7
0.200	2.3	132.5	30.8	-163.3	0.36	1.2
0.300	1.6	64.5	38.4	-102.9	0.45	1.5
0.400	1.1	32.2	43.8	-76.0	0.51	1.7
0.470	0.9	19.0	46.5	-65.5	0.54	1.8
0.500	0.8	14.8	47.5	-62.3	0.55	1.8
0.600	0.5	5.3	49.9	-55.2	0.58	1.9
0.700	0.2	0.8	51.1	-51.9	0.60	2.0
0.760		0.0	51.3	-51.3	0.60	2.0
0.800						
0.900						
0.990						
1.000						
1.100						
1.160						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.45	m^2 =	4.895833 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.3	273.1	38.0	-311.0	0.18	0.6
0.200	1.2	77.3	54.3	-131.5	0.26	0.8
0.300	0.7	23.0	63.2	-86.2	0.30	1.0
0.400	0.2	3.5	67.6	-71.1	0.32	1.1
0.470		0.0	68.4	-68.4	0.33	1.1
0.500						
0.600						
0.700						
0.760						
0.800						
0.900						
0.990						
1.000						
1.100						
1.160						

Drops Vertically into the Pool

Height of COM	h = 1.30	m	
Mass of Person	m = 53.51	kg =	118 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.59	m =	5.22916667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.5	292.9	11.3	-304.2	0.28	0.9
0.200	4.5	191.3	20.2	-211.5	0.50	1.7
0.300	3.7	131.5	27.6	-159.0	0.69	2.3
0.400	3.1	93.0	33.7	-126.7	0.84	2.8
0.500	2.6	66.6	38.9	-105.4	0.97	3.2
0.540	2.5	58.3	40.7	-99.0	1.02	3.3
0.600	2.2	47.7	43.3	-90.9	1.08	3.5
0.700	1.9	33.7	47.0	-80.7	1.17	3.8
0.800	1.6	23.3	50.0	-73.3	1.25	4.1
0.900	1.3	15.4	52.6	-68.0	1.31	4.3
0.910	1.2	14.7	52.8	-67.5	1.32	4.3
1.000	1.0	9.5	54.6	-64.1	1.36	4.5
1.100	0.7	5.2	56.2	-61.4	1.40	4.6
1.200	0.5	2.3	57.3	-59.6	1.43	4.7
1.300	0.2	0.6	57.9	-58.5	1.44	4.7
1.400		0.0	58.1	-58.1	1.45	4.8

Drops Diagonally into the Pool

Height of COM	h = 1.07	m	
Mass of Person	m = 53.51	kg =	118 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.13	m =	3.69757921 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.5	292.0	15.9	-308.0	0.28	0.9
0.200	4.4	188.9	28.5	-217.5	0.50	1.7
0.300	3.6	127.6	38.8	-166.4	0.68	2.2
0.400	3.0	87.8	47.3	-135.1	0.83	2.7
0.500	2.5	60.5	54.3	-114.8	0.96	3.1
0.540	2.3	51.9	56.8	-108.7	1.00	3.3
0.600	2.1	40.9	60.2	-101.1	1.06	3.5
0.700	1.7	26.7	64.9	-91.6	1.15	3.8
0.800	1.3	16.3	68.7	-85.0	1.21	4.0
0.900	0.9	8.9	71.6	-80.5	1.26	4.1
0.910	0.9	8.3	71.8	-80.1	1.27	4.2
1.000	0.6	3.9	73.6	-77.5	1.30	4.3
1.100	0.3	1.0	74.8	-75.8	1.32	4.3
1.200		0.0	75.2	-75.2	1.33	4.4
1.300						
1.400						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.91	m	
Mass of Person	m = 53.51	kg =	118 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.24	m^2 =	2.614583 ft^2
Length of Person	L = 0.80	m =	2.61458333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.0	420.9	19.3	-440.2	0.24	0.8
0.200	2.7	192.3	31.3	-223.6	0.39	1.3
0.300	2.0	102.6	39.8	-142.4	0.50	1.6
0.400	1.5	57.7	46.1	-103.8	0.58	1.9
0.500	1.1	32.2	50.8	-83.0	0.63	2.1
0.540	1.0	25.1	52.4	-77.4	0.65	2.1
0.600	0.8	16.7	54.3	-71.0	0.68	2.2
0.700	0.5	7.3	56.7	-64.0	0.71	2.3
0.800	0.3	2.0	58.2	-60.2	0.73	2.4
0.900	0.0	0.0	58.7	-58.7	0.73	2.4
0.910		0.0	58.7	-58.7	0.73	2.4
1.000						
1.100						
1.200						
1.300						
1.400						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 53.51	kg =	118 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.49	m^2 =	5.229167 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.7	394.8	41.3	-436.1	0.20	0.6
0.200	1.5	127.5	60.8	-188.4	0.29	1.0
0.300	0.9	47.3	72.5	-119.8	0.35	1.1
0.400	0.5	14.3	79.4	-93.7	0.38	1.2
0.500	0.1	1.5	82.5	-84.0	0.39	1.3
0.540		0.1	82.9	-82.9	0.40	1.3
0.600						
0.700						
0.800						
0.900						
0.910						
1.000						
1.100						
1.200						
1.300						
1.400						

14-year-old Boy Calculations

Drops Vertically into the Pool

Height of COM	h = 1.28	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.54	m =	5.04166667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	256.2	11.3	-267.5	0.27	0.9
0.200	3.9	151.8	19.7	-171.5	0.48	1.6
0.300	3.2	96.7	26.4	-123.1	0.63	2.1
0.400	2.6	63.7	31.7	-95.5	0.76	2.5
0.470	2.2	47.9	34.9	-82.8	0.84	2.8
0.500	2.1	42.4	36.1	-78.5	0.87	2.8
0.600	1.7	27.8	39.7	-67.5	0.95	3.1
0.700	1.3	17.6	42.5	-60.1	1.02	3.4
0.770	1.1	12.3	44.1	-56.4	1.06	3.5
0.800	1.0	10.4	44.7	-55.1	1.08	3.5
0.900	0.7	5.4	46.4	-51.8	1.12	3.7
1.000	0.5	2.1	47.5	-49.7	1.14	3.7
1.010	0.4	1.9	47.6	-49.5	1.14	3.8
1.100	0.2	0.4	48.1	-48.5	1.16	3.8
1.110	0.2	0.3	48.2	-48.5	1.16	3.8
1.170		0.0	48.3	-48.3	1.16	3.8

Drops Diagonally into the Pool

Height of COM	h = 1.05	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.09	m =	3.56499669 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	255.2	16.0	-271.2	0.27	0.9
0.200	3.9	149.1	27.9	-177.0	0.47	1.6
0.300	3.1	92.6	37.1	-129.7	0.63	2.1
0.400	2.5	58.6	44.4	-103.0	0.76	2.5
0.470	2.1	42.3	48.7	-90.9	0.83	2.7
0.500	1.9	36.6	50.3	-86.8	0.85	2.8
0.600	1.5	21.8	54.8	-76.6	0.93	3.1
0.700	1.1	11.7	58.2	-70.0	0.99	3.2
0.770	0.8	6.8	60.0	-66.9	1.02	3.3
0.800	0.7	5.2	60.6	-65.8	1.03	3.4
0.900	0.4	1.4	62.1	-63.5	1.06	3.5
1.000	0.0	0.0	62.6	-62.6	1.06	3.5
1.010		0.0	62.6	-62.6	1.06	3.5
1.100						
1.110						
1.170						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.89	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.23	m^2 =	2.520833 ft^2
Length of Person	L = 0.77	m =	2.52083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.5	321.8	18.8	-340.6	0.23	0.7
0.200	2.3	131.5	29.6	-161.1	0.36	1.2
0.300	1.6	63.9	36.8	-100.7	0.44	1.5
0.400	1.1	32.0	41.9	-74.0	0.50	1.7
0.470	0.9	19.0	44.6	-63.6	0.54	1.8
0.500	0.8	14.9	45.5	-60.4	0.55	1.8
0.600	0.5	5.5	47.8	-53.3	0.57	1.9
0.700	0.2	0.9	49.0	-49.9	0.59	1.9
0.770		0.0	49.2	-49.2	0.59	1.9
0.800						
0.900						
1.000						
1.010						
1.100						
1.110						
1.170						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.47	m^2 =	5.041667 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.2	269.8	37.5	-307.3	0.18	0.6
0.200	1.2	75.6	53.4	-129.1	0.25	0.8
0.300	0.6	22.3	62.2	-84.4	0.30	1.0
0.400	0.2	3.3	66.4	-69.7	0.32	1.0
0.470		0.0	67.1	-67.1	0.32	1.1
0.500						
0.600						
0.700						
0.770						
0.800						
0.900						
1.000						
1.010						
1.100						
1.110						
1.170						

Drops Vertically into the Pool

Height of COM	h = 1.33	m	
Mass of Person	m = 50.79	kg =	112 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.64	m =	5.375 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.4	286.4	10.9	-297.4	0.28	0.9
0.200	4.4	184.1	19.5	-203.6	0.50	1.6
0.300	3.6	125.0	26.5	-151.5	0.68	2.2
0.400	3.0	87.5	32.3	-119.8	0.83	2.7
0.500	2.5	62.1	37.1	-99.3	0.95	3.1
0.530	2.4	56.1	38.4	-94.5	0.99	3.2
0.600	2.1	44.1	41.2	-85.3	1.06	3.5
0.700	1.8	30.9	44.7	-75.6	1.15	3.8
0.800	1.5	21.0	47.6	-68.6	1.22	4.0
0.890	1.2	14.3	49.7	-64.0	1.27	4.2
0.900	1.2	13.7	49.9	-63.6	1.28	4.2
1.000	0.9	8.2	51.8	-60.0	1.33	4.4
1.100	0.7	4.3	53.2	-57.5	1.36	4.5
1.180	0.5	2.2	54.0	-56.1	1.38	4.5
1.200	0.4	1.7	54.1	-55.8	1.39	4.6
1.300	0.2	0.3	54.6	-55.0	1.40	4.6
1.370		0.0	54.7	-54.7	1.40	4.6

Drops Diagonally into the Pool

Height of COM	h = 1.09	m	
Mass of Person	m = 50.79	kg =	112 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.16	m =	3.80069895 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.4	285.6	15.4	-301.0	0.28	0.9
0.200	4.3	181.7	27.5	-209.2	0.50	1.6
0.300	3.5	121.2	37.3	-158.5	0.68	2.2
0.400	2.9	82.5	45.3	-127.8	0.82	2.7
0.500	2.4	56.2	51.9	-108.1	0.94	3.1
0.530	2.3	50.0	53.7	-103.6	0.97	3.2
0.600	2.0	37.6	57.4	-95.0	1.04	3.4
0.700	1.6	24.2	61.8	-85.9	1.12	3.7
0.800	1.2	14.5	65.2	-79.7	1.18	3.9
0.890	0.9	8.2	67.6	-75.8	1.23	4.0
0.900	0.9	7.6	67.9	-75.5	1.23	4.0
1.000	0.5	3.1	69.6	-72.8	1.26	4.1
1.100	0.2	0.6	70.6	-71.3	1.28	4.2
1.180		0.0	70.9	-70.9	1.29	4.2
1.200						
1.300						
1.370						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.92	m	
Mass of Person	m = 50.79	kg =	112 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.25	m^2 =	2.6875 ft^2
Length of Person	L = 0.82	m =	2.6875 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.8	403.4	18.5	-421.8	0.24	0.8
0.200	2.6	178.7	29.6	-208.3	0.38	1.2
0.300	1.9	93.5	37.4	-130.9	0.48	1.6
0.400	1.4	51.7	43.2	-94.9	0.55	1.8
0.500	1.0	28.2	47.4	-75.7	0.61	2.0
0.530	0.9	23.3	48.5	-71.8	0.62	2.0
0.600	0.7	14.2	50.5	-64.7	0.65	2.1
0.700	0.5	5.8	52.7	-58.5	0.68	2.2
0.800	0.2	1.4	53.9	-55.2	0.69	2.3
0.890		0.0	54.2	-54.2	0.69	2.3
0.900						
1.000						
1.100						
1.180						
1.200						
1.300						
1.370						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 50.79	kg =	112 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.50	m^2 =	5.375 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.5	365.3	40.1	-405.4	0.19	0.6
0.200	1.4	113.8	58.5	-172.3	0.28	0.9
0.300	0.9	40.4	69.3	-109.6	0.33	1.1
0.400	0.4	11.0	75.4	-86.4	0.36	1.2
0.500	0.1	0.6	77.9	-78.5	0.37	1.2
0.530		0.0	78.0	-78.0	0.37	1.2
0.600						
0.700						
0.800						
0.890						
0.900						
1.000						
1.100						
1.180						
1.200						
1.300						
1.370						

Drops Vertically into the Pool

Height of COM	h = 1.38	m	
Mass of Person	m = 66.21	kg =	146 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.74	m =	5.70833333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	318.0	10.5	-328.6	0.29	0.9
0.200	4.8	221.9	19.2	-241.1	0.52	1.7
0.300	4.1	160.8	26.6	-187.4	0.72	2.4
0.400	3.5	119.4	32.9	-152.3	0.90	2.9
0.500	3.1	89.8	38.3	-128.2	1.04	3.4
0.600	2.7	68.0	43.1	-111.0	1.17	3.8
0.700	2.3	51.3	47.2	-98.5	1.28	4.2
0.800	2.0	38.4	50.7	-89.1	1.38	4.5
0.900	1.7	28.2	53.8	-82.0	1.47	4.8
1.000	1.4	20.2	56.4	-76.6	1.54	5.0
1.100	1.2	13.9	58.6	-72.5	1.60	5.2
1.200	1.0	9.0	60.4	-69.4	1.65	5.4
1.300	0.7	5.2	61.8	-67.1	1.68	5.5
1.400	0.5	2.6	62.9	-65.4	1.71	5.6
1.500	0.3	0.9	63.5	-64.4	1.73	5.7
1.600	0.1	0.1	63.8	-63.9	1.74	5.7
1.640		0.0	63.9	-63.9	1.74	5.7

Drops Diagonally into the Pool

Height of COM	h = 1.12	m	
Mass of Person	m = 66.21	kg =	146 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.23	m =	4.03640121 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	317.3	14.9	-332.2	0.29	0.9
0.200	4.8	219.8	27.2	-247.0	0.52	1.7
0.300	4.1	157.4	37.5	-194.9	0.72	2.4
0.400	3.5	114.7	46.3	-160.9	0.89	2.9
0.500	3.0	84.0	53.8	-137.8	1.04	3.4
0.600	2.5	61.3	60.2	-121.5	1.16	3.8
0.700	2.1	44.0	65.6	-109.7	1.26	4.1
0.800	1.8	30.8	70.2	-101.0	1.35	4.4
0.900	1.5	20.6	74.0	-94.6	1.43	4.7
1.000	1.1	12.9	77.1	-90.0	1.48	4.9
1.100	0.9	7.2	79.4	-86.6	1.53	5.0
1.200	0.6	3.2	81.1	-84.3	1.56	5.1
1.300	0.3	0.9	82.1	-83.0	1.58	5.2
1.400	0.0	0.0	82.5	-82.5	1.59	5.2
1.500						
1.600						
1.640						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.94	m	
Mass of Person	m = 66.21	kg =	146 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.27	m^2 =	2.854167 ft^2
Length of Person	L = 0.87	m =	2.85416667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.2	511.0	18.2	-529.2	0.25	0.8
0.200	3.0	247.9	30.0	-277.8	0.41	1.3
0.300	2.2	139.4	38.5	-177.9	0.52	1.7
0.400	1.7	83.6	45.1	-128.7	0.61	2.0
0.500	1.4	51.0	50.2	-101.2	0.68	2.2
0.600	1.0	30.5	54.2	-84.6	0.74	2.4
0.700	0.8	17.1	57.2	-74.3	0.78	2.6
0.800	0.5	8.4	59.4	-67.8	0.81	2.7
0.900	0.3	3.1	60.8	-63.9	0.83	2.7
1.000	0.1	0.4	61.6	-62.0	0.84	2.8
1.100						
1.200						
1.300						
1.400						
1.500						
1.600						
1.640						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 66.21	kg =	146 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.53	m^2 =	5.708333 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.9	506.8	43.1	-549.9	0.21	0.7
0.200	1.7	176.6	64.7	-241.3	0.31	1.0
0.300	1.1	72.8	78.2	-151.0	0.37	1.2
0.400	0.7	27.9	86.8	-114.7	0.41	1.4
0.500	0.3	7.1	91.7	-98.8	0.44	1.4
0.600	0.0	0.1	93.5	-93.6	0.45	1.5
0.700						
0.800						
0.900						
1.000						
1.100						
1.200						
1.300						
1.400						
1.500						
1.600						
1.640						

Product Solutions

biscuits • bones • treats • desserts

GRAND OPENING

upswing
in overall
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NINJA CROSS™

NinjaCross MiniNinja Rules

1. Participants must be a minimum of 48-inches tall
2. Participants maximom weight of 275lbs
3. Wait your turn to start, follow direction by facility staff at all times
4. Diving, jumping, running, pushing, etc. is strictly prohibited
5. Participants to use systems solely at their own risk – this is a skill-based system and is meant to be challenging. Owner, operator, manufacturer and any additional parties will not be held responsible for any injury on the system
6. Climbing obstacles cables, structure column legs or any other components on the system is strictly prohibited
7. Touching obstacle frame or support truss, electronics, or any other components other than the obstacles is strictly prohibited
8. Only use if you are capable of safely swimming the length of the pool and able to hold your breath under-water for 10-seconds or more. Non-Swimmers are not permitted.
9. Only 1 participant per obstacle set at a time, no more than 3 participants on the system at one time
10. Use only under supervision of lifeguard or attendant
11. If you fall into water, move on to next obstacle or swim out of the lane
12. If you feel exhausted or weak, stop participation and swim out of lane to closest pool wall
13. Do not push, shove or harass other guests – bullying will not be tolerated and you may be asked to leave the facility
14. Do not use this equipment while under the influence of alcohol or drugs
15. No diving allowed anywhere around this system
16. Leave MiniNinja pool area promptly after completing the course or if you are unable to complete the course
17. Participants assume all risk of injury due to misuse of the NinjaCross MiniNinja or failure to follow rules

NINJA CROSS

NinjaCross Systems

MiniNinja

Standard Operating Procedures and Operations Manual V1.1

Contact Ninja*Cross* Systems at:

Phone- 800-778-9702

Email- Support@NinjaCrossSystems.com

Section 1

Introduction

The purpose of this operations manual is to provide the owner/operator with the basic rules and maintenance information necessary to operate the NinjaCross MiniNinja System in a manner designed to minimize problems and ensure the safety of the participant(s). This manual deals with the operation of the NinjaCross equipment only. It does not address pool operations, health codes, water quality, or local ordinances.

Facilities should follow the manufacturer's guidelines for installation, safe inspection, maintenance, operations and use of its various fitness systems and features. However, your employer should provide you with a specific set of guidelines and training if you are responsible for these inspections

Most local regulatory agencies have public swimming pool standards. It is recommended that local codes, regulations, and guidelines be followed. This will insure a harmonious relationship between the pool/slide operation and the local authorities.

To assist owners and operators in providing a safe, fun, and enjoyable experience for all facility patrons, NinjaCross Systems provides the following additional services;

- Annual NinjaCross Inspections
- Annual on-site safety training for lifeguards and operators
- Maintenance programs to prolong the life of your investment

Section 2

Terms

Box Truss - a type of truss that uses four major cords with connecting cords to form a strong structure that takes the shape of a rectangular box.

Corner Block - a 12” square aluminum block that mounts to the Aluminum Box truss section. All Static Lines attach at a Corner Block and all cross members of the Obstacle Frame attached at Corner Blocks.

Designated Safety Area - the area that includes all pool space under the obstacle frame and the adjacent 8-feet on either side of the Obstacle Frame stretching from end of pool to opposite end.

Eye Clamp - A clamp that allows attachment of a NetForm Rope or other item to the Obstacle Frame.

Mounting Plate - the square aluminum plate that secures the Obstacle Frame to the pool deck. The plate is anchored by wedge anchors.

NetForm Rope - the rope that connects an obstacle to the Obstacle Frame

Obstacle - a combination of aluminum parts, ropes, and hardware that create a means for the participant to traverse.

OAB (Obstacle Attachment Bar) - An aluminum bat that attached to the Obstacle Frame and allows Obstacles with dual ropes to be attached.

Obstacle Frame - the aluminum truss that Obstacles hang from, Static Cables and Lifting Cables attach to, and BackUp System attaches to.

Obstacle Frame Leg - the aluminum truss vertical sections that hold the Obstacle Frame at elevation. These legs are mounted to the pool deck via the Mounting Plates.

Participant - the guest that is using the NinjaCross MiniNinja system

Pinch Block - an aluminum block with indents that allows it to secure into the tube of the Obstacle Frame. Used for connecting Obstacles to the Obstacle Frame.

Safety Padding - a section of padding applied to deck and pool wall that protects participant from falls against the pool deck.

Swivel Clamp - A dual clamp system that allows attachment of the OAB to the Obstacle Frame.

Section 1

NinjaCross MiniNinja Standard Rules

1. Follow the directions of facility personnel at all times

2. Wait your turn prior to starting

3. Diving, jumping, running, pushing, etc. is strictly prohibited

4. Participants to use system solely at their own risk - this is a skill-based system and is meant to be challenging

5. Climbing obstacle cables, legs, or any other components on their system is strictly prohibited

6. Touching obstacle frame or support truss, electronics, or any other components other than the obstacles is strictly prohibited

7. Do climb the ropes or onto the Obstacle Frame. Do not try to hold onto the Obstacle Frame

8. Only use if you are capable of swimming and able to hold your breath under water for 10-seconds or more

9. Only one participant per obstacle set at a time. A maximum or 2 participants may be on a single lane at any one time. The minimum distance between participants shall be no less than 10’

10. Use only under the supervision of lifeguard or attendant

11. Swinging, leaping, jumping, or swimming in adjacent lane is strictly prohibited

12. No standing on Above Water Level obstacles

13. If you fall on an obstacle, move onto the next obstacle and attempt to complete

14. If you feel exhausted or weak, stop participation and swim out of lane to closet pool wall

15. Do not push, shove, or harass other guests - bullying will not be tolerated, and you will be asked to leave facility.

16. Recommended Minimum age 5 years old

17. Minimum Height 48” tall

18. Maximum Weight 270lbs

19. Participant must not wear lifejacket, shoes (including swim shoes), loose jewelry, or other item of clothing that may get caught in obstacles

20. Lifeguards are responsible for final determination of swim ability, age, and height according to the existing rules of the facility.

21. Intoxicated person are not allowed to use the system or operate the system

22. No spectators in the designated safety area of the pool

Section 2

End of Day Procedures

End of Day Washdown

•This procedure should be followed on a daily basis

• Rinse the Obstacle Frame including the Ropes, Plates, and other attachments with fresh water

Section 3

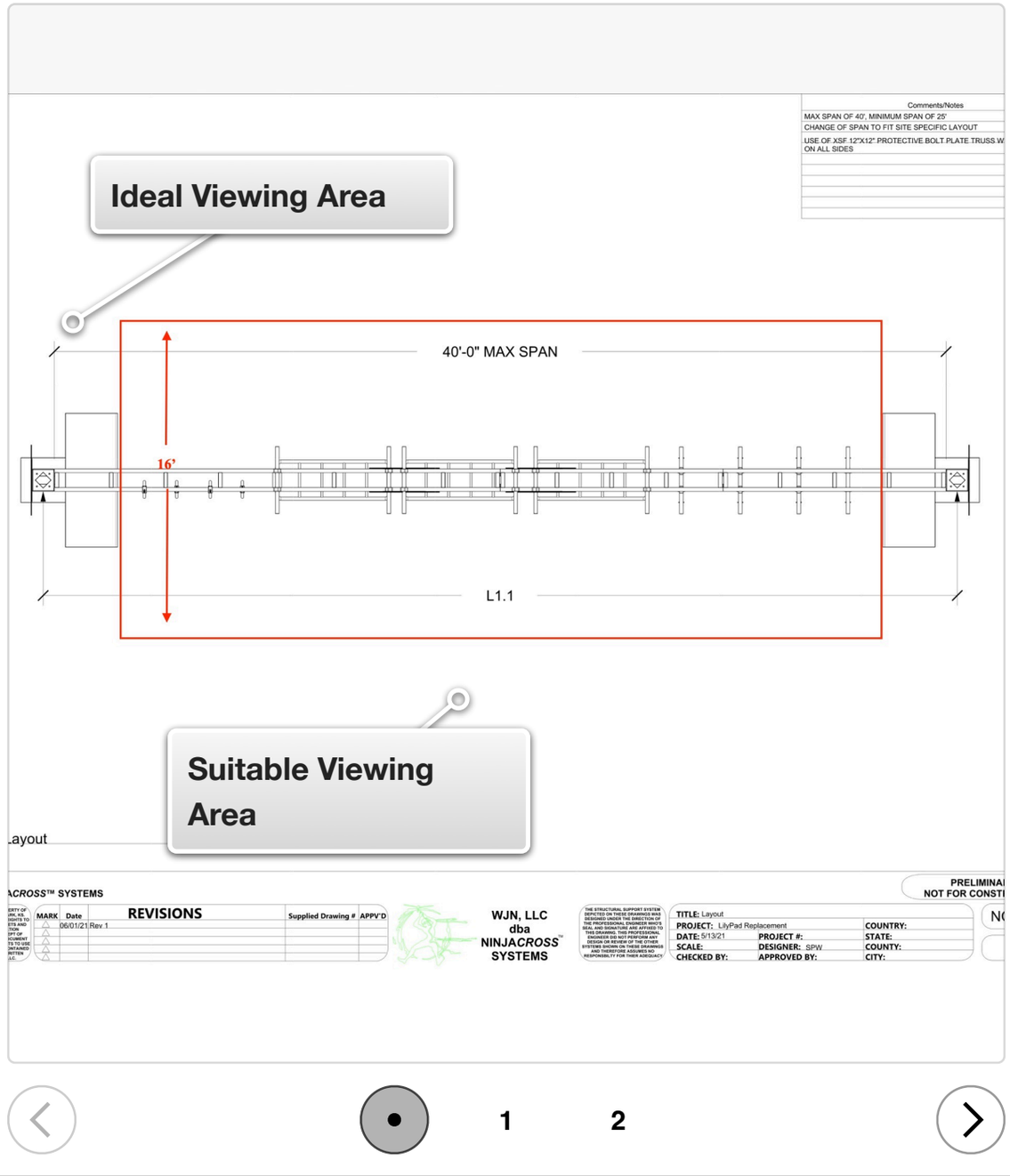
Designated Safety Area

he Designated Safety Area is the zone where only participants may be in the pool during the operating time of the NinjaCross MiniNinja System. The safety area is detailed as the area directly under the Obstacle Frame as well as an additional 8-feet on either side of the Obstacle Frame stretching from end of pool to end of pool.

During operations, spectators are prohibited from entering the Designated Safety Area.

Participants who quit the course without finishing shall be instructed to exit the course to the outside of the Designated Safety Area without crossing the path of other participants and exit the Designated Safety Area as quickly and safely as possible.

Interactive 3.1 Designated Safety Area



Section 4

Seasonal Shut Down Procedures

Long Term Shutdown

Procedures

When storing the Ninja*Cross* MiniNinja system for winter or long-term storage, the following steps should be taken.

1. Remove all obstacles and store in a secure safe location. Take care in storing obstacles as to not damage the materials or coating.

2. Store Ropes in a dry location free of any chemicals.

Section 1

Obstacle Types

There are two types of obstacles with the NinjaCross MiniNinja System a) OAB mounted obstacles, and b) Direct frame mounted obstacles.

OAB mounted obstacles are those obstacles that use 2 or more cables attached to the obstacle and require a spacing of more than 12” between the NetForm ropes. The OAB attaches to the Obstacle Frame by way of 2 Swivel Clamps. Obstacles attach to the OAB via the stud connection on the OAB and the shackles of the NetForm Rope.

Examples of OAB Mounted Obstacles are:

Trapeze Bars Low Bars Ladders

Direct frame mounted obstacles are those obstacles that attach to the Obstacle Frame directly by use of an Eye Clamp or other method.

Examples of Direct Mounted Obstacles are:

Sea of Discs Overhead Rings CannonBall Alley

Section 2

Obstacle Mounting Procedures

In order to mount any obstacle using a Swivel Clamp or Eye Clamp the following procedures need to be followed

1. Ensure that the Obstacle Frame is fully deployed in its operational position and the pool is clear of all swimmers.

2. Choose location for obstacle to be mounted.

3. Choose correct type of clamp for the obstacle to be installed

4. Unscrew the wing nut on the clamp to allow clamp to easily open

5. Place clamp in position, close the clamp over the Obstacle Frame tube, close bolt into clamp tab ensuring that the wing nut and washer clear the top of the clamp.

6. Tighten the wing nut until snug, do not over tighten as damage may occur to the Obstacle Frame truss

7. Attach obstacle to Eye Clamp or attach OAB to Swivel Clamps.

   a. If using an Eye Clamp, open the shackle at end of the NetForm Rope by turning the shackle pin counterclockwise using an Allen wrench. Place shackle over the open eye of the clamp and insert shackle pin into the shackle through the eye of the clamp. Tighten shackle pin (the use of blue Loctite will ensure shackle does not come loose.)

   b. If using an OAB, open the shackle at end of the NetForm Rope by turning the shackle pin counter-clockwise using an Allen wrench. Place shackle over the open stud of the OAB and insert shackle pin into the shackle through the stud of the OAB. Tighten shackle pin (the use of blue Loctite will ensure shackle does not come loose.)

When moving Obstacles from initial installed location, please refer to the Obstacle Water Depth Chart included in this manual to ensure obstacles are installed over the proper depth of pool.

Access to truss can be by use of a secured ladder in the pool leaned up against the Obstacle Frame or by use of the EZ Dock floating dock system. Care must be taken to not put excessive lateral force on the Obstacle Frame at any time, and at no time should staff sit, stand, or walk on the Obstacle Frame for access.

Obstacle Water Depth

Obstacle	Min Water Depth in Feet
Overhead Rings	4
Rising Rings	4
Cannonball Alley	5
Low Bar	4
Trapeze Bar	4
Ladder	4
Camelback	5

Section 3

Obstacle Frame

The Obstacle Frame is a 12”x12” aluminum box truss connected by way of Corner Blocks. The Obstacle Frame is the connection point for all Obstacles. The Obstacle Frame is designed to distribute the weight of the Obstacles and participants over a specified range according to the individual design of each system.

The Obstacle Frame is bolted together with 5/8”x2.5” Stainless Steel or Galvanized Bolts. The bolts utilize 5/8” washers and 5/8” nylon washers. The Nylon Washers prevent galvanic reactions from occurring on the different metal types of the bolts and Obstacle Frame.

12”x12” 6-way Corner Blocks are installed every at the vertical legs. All cross members of the Obstacle Frame are connected at Corner Blocks. Corner Blocks utilize the same 5/8” hardware as other parts of the Obstacle Frame.

Section 1

Obstacle Frame Maintenance

Cleaning

Powder coated aluminum should require little maintenance, other than to gently clean with water and a mild liquid detergent to remove any dirt or splashes. A microfiber cloth or sponge should be used to wipe over the surfaces of the furniture. Removing surface water with a drying cloth (like you would use on your car) will help avoid water spots. Avoid using any abrasive cleaning agents or materials, as these could mark the surface of the powder coat. Do not use steel wool or Scotch-Brite on powder coated surfaces.

Section 2

Obstacle Maintenance

Aluminum Obstacles

Cleaning

Powder coated aluminum should require little maintenance, other than to gently clean with water and a mild liquid detergent to remove any dirt or splashes. A microfiber cloth or sponge should be used to wipe over the surfaces of the furniture. Removing surface water with a drying cloth (like you would use on your car) will help avoid water spots. Avoid using any abrasive cleaning agents or materials, as these could mark the surface *of the powder coat. Do not use steel wool or Scotch-Brite on powder coated surfaces.*

Paint and Coatings Care

Over the course of use, the obstacles will receive chips and scraps on the coated surfaces. It is important that these chips and scraps be attended to as soon as they are discovered to prevent them from worsening. When a chip or scrap is discovered it is important to follow these procedures.

1. Remove obstacle from the water
2. Completely dry the obstacle and wipe clean any dirt or residues
3. Apply touch up paint to effected area
4. Allow paint to completely dry before allowing obstacle to get wet

Ropes

Cleaning

Rinse with clean fresh water, do not use chemicals or abrasives.

Section 3

Material Specific Maintenance

The following pages have information on the proper methods for cleaning specific types of metals found in the Ninja*Cross* MiniNinja System. If you have any questions, please contact Ninja*Cross* Systems for advise.

Care and Cleaning of Stainless Steel

Introduction

Cleanliness and stainless steel are closely related and, in many applications, each is dependent upon the other. In the handling of food, chemicals, pharmaceuticals and in the use of stainless steel as a construction material (roofs, wall panels, entry ways, signs, etc.), stainless steel provides the degree of corrosion resistance that is necessary to prevent product contamination or surface rusting. However, stainless steel performs best when clean — cleanliness is essential for maximum resistance to corrosion.

This handbook describes various practices for cleaning stainless steel during manufacture and in use. This includes methods for removing free-iron contamination on stainless steel surfaces that may have been picked up from metalworking tools; and for removing general accumulation of dirt, grime and surface stains that occur during normal handling and exposure to the elements.

The reader should keep in mind that there are few specific rules for a cleaning procedure. Accordingly, the methods discussed in this handbook are suggestions. Each manufacturer or user, after obtaining competent advice with respect to their individual requirements, should select methods appropriate to those requirements.

What is Stainless Steel?

Stainless steel is not a single alloy, but rather the name applies to a group of iron-based alloys containing a minimum 10.5% chromium. Other elements are added and the chromium content increased to improve the corrosion resistance and heat resisting properties, enhance mechanical properties, and/or improve fabricating characteristics. There are over 50 stainless steel grades that were originally recognized by the American Iron and Steel Institute (AISI). Three general classifications are used to identify stainless steel. They are:

1) Metallurgical structure.

2) The AISI numbering system (200, 300 and 400 series numbers).

3) The Unified Numbering System, which was developed by the American Society for Testing Materials (ASTM) and the Society of Automotive Engineers (SAE) to apply to all commercial metals and alloys.

The various types of stainless steel are detailed in a designer handbook, “Design Guidelines for the Selection and Use of Stainless Steel,” available from the Specialty Steel Industry of North America (SSINA). Several other publications are also available, including: “Stainless Steel Fabrication,” “Stainless Steel Fasteners,” “Stainless Steel Finishes,” “Stainless Steel Specifications,” and “Stainless Steel Architectural Facts,” to mention a few.

Alloy Types

304 is the basic chromium-nickel austenitic stainless steel and has been found suitable for a wide range of applications. It is the most readily available in a variety of product forms. This grade is easy to form and fabricate with excellent resistance to corrosion.

•304L is the low carbon version of 304. It is sometimes specified where extensive welding will be done.

•316 offers a more corrosion-resistance through the addition of molybdenum. This grade is desirable where the possibility of severe corrosion exists, such as heavy industrial atmospheres and marine environments.

•316L is the low carbon version of 316.

•430 is a straight chromium ferritic stainless steel with lower corrosion resistance than the 300 series. It is principally employed for interior use.

Cleaning of Stainless Steel

Stainless steels need to be cleaned for aesthetic considerations and to preserve corrosion resistance. Stainless steel is protected from corrosion by a thin layer of chromium oxide. Oxygen from the atmosphere combines with the chromium in the stainless steel to form this passive chromium oxide film that protects from further corrosion. Any contamination of the surface by dirt, or other material, hinders this passivation process and traps corrosive agents, reducing corrosion protection. Thus, some form of routine cleaning is necessary to preserve the appearance and integrity of the surface. Stainless steels are easily cleaned by many different methods. They actually thrive with frequent cleaning, and unlike some other materials, it is impossible to “wear out” stainless steel by excessive cleaning. The effect of surface/pattern roughness, grain/pattern orientation and designs that allow for maximum rain cleaning (exterior applications)should be considered.

Types of surface contaminants

• Dirt -Like any surface that is exposed to the environment, stainless steel can get dirty. Dirt and soil can consist of accumulated dust and a variety of contaminates that come from many sources, ranging from the wind to everyday use. These contaminates will vary greatly in their effect on appearance and corrosively and ease of removal. While some may be easily removed, others may require specific cleaners for effective removal. It may be necessary to identify the contaminate or experiment with various cleaners. Frequently, warm water with or without a gentle detergent is sufficient.

Next in order are mild non-scratching abrasive powders such as typical household cleaners. These can be used with warm water, bristle brushes, sponges, or clean cloths. Ordinary carbon steel brushes or steel wool should be avoided as they may leave particles embedded on the surface which can lead to RUSTING. For more aggressive cleaning, a small amount of vinegar can be added to the scouring powder. Cleaning should always be followed by rinsing in clean hot water. When water contains mineral solids, which leave water spots, it is advisable to wipe the surface completely with dry towels.

• Fingerprints and Stains -Fingerprints and mild stains resulting from normal use in consumer and architectural applications are the most common surface contaminates. Fortunately, these usually affect only appearance and seldom have an effect on corrosion resistance. They are easy to remove by a variety of simple cleaning methods. Fingerprints are probably the most troublesome marks to remove from the surface of smooth polished or bright finished stainless steel. Fortunately, they can be removed with a glass cleaner or by gentle rubbing with a paste of soda ash (sodium carbonate) and water applied with a soft rag. Once again, this should be followed by a thorough warm water rinse. There are several special surface finishes where fingerprints present special problems: polished No. 6, etched, some abrasive blasted finishes, and light electrochemical colors applied over satin or brushed finishes.

(NOTE: there are several special finishes designed to withstand fingerprints: embossed, swirl patterns, lined patterns, etc.).

• Shop oil and Grease -Shop oils, which may carry grease, grit and metal chips, commonly produce surface soiling after many shop operations. Greases and other contaminates may also soil surfaces in food preparation and many other household and commercial situations. These soils may be corrosive in themselves or may not allow the surface to maintain passivity, and so periodic removal is a necessity. Initially, soap or detergent and water may be tried or a combination of detergent and water plus a solvent. The removal of oil and grease from stainless steel parts by immersion in chemical solvents is frequently used with cold-formed or machined parts that are laden with lubricants. This process, in its simplest form, consists of bringing liquid solvent into contact with the surface to be cleaned and allowing dissolution to take place; for example, washing a surface with trichloroethylene or similar liquid or stirring a batch of small parts in a container of solvent. Non-halogenated solvents, such as acetone, methyl alcohol, ethyl alcohol, methyl ethyl ketone, benzene, isopropyl alcohol, toluene, mineral spirits, and turpentine work well.

Many of these solvents are widely used as individual cleaners, but there are thousands of blended or compound cleaners on the market. Users are advised to contact suppliers of solvents for information on their applications on stainless steel.

Types of Cleaners and Methods

General Precautions

In selecting cleaning practices, consider the possibility of scratching and the potential for post-cleaning corrosion caused by incompletely removed cleaners. Scratching can occur on a bright mirror finish by cleaners that contain hard abrasives, or even by "grit" in wash water. This is usually not a problem on dull finishes, or those surfaces finished with a coarse polishing grit. The best preventative measure is to avoid using abrasive cleaners unless absolutely necessary. When abrasives are needed, first experiment on an inconspicuous area. A "soft abrasive," such as pumice, should be used. Abrasives can permanently damage some colored and highly polished finishes. Advice should be obtained from the finish supplier when cleaning special finishes. Many cleaners contain corrosive ingredients which require thorough post-clean rinsing with clean water; however, thorough rinsing is recommended for all cleaning procedures.

• **Clean Water and Wipe** - The simplest, safest, and least costly method that will adequately do the job is always the best method. Stainless surfaces thrive with frequent cleaning because there is no surface coating to wear off stainless steels. A soft cloth and clean warm water should always be the first choice for mild stains and loose dirt and soils. A final rinse with clean water and a dry wipe will complete the process and eliminate the possibility of water stains.

• **Solvent Cleaning** -Organic solvents can be used to remove fresh fingerprints and oils and greases that have not had time to oxidize or decompose. The preferred solvent is one that does not contain chlorine, such as acetone, methyl alcohol, and mineral spirits. There are many compounded or blended organic cleaners that are commercially available and attempt to optimize both clean ability and safety attributes. Cleaning can be accomplished by immersing smaller articles directly into the solvent, wiping with solvent-impregnated cloths, or by sophisticated vapor or spray methods. The wiping technique sometimes leaves a streaked surface.

Effective Cleaning Methods

• **Household Cleaners -** Household cleaners fall into two categories: detergent (non-abrasive) and abrasive cleaners. Both are effective for many mild dirt, stain, and soil deposits, as well as light oils such as fingerprints. The abrasive cleaners are more effective but introduce the possibility of scratching the surface. However, the degree of abrasiveness will vary greatly with the particular product, and some brands will produce noticeable scratching on only the most highly polished and some colored surfaces. All of these cleaners vary widely with respect to their acidity and the amount of chloride they contain. A neutral cleaner low in chloride is preferred unless the user is assured that the surface can be thoroughly rinsed after cleaning. The fact that the label states “for stainless steel” is no guarantee that the product is not abrasive, not acidic, or low in chloride. The cleaning method generally employed with these cleaners is to apply them to the stainless surface and follow by cloth wiping, or to wipe directly with a cleaner-impregnated soft cloth. In all cases, the cleaned surface should be thoroughly rinsed with clean water and wiped dry with a soft cloth if water streaking is a consideration.

• **Commercial Cleaners -** Many commercial cleaners compounded from phosphates, synthetic detergents, and alkalis are available for the cleaning of severely soiled or stained stainless surfaces. When used with a variety of cleaning methods, these cleaners can safely provide effective cleaning. Manufacturers should be consulted and their recommendations followed whenever using cleaners of this kind. The general precautions stated above also pertain to these cleaners.

Care of Stainless Steel

The cleaner stainless steel can be kept while in storage, being processed or during use, the greater the assurance of optimum corrosion resistance. Some tips on the care of stainless steel are listed below:

1) Use paper or other protective wrapping on the surface of the stainless steel until processing is complete.*

2) Handle stainless steel with clean gloves or cloths to guard against stains or finger marks.

3) Avoid the use of oily rags or greasy cloths when wiping the surface.

4) Do routine cleaning of exposed surfaces. Buildings with window washing systems can utilize this method to clean exterior panels.

5) Where possible, after cleaning, rinse thoroughly with water.

6) Cleaning with chloride-containing detergents must be avoided.

7) Even the finest cleaning powders can scratch or burnish a mill-rolled finish. On polished finishes, rubbing or wiping should be done in the direction of the polish lines, NOT across them.

8) **DO NOT USE SOLVENTS** in closed spaces or while smoking.

*Many adhesive-backed papers and plastic sheets or tape applied to stainless steel for protection “age” in fairly short periods of time and become extremely difficult to remove.

Manufacturers should be contacted regarding information as to how long protective films or paper can be left in place.

Acknowledgments

The Specialty Steel Industry of North America (SSINA) acknowledges that this new handbook contains information originally published by the Committee of Stainless Steel Producers, American Iron and Steel Institute, which no longer exists. Current SSINA member companies were represented on that committee. The SSINA wishes to acknowledge the contributions of the Nickel Development Institute and its consultant, Technical Marketing Resources (Pittsburgh, PA) for help in preparing the contents of this handbook.

Powder Coating Care and Maintenance

Proper Care of Powdered Surfaces Is Essential

Powder coatings that are applied to metal products exposed to the weather will inevitably degrade over time. A number of conditions, including those found in nature, will contribute to shortening the life of this type of protective finish.

•Sun

•Rain

•Wind

•Pollution

•Cold weather

•Salt water

•Electrical current

•Dissimilar metals

How to Maintain Powder Coated Surfaces

1. Avoid harsh chemicals: Unlike spray paint, powder coating is much more resistant to things like rust, corrosion, peeling and fading. However, that resistance does not mean it's completely fine to use chemical cleaners and solvents to clean powder coated items. Harsh cleaners and solvents like acetone can actually damage powder coating.

2. Clean gently: You can still clean powder coated surfaces. Just wipe off dust with a soft clock. If more cleaning is necessary, use a highly diluted, mild soap in water and a soft towel or soft sponge to very gently clean. Rinse with a little water, then dry with another soft towel.

3. Wax: If your powder coated metal has lost its gloss and shine, after removing dirt with mild soap, you can apply a thin layer of wax just like you do after you wash your car. After the wax dries, wipe all of it off and powder coated metal will look like new.

4. Don't paint: If you're wondering if you can touch up imperfections and rust with paint, the answer is no. Because of how the powder coating process works, paint won't adhere to powder coated surfaces. If your powder coating is starting to show signs of wear and tear, it's time to have a professional either repair or redo the powder coating.

5. Maintenance schedules: We recommend you regularly inspect and clean your powder coated items. How often you wipe your metal surfaces clean depends on the amount of dirt and grime in the area, the time of year, and if there's been any intense weather like a hurricanes or tornados.

NetForm Ropes

System Inspection

NetForm structures and associated hardware including backing nets, cables and fasteners should be inspected by a competent person after installation and on a regular scheduled basis thereafter. It is good practice to keep a dated and signed maintenance log of each netting system to assure that all safety measures have been followed.

The system must be inspected following alterations, repairs and impact loading. If any welding or cutting operations occur near the structures, weld protection must be provided for that area, and more frequent inspections should be conducted in proportion to the dangers involved.

NetForm should be inspected on a daily and weekly basis.

• Daily Inspection should include a quick visual of the NetForm and any backing netting, to look for any obvious broken net mesh or frays. Report for replacement any missing NetForm cross joints or tees.

• Weekly Inspection should include any lashing cord that may be used in the NetForm system, including loose and broken lashes. Repair as necessary. Visually check and hand-test all rope handrails, hardware, cables, anchors, etc. All hardware should be in place with no substitutes. Document any faults with a photograph to help expedite repairs.

General Environmental Inspection

NetForm, backing nets or hardware that show deterioration from mildew, corrosion, wear, or stress, that may affect their strength, must be immediately removed from service for further inspection, repair or disposal.

• Inspect the NetForm and backing nets for cuts, pulls, fraying of material and discoloration indicating material aging.

• Inspect cross joints and tees for stress cracking.

• Inspect support cables for cuts, twists, kinks, fraying of strands and corrosive rust.

• Inspect support and anchor hardware to assure fasteners are properly secured and that no pieces are missing. Look for damaging rust that may affect hardware strength or abrade the NetForm or backing nets.

Repairs

Field repairs and modifications may be done with guidance and materials from the manufacturer. Photographs are always the best way to convey the extent of a fault area. If replacement of a net panel or system is required, the manufacturer will determine the best method of replacement.

ABS Wrap/Signage Care

• Clean debris from wraps and signage as they appear dirty. Failure to remove debris may make care more difficult over time.

• Test any cleaning solutions on a small section of wrap before using to clean wrap.

• Use a wet, non-abrasive detergent and a soft clean rag for cleaning.

• Rinse thoroughly with clean water. Dry with a microfiber cloth.

• If choosing to wax the wrap, use only waxes that do not contain petroleum distillates

• Do not use mechanical brushes or pressure washers to clean the wraps. Doing so may damage the graphics or wraps themselves.

Vertical Truss Leg Wraps are not included in base MiniNinja System. NinjaCross Systems suggests the use of wraps to prevent access to the Obstacle Frame.

Section 1

Daily Pre-use Inspections

Prior to use each day, the system must undergo a complete Pre-use Daily Inspection to ensure that the system components are in proper working order and ready for use. This is a comprehensive inspection that is done at start of each day.

The complete system SHALL undergo the following inspections as laid out and documented. Any problems, concerns, or points of interests SHALL be noted in the inspection logs for review by NinjaCross Systems.

1. Ensure that the Obstacle Frame Legs are secured to the mounting plates.

2. Ensure Obstacle Frame is secure and not damaged.

3. Ensure that all Obstacles are in proper placement and not entangled in the Obstacle Frame, OAB's, or Signage.

4. Check the pool and surrounding deck for parts, hardware, or materials that may have fallen.

5. Ensure all Obstacles are at their proper depth in the pool and are located as designed.

6. Inspect NetForm Ropes for damage, broken strands, or opening or fraying. Check for mildew or staining.

7. Have lifeguards run through both lanes to ensure system is operating correctly.

8. Ensure that all signage is undamaged, visible without obstructions, and can be viewed by participants on the deck.

9. Document inspection and note any concerns or problems.

Section 2

Quarterly Inspection

The complete system **SHALL** undergo the following quarterly inspections as laid out and documented. Any problems, concerns, or points of interests **SHALL** be noted in the inspection logs for review by NinjaCross Systems.

1. Obstacle Frame -

a. Check that Obstacle Frame joints, where two Truss Sections meet or a Truss Section and Corner Block meet, are secure and not loose.

b. Ensure that all hardware is present at every joint, each Truss Section is bolted to a Truss Section or Corner Block with 4 bolt assemblies.

c. Check for chipped paint

d. Checked for cracked paint, cracked paint may indicate a stress fracture in the truss cord.

e. Ensure that the Obstacle Frame is level both side to side and front to back

f. Rinse frame with fresh water

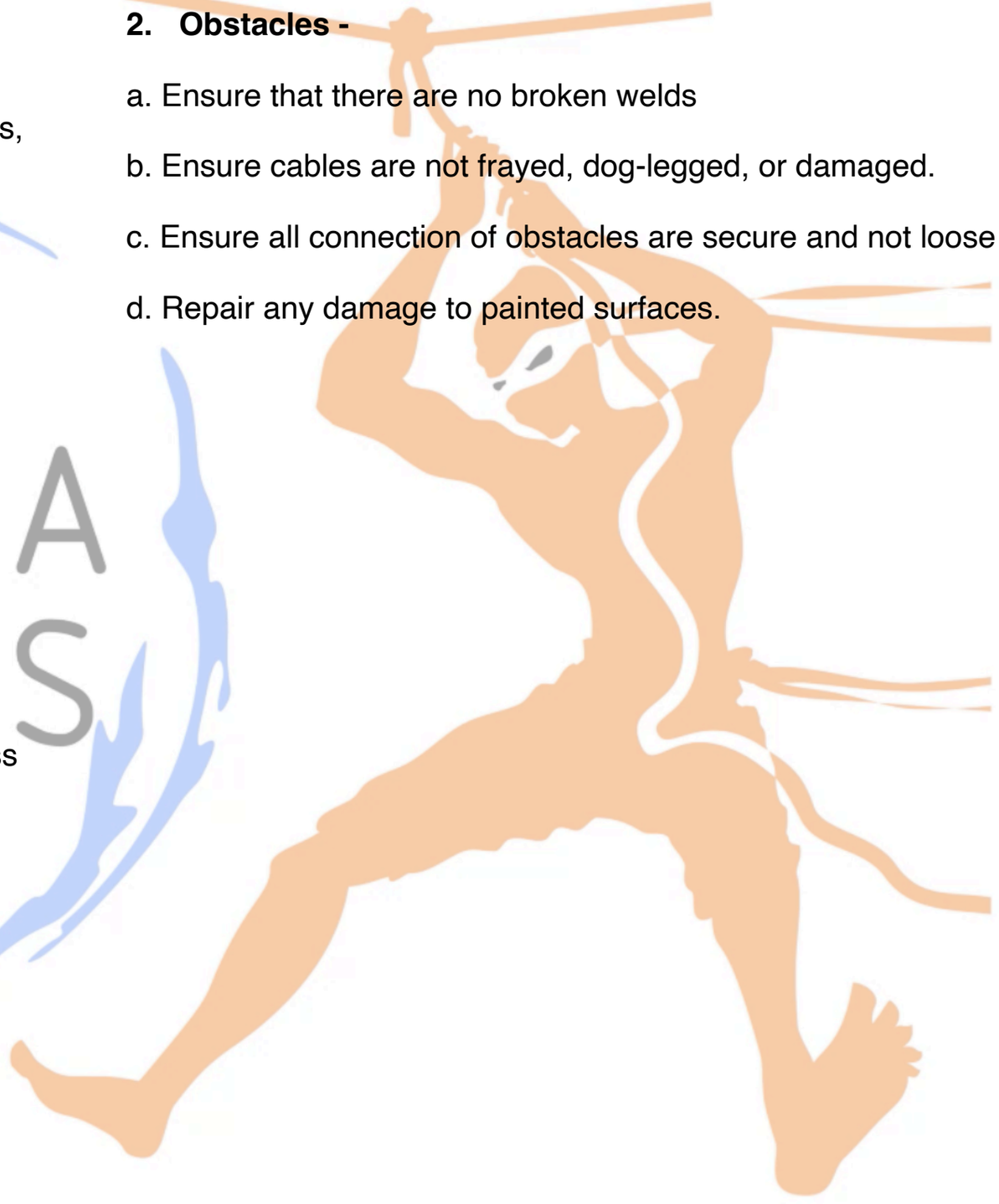
2. Obstacles -

a. Ensure that there are no broken welds

b. Ensure cables are not frayed, dog-legged, or damaged.

c. Ensure all connection of obstacles are secure and not loose

d. Repair any damage to painted surfaces.



Section 3

Yearly Inspection

All NinjaCross MiniNinja System components **SHALL** be inspected annually by NinjaCross Systems or an authorized representative. Failure to have the system inspected will result in NinjaCross Systems notifying all relevant inspection authorities that the system cannot be declared safe to use by manufacturer.

A minimum of 4-weeks' notice to NinjaCross Systems must be given for scheduling the annual inspection. Contact NinjaCross Systems via your sales contact or directly at Support@NinjaCrossSystems.com

Annual Inspection **SHALL** include and inspection of the following items to ensure the safe and proper working order of the NinjaCross MiniNinja System.

1. Obstacle Frame System including mounting plate
2. Obstacles
3. Inspection and Maintenance Logs

Section 4

Inspection Forms

Ninja*Cross* Systems has provided the following sample inspection forms for use or as a guideline to creating your own inspection forms. At minimum, all inspection forms must include the items including in each form.

Inspection Forms

Ninja*Cross* **:** Daily Pre-use Inspection

Description **:** Daily safety checks to be performed prior to use.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	Monthly
Obstacle Frame Mounting Plate																																
Obstacle Frame																																
Obstacle Ropes																																
Obstacles																																

Notes **:**

Inspection Forms

Ninja*Cross* **:** Quarterly Inspection

Description **:** System inspection every 3 months

	1st Quarter	2nd Quarter	3rd Quarter	4th Quarter
Obstacle Mounting Plate and Anchors				
Obstacle Frame Connections				
Obstacle Connections				
Obstacle Rope integrity				
Obstacle integrity				

Notes **:**

Certification and Training

Detailed instructions, extensive training, and actual operations specific to each NinjaCross MiniNinja System will be provided and addressed during the on-site Training and Certification conducted by The Owner/Operator of the MiniNinja System. The Owner/Operator will be responsible for instructing the operations team on Operations, Daily, Quarterly, Seasonal and Annual Inspections, Safe Usage, Procedures to follow in order to minimize possible injuries as well as cleaning and maintenance to

insure the longevity of your obstacle course & fitness system.

Personnel Training

(Please Note the Following Contains the Manufactures Minimum Recommendations but are Subject to Your Facilities Local and State Codes as well as contracted Third Party Organizations such as the American Red Cross)

Having properly trained and conscientious employees on site is the most important safety factor in the operation of the NinjaCross MiniNinja System.

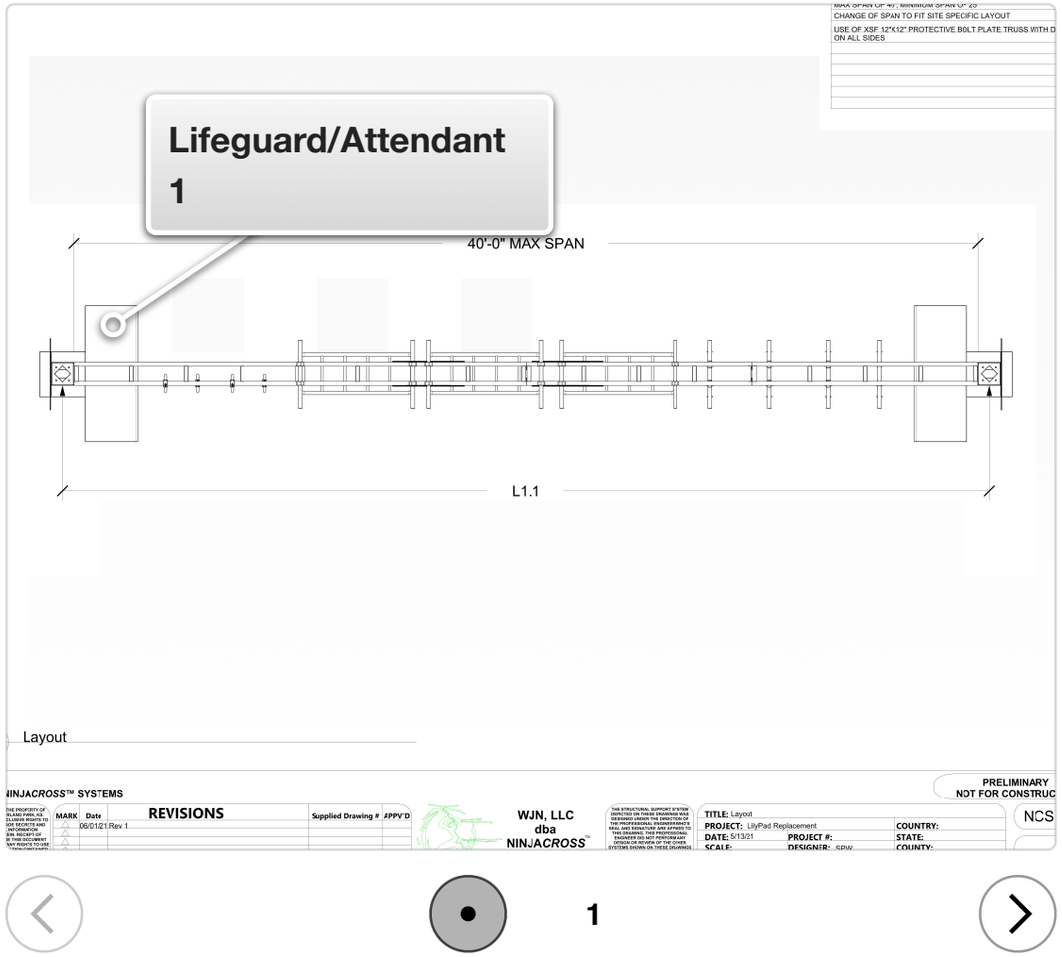
It is our recommendation that all employees who are responsible for the NinjaCross MiniNinja System operations be certified lifeguards and be qualified in both first-aid and life-saving techniques through the American Red Cross training or the equivalent. At least one person who has completed the Standard First Aid and Personal Safety course, as offered by the American Red Cross, or the equivalent should be on duty always during operating hours. This person should also be competent in carrying out any emergency procedures peculiar to the slide he or she is operating. Under most conditions, this is also a recommendation of the insurance carrier if applicable.

Each owner/operator shall have written operating procedures for the NinjaCross MiniNinja System, which are an integral part of their staff-training program. These procedures shall include but not be limited to:

Lifeguard/Attendant Station 1 - one trained lifeguard/attendant SHALL be stationed at the edge of the pool at the starting location. This staff duties are to ensure that all Participants start in the water, to ensure the proper spacing of Participants at the start, and to observe Participants at the start of the course.

All NinjaCross MiniNinja personnel should be alert to controlling crowd behavior and the proper entry rate into the pool; therefore, we recommend the line to participate be formed on the pool deck rather than the pool edge. One Participant may be stationed at the edge of pool to start the course, while any additional may be at a point away from the pool edge preparing to move into starting position at the command of the lifeguard/attendant. Once the Participant who is at edge of pool starts the course the Participant

Interactive 7.1 Lifeguard/Attendant locations



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on the deck enters the starting area at the edge of the pool then the line then moves up one position.

Lifeguards at the start of the course should address each and every Participant when it is their turn and then inform the Participant on the rules of the course prior to starting the course. All Participants should be instructed how to use the course and not allowed to run, jump, or leap into the pool. The Lifeguard(s) stationed at start will address each Participant first by asking that they follow their instructions and Do Not proceed into the pool until they are given the okay to do so.

Safe and orderly exit from the pool area helps reduce the risk of disoriented riders colliding with other pool guests. Lifeguards shall instruct Participants to exit the Designated Safety Area in the correct manner and direction.

An uninterrupted view of the pool and Obstacle Frame must be maintained at all times. It is recommended that all lifeguards be familiar with all the jobs related to the Obstacle Frame. Rotating lifeguards between positions keeps interest and attention high.

Section 2

Facility Requirements

Communications

Each facility shall ensure they have a communication plan in place for all staff working the NinjaCross MiniNinja System and have trained them in the proper use of signals, devices, or other methods.

Signage:

The owner/operator shall place signage as specified. These signs shall include safety, warning, and instructional signage reflecting manufacturer recommendations. Signage shall be prominently displayed at the course entrance or other appropriate area and shall include but not be limited to:

- **Instructions, which include:**
  - Expected participant conduct,
  - Dispatch procedures,
  - Exiting procedures, and
  - Obey attendant/lifeguard instructions.
- **Warnings, which include:**
  - NinjaCross MiniNinja characteristics, such as challenging & competitive
  - Water depth if not posted near pool edge already
- **Requirements which include:**
  - Participants being free of medical conditions, including but not limited to pregnancy and heart, back, or musculoskeletal problems,
  - Mental conditions that may prevent comprehension or adherence to posted rules,
  - Maximum/minimum height and weight, and
  - Any swimming or physical ability requirement or both.

Section 1

System Overview

Your Ninja*Cross* MiniNinja System is an indoor or outdoor system that includes the deck mounted anchor points and mounts. This section will give an overview of the different materials that make up the components of the system.

Stainless Steel Components

1. Bolting Hardware
2. Shackles

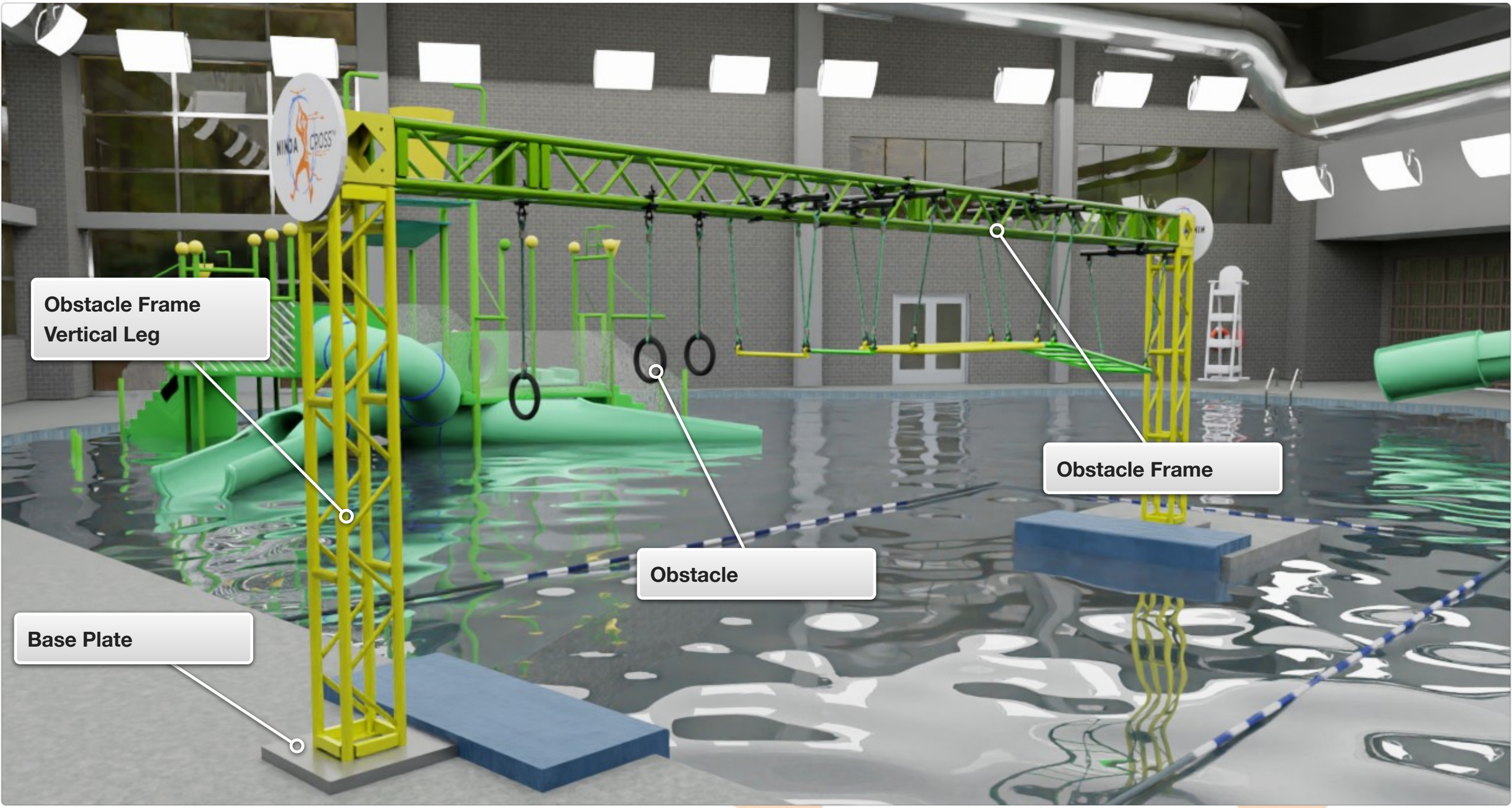
Aluminum Components

1. All metal Obstacles and OAB's
2. Obstacle Frame Truss and Corner Blocks
3. Truss Picks and Clamps

Other Materials

1. Signs - ABS
2. Backup System - powder-coated steel with galvanized cable
3. Ropes - InCord NetForm, Polyester Fiber Braided Steel Wire
4. Discs, Rings, and other Obstacles - HDPE

Interactive 8.1 System Overview



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Section 2

Obstacle Frame Components

The Obstacle Frame consists of 3 primary components

1. 12”x12” Box Truss

2. 12” 6-way Corner Blocks

3. Mounting Plate

The parts of the Obstacle Frame System include:

1. **12”x12” Box Truss -** this aluminum box truss comprises the main structural component of the Obstacle Frame. Each section is at maximum 10’ long with the shortest being 2’ long. The type of Box Truss used is a bolt plate type that utilizes 5/8” bolt hardware.

2. **12”x12” 6-Way Corner Block -**is a 12” square block used to connect sections of Box Truss. The block is the only point where Static Lines are permitted to be installed.

3. **Mounting Plate -** this is a square aluminum plate designed to allow anchorage of the MiniNinja system to the concrete deck. The Mounting Plate is secured to the deck via wedge anchors and secured to the vertical Box Truss legs via bolting hardware.

Gallery 8.1 Obstacle Truss System

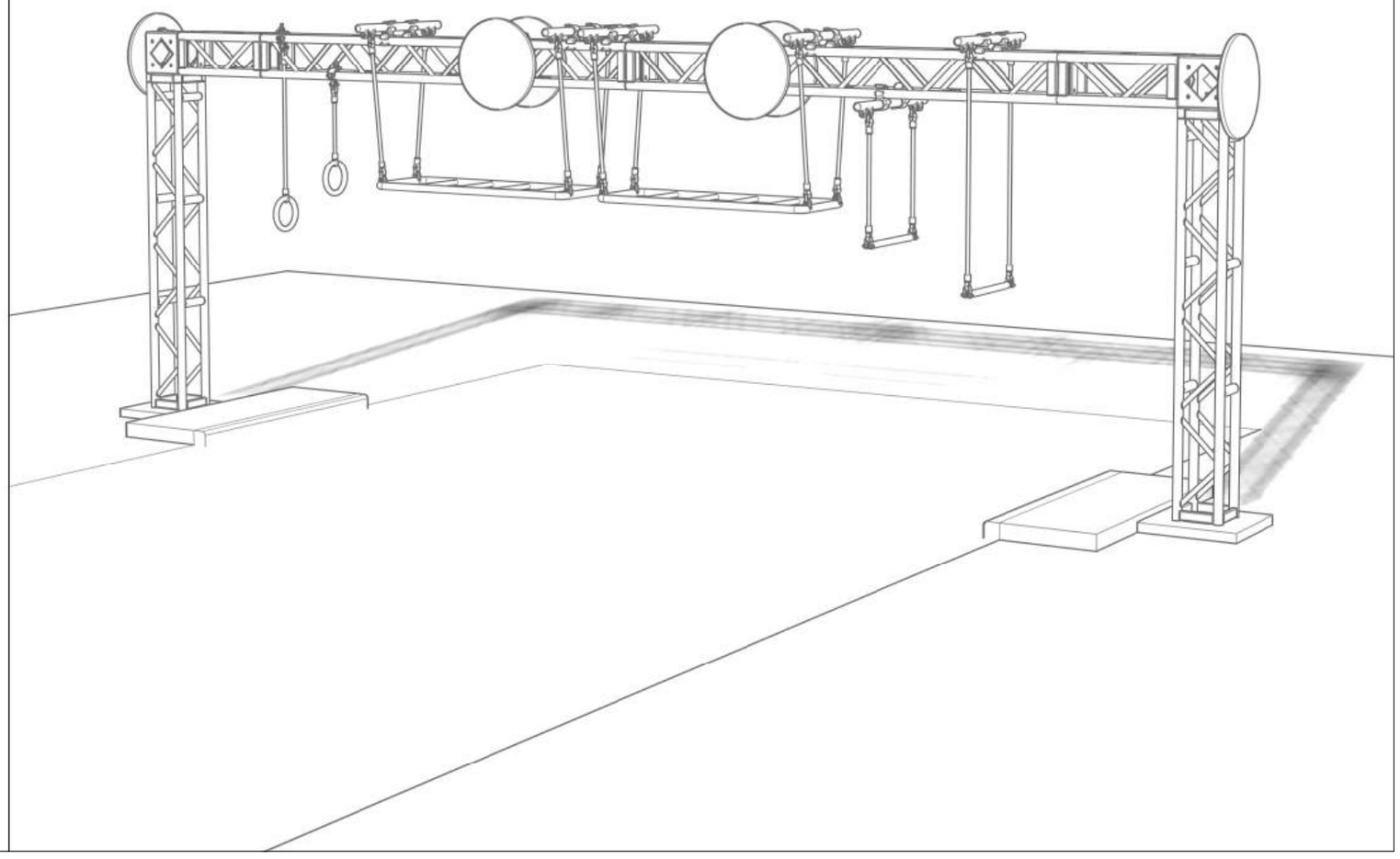
Corner Block

BASE PLATE AND ANCHOR NOTES:

FOR SLABS BETWEEN 4" AND 6" THICK:
INSTALL CUSTOM XSF BASE PLATE WITH (4) 5/8"Ø THREADED ROD ANCHORS IN AN 18" SQUARE PATTERN. EMBED 2¾" USING HILTI HIT-RE 500 V3 ADHESIVE.

FOR SLABS 6" THICK OR GREATER:
ANCHOR TRUSS DIRECTLY DOWN TO SLAB WITH (4) 5/8"Ø THREADED RODS AND HILTI HIT-RE 500 V3 ADHESIVE. USE A MINIMUM 4½" EMBEDMENT.

CONCRETE COMPRESSION STRENGTH SHALL BE 4000 PSI OR GREATER IN ALL CASES.



PRELIMINARY
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NINJA*CROSS*™
SYSTEMS

TITLE: Cover Sheet		
PROJECT: LilyPad Replacement		**COUNTRY:**
DATE: 5/13/21	**PROJECT #:**	**STATE:**
SCALE:	**DESIGNER:** SPW	**COUNTY:**
CHECKED BY:	**APPROVED BY:**	**CITY:**

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Comments/Notes
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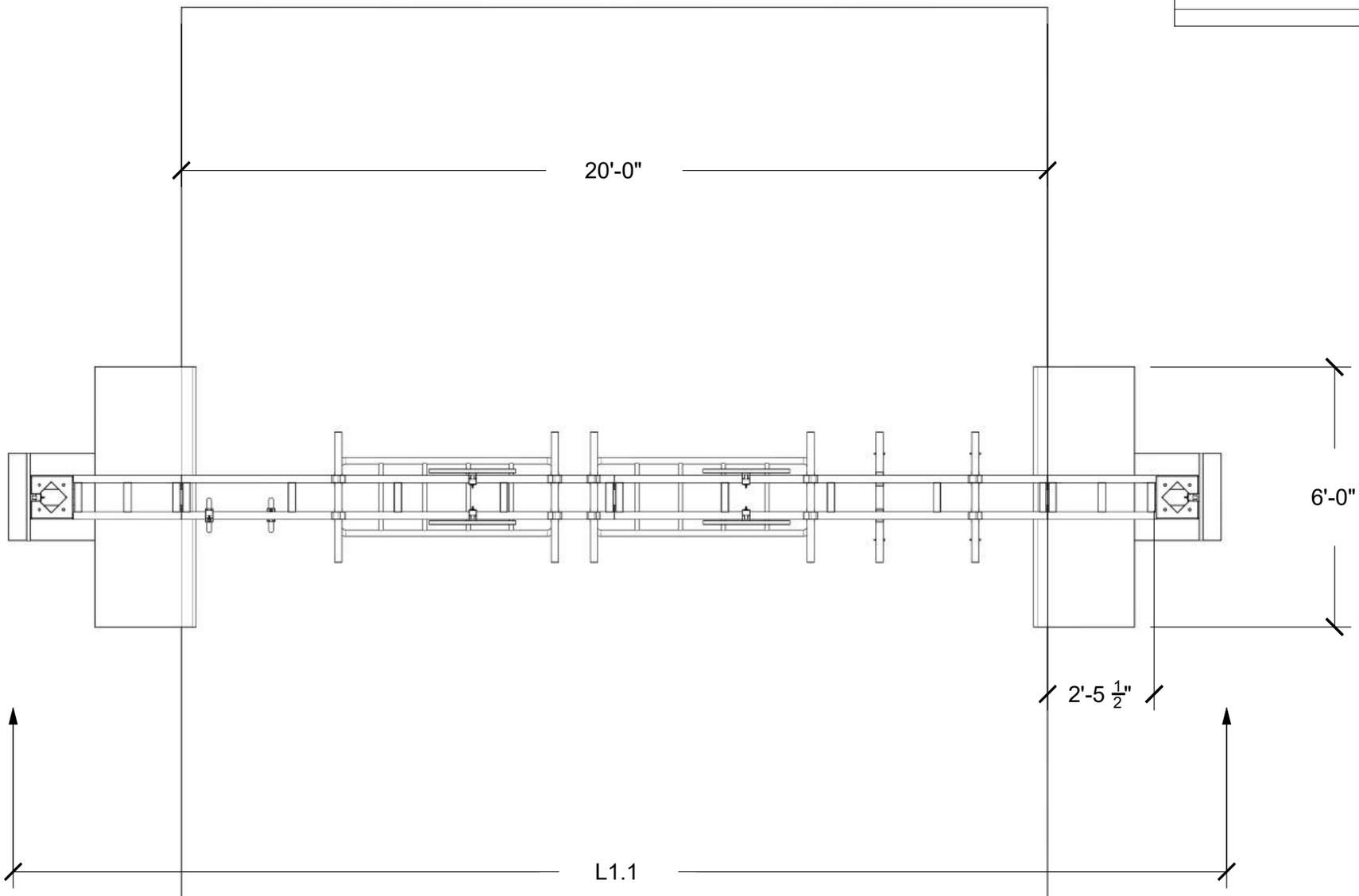

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NINJA*CROSS*™
SYSTEMS

TITLE: Layout		
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SCALE:	**DESIGNER:** SPW	**COUNTY:**
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Comments/Notes
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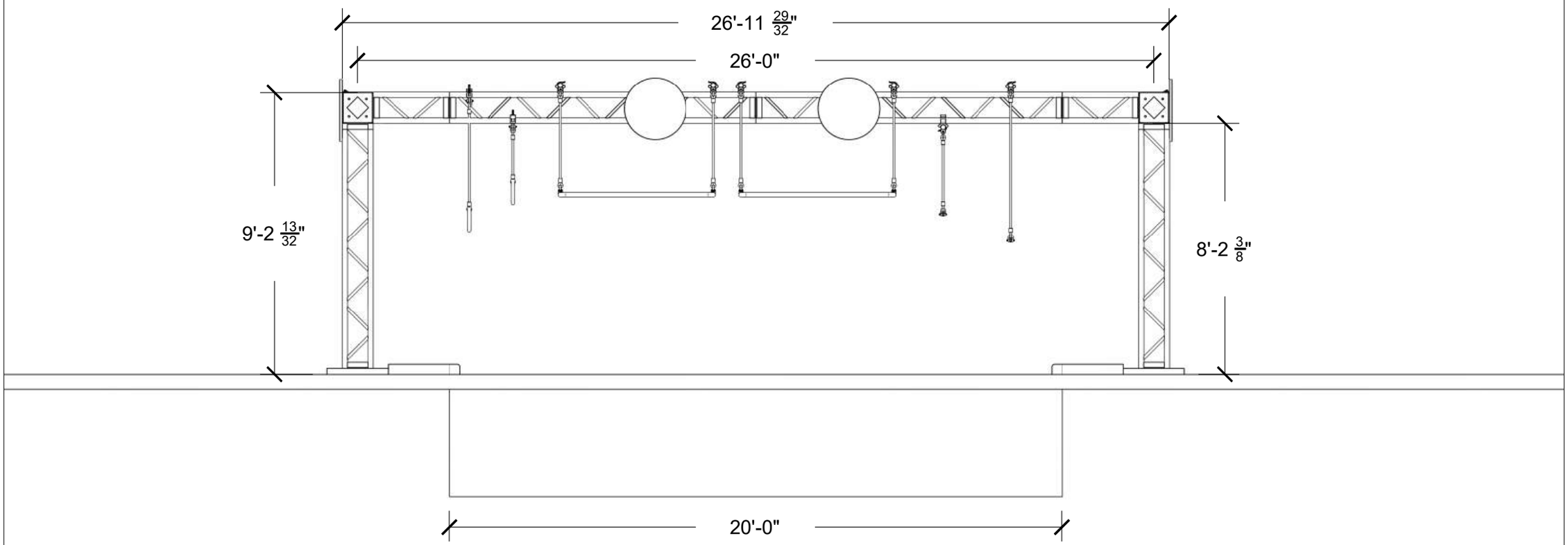

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SYSTEMS

THE STRUCTURAL SUPPORT SYSTEM DEPICTED ON THESE DRAWINGS WAS DESIGNED UNDER THE DIRECTION OF THE PROFESSIONAL ENGINEER WHO'S SEAL AND SIGNATURE ARE AFFIXED TO THIS DRAWING. THIS PROFESSIONAL ENGINEER DID NOT PERFORM ANY DESIGN OR REVIEW OF THE OTHER SYSTEMS SHOWN ON THESE DRAWINGS AND THEREFORE ASSUMES NO RESPONSIBILTY FOR THER ADEQUACY.

TITLE: Layout		
PROJECT: LilyPad Replacement		**COUNTRY:**
DATE: 5/13/21	**PROJECT #:**	**STATE:**
SCALE:	**DESIGNER:** SPW	**COUNTY:**
CHECKED BY:	**APPROVED BY:**	**CITY:**

NCS L1.1

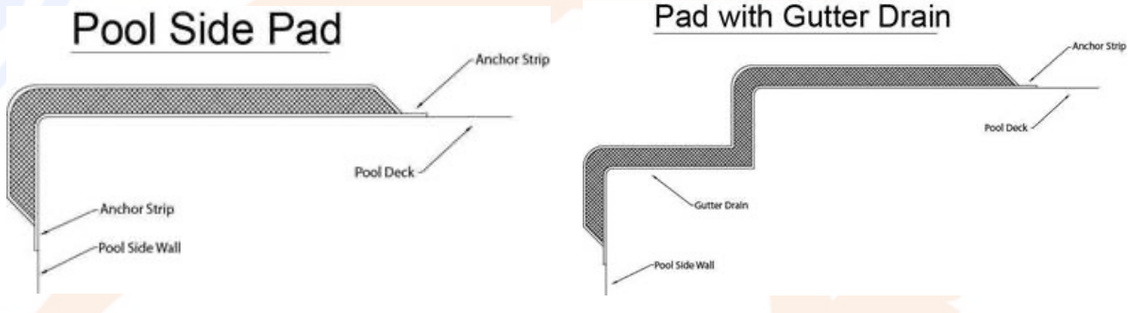
Disclaimers and Important Manufacturer Information

- **The NinjaCross MiniNinja System & ancillary components require installation by qualified personnel. Use of non-qualified trades' people or use of non-approved parts will void the manufacturer's Warranty.**

- **NinjaCross MiniNinja maintenance is the responsibility of the owner. It is recommended a maintenance log be kept documenting water quality including all performed maintenance. See suggested inspection check lists, water quality log, and maintenance section for guidelines on how to maintain the system, in addition to keeping your Warranty valid. These documents may be called on if warranty issues arise.**

- **When receiving manufacturer shipments, inspect all items for damage and quantity immediately. Failure to do so could result in costly repair or replacement costs at the expense of the owner/installer. When receiving any shipments, be sure to inform the driver of any discrepancies and report as indicated on the shipping documentation when signing for receipt of goods. All claims must be reported within 48 hours of receipt of goods. Claims reported outside of this time cannot be guaranteed. If nothing has been noted on the Bill of Lading a claim may not be accepted. If you are unable to inspect the shipment at time of receipt you must note on the Bill of Lading "Subject to inspection".**

- **NinjaCross Systems does not supply the Safety Padding. Safety Padding is the sole responsibility of the Owner/ Operator. Pool Side Pads are designed to be placed on the side of the pool to protect patrons as they enter and exit the MiniNinja area. Pads typically form an L-Shape covering the length of your area and protect the top walk area, the pool side wall and the pool edge. Pads can also be made in a "stair-step" shape to protect pool walls with drain gutters.**



NAC
ARCHITECTURE

Patty Hayes, Board Chair
Washington State Board of Health
PO Box 47990
Olympia, WA 98504-7990

AQUATIC CENTER at MLK JR. PARK, Yakima

Variance Letter Date: 2024.06.20

STATE IDENTIFICATION: State ID Facility #: F0476 Project #:2024003

Facility Information:
Aquatic Center at MLK Jr. Park (New outdoor pool facility with 5,300sf pool building and two leisure pools)
Plan Submittal: Drawing Plans have been submitted for review.

Aquatic Center at MLK Jr. Park, City of Yakima

Owner Contact:	Ken Wilkinson	Phone: 509-576-6416
Owner Address:	129 N 2nd street	Yakima, WA 98901
Facility Address:	610 S 9th Street	Yakima, WA 98901
Owner Representative:	Brooke Hanley (NAC Architecture) 509-838-8240	

Variance Request Contact:
NAC Architecture: Brooke Hanley Phone: 509-838-8240 Email: bhanley@nacarchitecture.com

Variance Request Citation:
WAC 246-262-160 states *the board may grant a variance from requirements of chapter **246-262** WAC if, in the sole discretion of the board, data and/or research provides sufficient evidence that the RWCF (attraction, device, equipment, procedure, etc.), will adequately protect public health and safety, as well as water quality.*

Variance Request: Code language related to Diving Envelope (WAC 246-262-010(21) & WAC 246-262-060(5)(vi)) for the **AquaZip'N Rope Swing** attraction.

Items noted in review letter include:

- **Aqua Zip'N Rope swing** attraction receiving pool shall conform to the CNCA or FINA standards (depth application and setbacks)

In the Department of Health review response letter issued by Justin Law dated May 22, 2024, Justin requests NAC Architecture (NAC) and WaterTechnology, Inc. (WTI) to address important concerns regarding public safety related to the receiving pool for the proposed **AquaZip'N Rope Swing** attraction in Pool B. The concern is to address the minimum depth of the pool to be compliant with the WAC 246-262-010(21) & WAC 246-262-060(5)(c)(vi) regarding diving envelopes for features where users enter the water from above the water surface.

On behalf of the City of Yakima; NAC & WTI respectfully requests your consideration of the current pool depth design at the rope swing for the future Aquatic Center at MLK Jr. Park. To support this request we provide the attached information, engineering exhibits, and following commentary:

- The review letter states that the "diving envelope" from WAC 246-262-010(21) applies to **all attractions** where users enter above pool water level and therefore requires the CNCA (enter less than 20" above the water surface) or FINA (enter 20" or greater above the water surface) water depths. We submit that the attached engineering calculations for the **AquaZip'N Rope Swing** product will demonstrate that the manufacturer's required water depths and the designed water depths provided at the Yakima Aquatic Center are more than sufficient to protect the safety of the users allowed to participate in this attraction. Calculations were completed for a 72" tall, 250lbs person, any body size smaller than the max would perform better, not worse. The manufacturer's minimum depth requirement is 4 feet. Although the current Yakima receiving pool water depth exceeds the manufacturer's recommendations, the applicant proposes to move the rope swing to the deeper water directly west to provide a consistent 6-foot deep zone for this attraction, in an effort to alleviate DOH concerns. The applicant proposes to remove the drop slide from the project and in its place locate the rope swing instead. Please review the attached data in support of using the manufacturer's depth requirements in lieu of the CNCA diving envelope dimensions.

- WAC 246-262-060(5)(c)(vi) appears to apply specifically to "diving envelopes in pools or areas of pools designated for diving activities". The applicant submits that diving activities are generally defined as plunging into the water headfirst. Diving headfirst into water results in the need for deeper water to avoid a head & neck collision with the pool floor which is different than a feet-first or tucked entry plunge where the body is significantly slowed in the first two feet of water. The **rope swing** safety guidelines (provided in the exhibits) will note that users are required to enter the water in a feet-first manner. Diving from the unit is prohibited. The engineering calculations completed also assumes a feet-first plummet into the water.

- The Model Aquatic Health Code also addresses the complexity of "other aquatic features" like this and would suggest that the manufacturer recommendations for design and operation would be adequate to install the feature.

  4.12.10[A] Other Aquatic Features Other AQUATIC FEATURES not otherwise addressed in the CODE, including but not limited to climbing walls, inflatables, and play structures, shall not be installed unless designed and operated in accordance with all manufacturer's installation and operations recommendations.

- 'A-frame' signs with all written safety guidelines will be publicly displayed near the rope swing (see page 8 for example) to meet the criteria of WAC 246-262-070(10). Participants will be screened by lifeguards to ensure they are within the minimum and maximum size requirements.

- See attached rope swing diagrams to understand how the hand holds are provided on the rope at even intervals between 57" and 87" above the deck. The relatively low height of the hand holds does not allow the users to gain much elevation above the water as they slide out over the surface.

- Safety padding rated for falls from 6ft or less are provided around the base of the rope swing structure and down the face of the pool wall to prevent injuries at the corner of the gutter. The rope swing itself has a safety catch, so when the user swings out over the water, they are prevented from sliding back toward the wall. Once the user drops into the pool, the rope self-retracts so the next user does not need to reach out over the water to grab the rope.

- This pool will be lifeguarded at all times while in operation and the lifeguard staff will be the first line of defense to screen bathers to make sure they are experienced swimmers, instruct swimmers on proper use of the attraction, and direct proper swimmer circulation to and from the activity within the pool to avoid congestion or collisions. The **rope swing** will have a dedicated lifeguard to closely supervise the safety of swimmers when the attraction is open for use.

- Injury statistics requested by the review letter are not available from the manufacturer or another source at this time.

- The **AquaZip'n** has also been designed and engineered to meet the following standards:
  - ASTM F2291-18 Amusement Rides and Devices
  - ASTM F2461-18 Aquatic Play Equipment
  - AISC Manual of Steel Construction
  - Other industry standards listed in the product data attached

- The City of Yakima specifically requested a pool design that would have a variety of intriguing activities for their patrons but would not need water deeper than 6-7ft. Pools deeper than 6-7ft come with their own safety risks and lifeguarding challenges. Shallow water is easier to supervise and guard. Rescues are much more likely to be needed in deep water where a bather in trouble cannot push off the bottom of the pool to bob back above the surface quickly until the lifeguard can assist them. Yakima is dedicated to making this facility fun while also as safe as possible for their community members and patrons.
-
- NAC submits that the design as described above and substantiated in the attached documentation meets the intent of providing a safe receiving pool for the **AquaZip'N Rope Swing** feature. NAC, WTI, and the City of Yakima respectfully requests a variance accordingly. If the State Board of Health has any follow-up conditions or actions required of the owner/operator, we are committed to implementing them.

NAC Architecture (NAC) has teamed with Water Technology (WTI) on numerous aquatic projects and so we have a history of producing these projects successfully. WTI has been designing Aquatic venues for over 40 years. WTI is widely known in the industry as one of the leading aquatic design firms in North America. As one of the industry's leaders, WTI has represented the waterpark industry during CPSC meetings on review of VGB rules and has also been involved in reviewing/editing sections of the MAHC. They are also represented in the Washington DOH committee to update the existing administrative code to adopt a more comprehensive aquatic code like the MAHC. The NAC and WTI commitment to safe aquatic facilities is proven. The design of the receiving pool at the **AquaZip'n Rope Swing** for the Yakima Aquatic Center will not put the health and safety of the public at risk. The City of Yakima, having operated a public pool for many years is experienced and committed to the safety and the welfare of their patrons. On behalf of the City of Yakima, NAC Architecture would like to thank you for your consideration of this Variance Request. Please feel free to contact me with any questions you may have regarding this request.

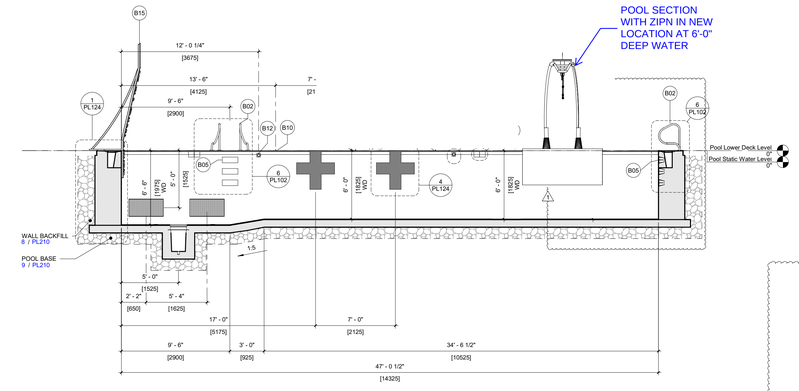
Thank you,

Brooke Hanley

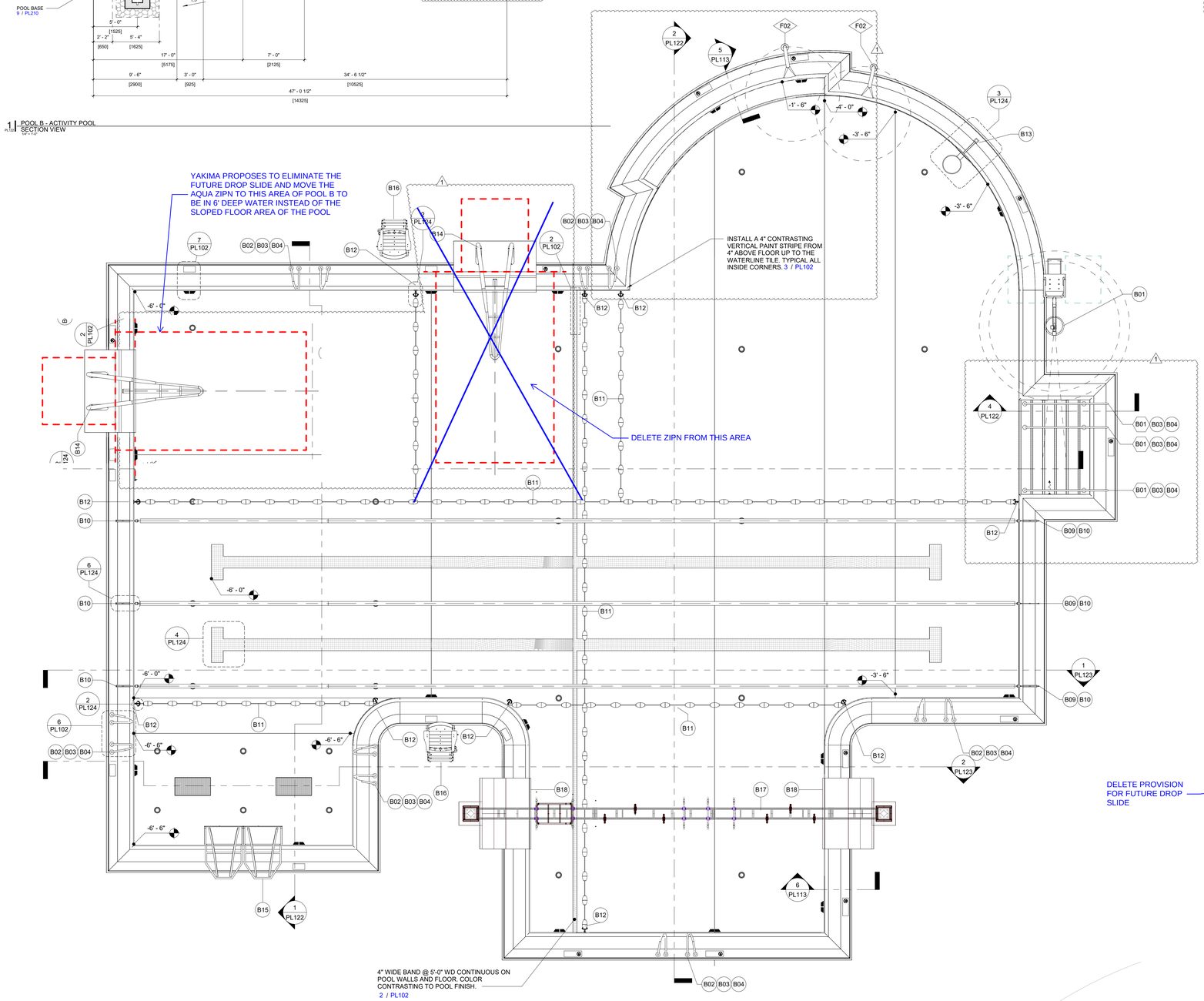
Brooke Hanley, AIA, Principal Architect, NAC Architecture

Attachments:

- AquaZip'n Safety Information and Fall Zone Engineering, including a floor plan and section of the receiving pool with proposed changes for the Yakima Aquatic Center.



1 | POOL B - ACTIVITY POOL SECTION VIEW
PL120 1/4" = 1'-0"



1 | POOL B - ACTIVITY POOL PLAN VIEW
PL120 1/4" = 1'-0"

POOL B-ACTIVITY DATA

DESCRIPTION	QTY	UNITS		
POOL PERIMETER	314'-0"	FEET		
WATER SURFACE AREA	3,832	SQUARE FEET		
POOL WATER TEMPERATURE	84	°F		
POOL VOLUME	136,514	GALLONS		
SURGE TANK OPERATING VOLUME	7,415	GALLONS		
TOTAL VOLUME OF WATER	147,268	GALLONS		
CIRCULATION RATE	1,033	GPM		
TURNOVER/VOLUME/FLOW	60 MIN.	19,330 GAL.	322	GPM
TURNOVER/VOLUME/FLOW	180 MIN.	127,938 GAL.	711	GPM
FILTRATION RATE	12.66	GPM/FT²		
BACKWASH FLOW	306	GPM		
SURGE FACTOR	1.06	GAL/SQFT		
AVAILABLE SURGE CAPACITY IN SURGE TANK	4075	GALLONS		

SCHEDULE - BASIS OF DESIGN - POOL B

POOL ID	EQUIPMENT ID	EQUIPMENT	QTY	MANUFACTURER	DESCRIPTION
B	01	POOL LIFT	1	SR SMITH, AQUA CREEK, OR EQUAL	STANDARD ANCHORED, ROTATIONAL POOL LIFT, WITH 400 LB MINIMUM LIFTING CAPACITY. MUST MEET ALL APPLICABLE ADA REQUIREMENTS, WHILE MAINTAINING REQUIRED DECK CLEARANCE. PACKAGE TO INCLUDE ARMRESTS, ANCHOR, LIFT COVER, BATTERY CHARGER, AND CADDY
B	02	GRAB RAILS (PAIRS)	6	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	PRETZEL BEND STYLE, 1.50" OD x .120 WALL THICKNESS, 500 GRIT FINISH MIN.
B	03	ESCUTCHEON PLATE	34	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	STAINLESS STEEL ROUND ESCUTCHEON FOR 1.50" O.D. RAILS
B	04	WEDGE ANCHOR	34	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CAST BRONZE, 4-1/4" LONG, ACCEPTS 1.500" OD TUBING
B	05	IN-WALL STEPS	18	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	17-1/2" x 6", INJECTION MOLDED PLASTIC, PEBBLE TEXTURE, 1/4" WALL THICKNESS
B	09	LANE DIVIDERS	3	COMPETITOR SWIM PRODUCTS	4" WAVE QUELLING RACING LANE LINE, COLORS BY OWNER / ARCHITECT
B	10	DaiFLEX LANE LINE ANCHOR	6	DALDORADO	12" – NON-CORROSIVE PVC FLIP UP LANE LINE ANCHOR TO BE USED WITH DALDORADO PARALLEL GRATING. INCLUDES FLIP-UP HATCH, BASE UNIT, & SILICON COVERED SS BRAIDED STRAP EXTENSION WITH HOOK. CAN BE USED WITH THE DaiFLEX 8" OR 14" LANE LINE EXTENSION.
B	11	SAFETY ROPE	6	PARAGON AQUATICS	3/4" POLYETHYLENE ROPE WITH 5"x9" HANDI-LOCK FLOAT, VERIFY LENGTH WITH PLANS
B	12	CUP ANCHOR	10	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	4" SQUARE 304L SS ANCHOR AND 304L SS EYE BOLT
B	13	BASKETBALL HOOP	1	SR SMITH	STAINLESS STEEL BASKETBALL HOOP WITH ROCKSOLID ANCHOR
B	14	AQUA ZIPN	1	AQUACLIMB	DECK-MOUNTED OVERHEAD ROPE SWING, WITH SELF-RETRACTING TROLLEY, POWDER-COATED STAINLESS STEEL WITH HIGH TENACITY POLYESTER ROPE. INCLUDES SAFETY PAD-UNIVERSAL, WITH 316 SS HILTI FLUSH MOUNT CONCRETE ANCHORS.
B	15	AQUACLIMB	1	AQUACLIMB	2 WIDE X 3 HIGH AQUATIC CLIMBING WALL
B	16	LIFEGUARD CHAIR	2	TAILWIND, KEIFER, SPECTRUM AQUATICS, SR SMITH OR APPROVED EQUAL	RECYCLED PLASTIC WITH 304 SS HARDWARE, COLOR BY OWNER/ARCHITECT 40" SEAT HEIGHT (OWNER'S SAFETY CONSULTANT TO SPECIFY LOCATION.)
B	17	NINJACROSS	1	NINJACROSS	AQUATIC OBSTACLE COURSE
B	18	SAFETY PAD	3	PLAYTIME	WALL AND DECK SAFETY PAD AT NINJACROSS SYSTEM

SCHEDULE - CUSTOM RAILGOODS - POOL B

POOL ID	EQUIPMENT ID	EQUIPMENT	QTY	MANUFACTURER	DESCRIPTION
B	01	HAND RAIL	3	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CUSTOM FABRICATED, 316L SS, 1.50" OD x .120 WALL THICKNESS, 500 GRIT FINISH MIN.
B	02	HAND RAIL	2	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CUSTOM FABRICATED, 316L SS, 1.50" OD x .120 WALL THICKNESS, 500 GRIT FINISH MIN.

SCHEDULE - WATER FEATURE - POOL B

DELETE PROVISION FOR FUTURE DROP SLIDE

POOL ID	FEATURE ID	FEATURE	QTY	MANUFACTURER	DESCRIPTION	GPM (ea)	GPM (Total)
~~B~~	~~F01~~	~~DROP SLIDE~~	~~1~~	~~SPLASHTAGULAR~~	~~FUTURE SLIDE. PROVIDE PIPING CAPPED ONLY~~	~~500~~	~~500~~
B	F02	WATER SPRAY	2	WATERPLAY	PIPE DELUGE-FAN SPRAY FEATURE	60	120

REVISIONS

REV.NO.	DESCRIPTION	DATE
1	CHANGE PROPOSAL	04/16/2024

CONFORMED SET

CITY OF YAKIMA
YAKIMA POOL
YAKIMA WA

WTI WATER TECHNOLOGY, INC.
World Leaders in Aquatic Planning, Design and Engineering
100 Park Avenue | Beaver Dam, WI 53916
t 920.887.7375

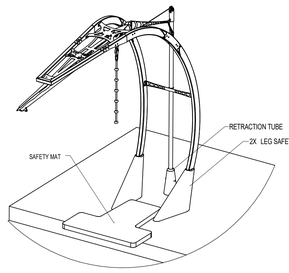
NAC ARCHITECTURE
nacarchitecture.com
1203 WEST RIVERSIDE AVENUE
SPOKANE WA 99201
P:509.838.8240

NAC NO 111-22082
ISSUE DATE: 4/16/24
PROJECT NUMBER: 22314
DRAWN BY: T.ED
CHECKED BY: ACC

7893 REGISTERED ARCHITECT MATTHEW W. FREEBY STATE OF WASHINGTON 4/16/2024

POOL B - ACTIVITY POOL PLAN

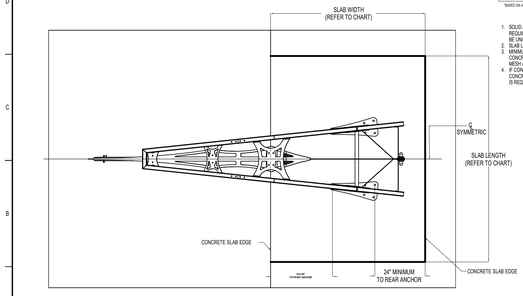
PL120



SAFETY GUIDELINES:

* EXPERIENCED SWIMMERS ONLY
* ONE PERSON USING THE ZIP'N AT A TIME
* ONLY ONE SWIMMER AT A TIME IN THE DROP ZONE
* NO DIVING AND NO BACKFLIPS; FEET FIRST ONLY
* MAXIMUM RIDER WEIGHT: PLEASE REFERENCE TABLE ON SHEET 3

SAFETY FEATURES

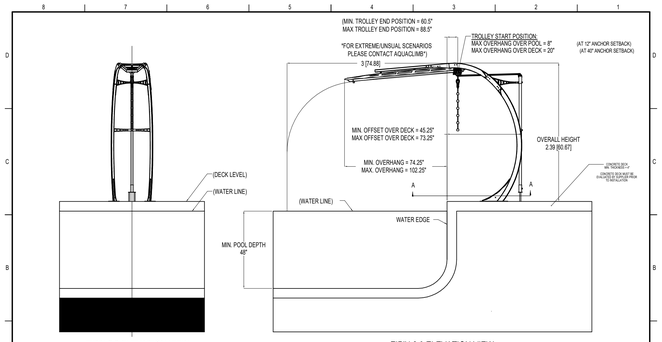


SLAB VS. RIDER HEIGHT DIMENSION REQUIREMENTS		
RIDER HEIGHT (lbs)	SLAB WIDTH MIN.	SLAB LENGTH MIN.
150	72"	72"
200	72"	84"
250	72"	96"

BASED ON A MIN. 4" THICK UNCRACKED, POURED CONCRETE SLAB

1. SOLID AREA OF UNCRACKED CONCRETE REQUIRED AT FRAME LOCATION. AREA SHALL BE UNINTERUPTED BY ANY JOINTS
2. SLAB LENGTH TO BE CENTERED ON ZIP'N FRAME
3. MINIMUM THICKNESS OF 4" REQUIRED FOR SLAB. SLAB TO BE WITH 6X6 W2.9 WELDED WIRE MESH AS R/F
4. 4" CONCRETE AREA IS NEW, MINIMUM CONCRETE STRENGTH OF 3000 PSI AT 28 DAYS IS REQUIRED

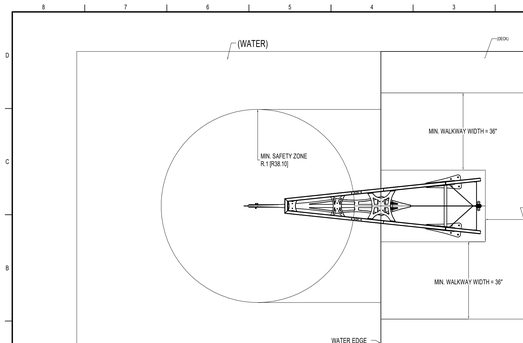
CONCRETE SLAB DETAILS



ZIP'N 2.0 ELEVATION VIEW
VIEWED LOOKING AT FRONT

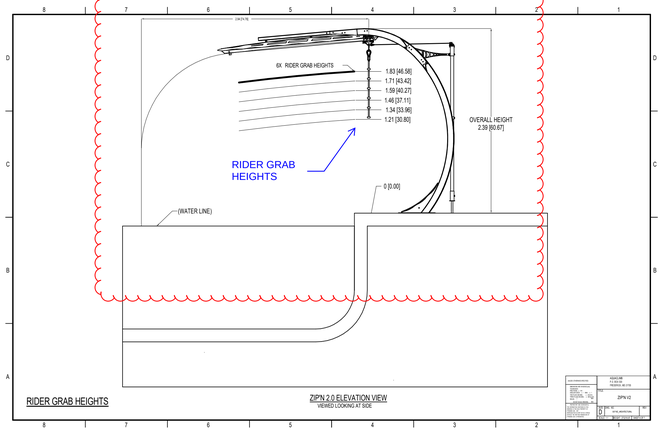
ZIP'N 2.0 ELEVATION VIEW
VIEWED LOOKING AT SIDE

SPATIAL REQUIREMENTS



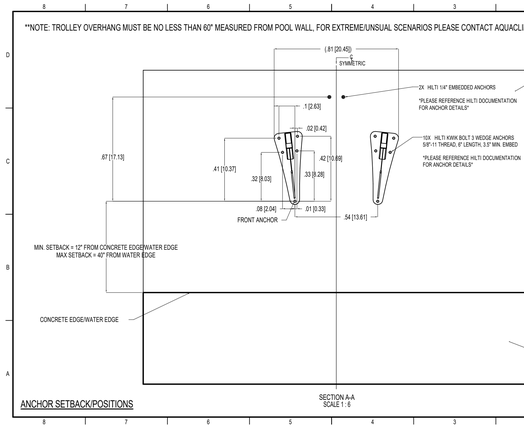
ZIP'N 2.0 PLAN VIEW
VIEWED FROM ABOVE

SAFETY ZONES



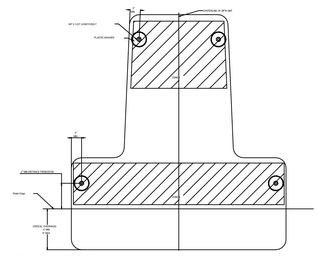
RIDER GRAB HEIGHTS

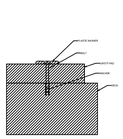
ZIP'N 2.0 ELEVATION VIEW
VIEWED LOOKING AT SIDE



ANCHOR SETBACK/POSITIONS

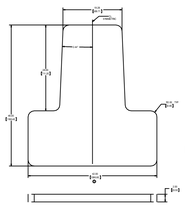
SECTION A-A
SCALE 1 : 6



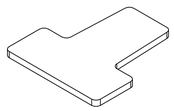


SAFETY PAD CROSS SECTION

NOTES
1. REMOVE ALL BURRS AND SHARP EDGES.
2. DIMENSIONS AND TOLDERANCE APPLY PRIOR TO FINISHING.
3. FINISH: TO BE SPECIFIED BY PROCUREMENT PROCESS

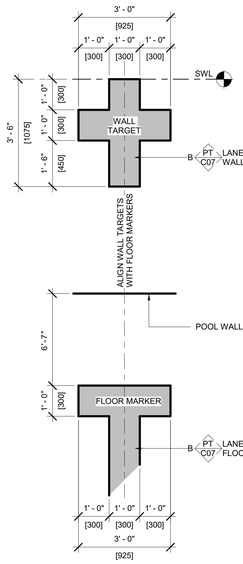


SAFETY PAD - UNIVERSAL



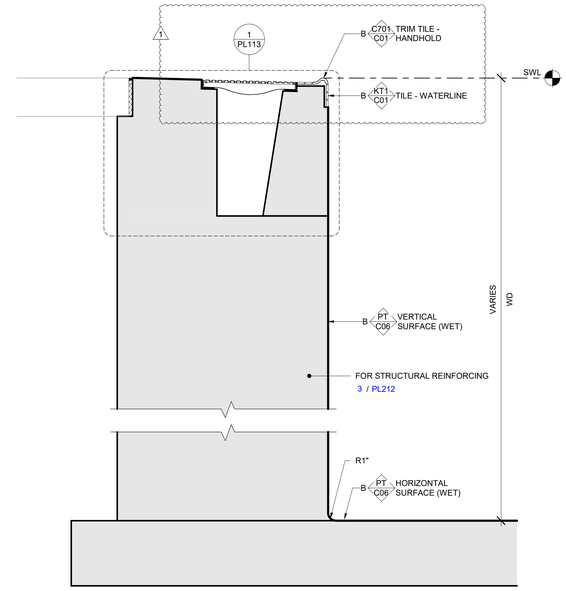
SAFETY PAD - UNIVERSAL

7 | AQUA ZIPN
PL124 | DETAIL VIEW
NOT TO SCALE

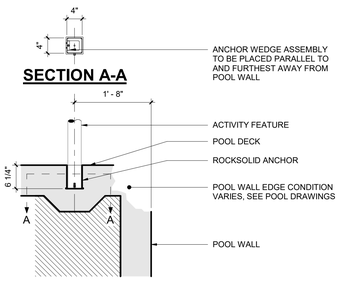


NOTES:
1. ALL PAINT SHALL BE IN A CONTRASTING COLOR TO POOL FINISH, BOTH FLOOR AND WALL. CONFIRM PAINT COLOR WITH OWNER/ARCHITECT

4 | FINISH - FLOOR MARKER AND WALL TARGET
PL124 | DETAIL VIEW
1/2" = 1'-0"



1 | POOL WALL - 5'-0" WATER DEPTH AND ABOVE
PL124 | DETAIL VIEW
1 1/2" = 1'-0"

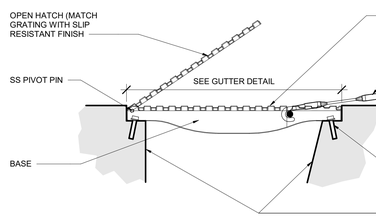


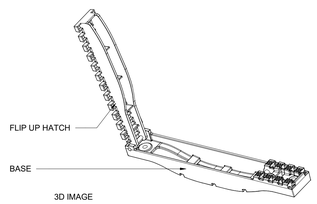
NOTES:
1. CONCRETE CLEAR COVER AT ALL ANCHOR EMBEDMENTS SHALL BE 6" MINIMUM. THICKEN SLAB IF NECESSARY TO ACHIEVE MINIMUM COVER.

5 | ANCHOR - ROCKSOLID INSTALL
PL124 | DETAIL VIEW
3/4" = 1'-0"

SECTION A-A FRONT VIEW

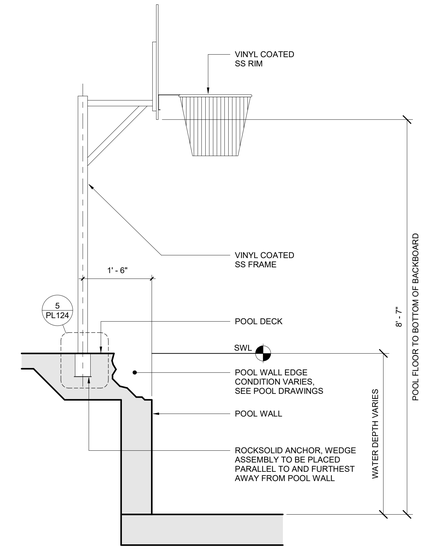
2 | ANCHOR - ROPE CUP
PL124 | DETAIL VIEW
3" = 1'-0"





3D IMAGE

6 | ANCHOR - FLIP ANCHOR
PL124 | DETAIL VIEW
3" = 1'-0"



NOTES:
1. CONCRETE CLEAR COVER AT ALL ANCHOR EMBEDMENTS SHALL BE 6" MINIMUM. THICKEN SLAB IF NECESSARY TO ACHIEVE MINIMUM COVER.

3 | ACTIVITIES - WATER BASKETBALL ANCHOR
PL124 | DETAIL VIEW
3/4" = 1'-0"

REVISIONS

REV.NO.	DESCRIPTION	DATE
1	CHANGE PROPOSAL	04/16/2024

CONFORMED SET

CITY OF YAKIMA
YAKIMA POOL
YAKIMA WA

WTI
WATER TECHNOLOGY, INC.
World Leaders in Aquatic Planning, Design and Engineering
100 Park Avenue | Beaver Dam, WI 53916
t 920.887.7375

NAC ARCHITECTURE
nacarchitecture.com
1203 WEST RIVERSIDE AVENUE
SPOKANE WA 99201
P:509.838.8240

NAC NO. 111-22082
ISSUE DATE: 4/16/24
PROJECT NUMBER: 22314
DRAWN BY: T.ED
CHECKED BY: ACC

7893 REGISTERED ARCHITECT MATTHEW W. FREEBY STATE OF WASHINGTON
4/16/2024

POOL B - ACTIVITY POOL DETAILS

PL124

AQUAZIP'N®

Combining the thrill of a zip line with the fun of a rope swing

With only 4 feet of depth required, AquaZip'N® can easily be added as an exciting poolside adventure at:

- Camps
- Country Club
- Colleges/Universities
- Swim Clubs
- Recreation/Aquatic Facilities
- Health/Fitness Centers
- Military Wellness & Recreation
- Private Residences

POOLSIDE ADVENTURES™

PoolsideAdventures.com
800.956.6692
info@poolsideadventures.com

AquaZip'N®: A UNIQUE Poolside Adventure

With nothing like it on the market, AquaZip'N delivers poolside fun and excitement in a fresh new way. With this easy addition to your pool, you will drive demand from guests of all ages and increase your facility's programming capabilities on top of these benefits:

High Throughput

Launching riders into the water quickly, AquaZip'N keeps the line moving with a proprietary self-retracting trolley so kids can experience it again and again.

Position Anywhere

With a minimum water depth requirement of 4 feet, AquaZip'N can be added easily for thrilling poolside adventures in the shallow or deep end.

Minimal Footprint

AquaZip'N requires little deck space with its sleek frame that hangs out over the water and doesn't interfere with normal lap swimming. And with no water source required, it is an easy amenity to add.

Activates the Deep End

As a safer alternative or enhancement to diving boards, AquaZip'N attracts tweens and teens to those under-utilized, deep areas of a pool.

Easy to Install

The AquaZip'N 3-piece system comes pre-fabricated for quick assembly and installation at your facility on any pool gutter configuration.

100% Made in America

AquaZip'N is designed, engineered and manufactured in the USA to conform to all industry standards.

To learn how you can bring the adventure of AquaZip'N® to your facility, contact us today:

POOLSIDE ADVENTURES™

PoolsideAdventures.com | 800.956.6692 | info@PoolsideAdventures.com

Building Courageous Kids for Life's Great Adventure

AQUAZIP'N® SPECIFICATIONS

System Description

Deck mounted, overhead self-retracting pool rope swing. Components consist of Steel support structure, self retracting trolley system with handline. Manufactured off site. Designed to withstand chlorinated environments.

Components

Rope System

Rope system consists of a ⅝", 3-Strand Twisted, High Tenacity Polyester, Plied Yarn. High tenacity for durability, low stretch, superior UV resistance, excellent resistance to acids/chlorines. Attached to the Trolley using high density plastic connector and 3" stainless steel carabiner. See manufacturer's full specification for details.

Support Frame

The support frame shall be fabricated of 304 stainless steel sections powder coated in Glacier White, consisting of multiple bolt-together assemblies. The Frame height is 115" and maximum width of 39" with an overall length of 147" from back of structure to end of track.

Anchors

Anchors are to include either Hilti Chemical Anchors using Hilti HIT-HY 200 Adhesive—⅝" diameter or HAS-R stainless steel wedge anchor (or approved equivalent) with a 3-⅛" minimum embedment, (5qty anchors) per leg. Install anchors per manufacturer instruction.

Fasteners

All fixed connections: Bolts, Flat Washers, Nuts, are attached by grade 18-8 stainless steel or higher. Anchors will be 18-8 Stainless Steel or higher grade.

Trolley Cable Retraction Assembly

3/16" Dyneema 12-strand Cable

Warranty

AquaZip'N® is warrantied to the original purchaser to be free from defects in material and workmanship from the date of installation, during normal use and installation, with exclusions of cosmetic defects through wear and tear: Limited 2-Year Warranty

Design Recommendations

Deck & Gutter

The pool deck in the AquaZip'N® installation area should be as level as possible. If the pool has a coping greater than 1-½", or does not meet the standard base concrete requirements below, additional hardware components may be required. Please complete the Poolside Adventures™ Gutter Configuration Worksheet available on our website and contact a Poolside Adventures™ representative to determine the proper installation hardware and anchoring required.

Concrete Requirements

Standard length anchoring system requires a minimum concrete depth of 4" (with 6x6 W2.0 welded wire mesh ASTM A185) with 3000 psi rating or greater, embedded to a minimum depth of 3-⅛". See Hilti anchor requirements for further details. Further concrete requirements for proper installation includes a 4" thick, 6' wide (away from pool edge) of uninterrupted, un-cracked concrete slab section. Length (parallel with pool edge) of concrete slab can vary based on desired maximum rider weight:

- 8' long for 250 lbs rider load rating
- 7' long for 200 lbs rider load rating
- 6' long for 150 lbs rider load rating

Clearances & Safety Recommendations

Please contact a Poolside Adventures™ representative for current product information regarding pool depth and clearance zone recommendations based on the deck and configuration to be installed.

State certified engineered drawings and/or drawings specific to actual site installation details may be required for approval of AquaZip'N® installation. Standard structural engineering drawings are available at no charge. State or site-specific engineered drawings may be an additional cost. Please contact the appropriate local governing department for more information.

Poolside Adventures™ product guides, installation instructions, owner's maintenance guide and other resources are available at www.poolsideadventures.com or can be requested by calling 800-956-6692.

Operations Manual AquaZip'N

The new AquaZip'N design allows for minimal maintenance and high throughput. The following is the inspection checklist.

Daily Checklist:

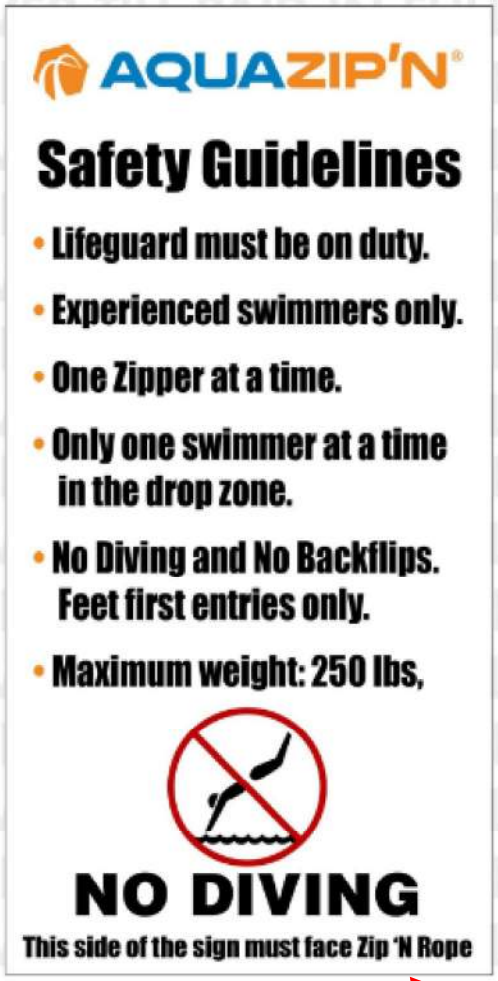
- Ensure proper trolley retraction by rolling trolley out over water, letting go and watching to see that trolley returns to original starting location.
- Check trolley wheels and bearings visually to ensure trolley is secure within its track.
- Visibly check retraction cable for wear & tear.
- Cable stretch is normal. However, if you notice the weight is contacting the bottom of the baseplate it is time to replace your retraction cable. Call Poolside Adventures at 800-956-6692 to order a replacement.
- Visibly check the rubber bumpers on the front and back of the track to ensure they are firmly in place and there is no visible cracking or imperfections.
- Spray silicone-based lubricant onto all wheel bearings to increase the smoothness and longevity of your trolley system.

Monthly Checklist:

- Inspect trolley to ensure secure attachments of retraction cable to trolley.
- Inspect hand rope for wear & tear.
- Inspect rubber bumpers on the front and back of the track for any cracks or imperfections. If any are found, please call Poolside Adventures at 800-959-6692 to order replacements.
- Check retraction cable for wear & tear.
    - Cable stretch and wear is normal. If you notice any significant wear on your retraction cable or if the weight is contacting the bottom of the baseplate when in operation it is time to replace your retraction cable. Call Poolside Adventures at 800-956-6692 to order a replacement.
- Check all bolts on the AquaZip'N structure to ensure they are firm & tight.
- Be sure acorn nuts are firmly secure on all threads able to be reached from the ground.
- Anchor bolts shall be taught to specifications.
- Inspect safety pad for visible signs of wear including cracks and gouges.

Seasonal/Annual Checklist:

- Remove trolley from track to complete thorough trolley inspection, ensuring all bolts are firm and all wheels and bearings are in good shape.
- Over time the wheels and bearings will need to be replaced. Call Poolside Adventures at 800-956-6692 to order replacement wheels.
- Store trolley indoors, in a cool dry location, during the off-season.
- Inspect concrete surface for cracking and weathering to which the PSI of concrete could become compromised.



"A" FRAME SIGN TO BE DISPLAYED AT ALL TIME THE AQUAZIP'N IS IN USE

Calculation Report

Hand Calculation on Projectile Analysis & Forces on the user

FEAmax LLC. Engineering Design, Analysis & Manufacturing Services.
Email: info@FEAmax.com; *Web*: www.FEAmax.com

5/3/2024

PROJECT INFO.

Change History:

Version Number	Date	Prepared by	Reviewed by	Contact
V 1.0	5/3/2024	Bill Bin	Frank Wang	Frank.Wang@feamax.com

CFD Requestor Info.:

Contact name:	Alex Salzman
Email:	Alex@PoolsideAdventures.com
Company name:	PYRAMIDE USA INC.
Address:	PO Box 530. Frederick, MD 21705

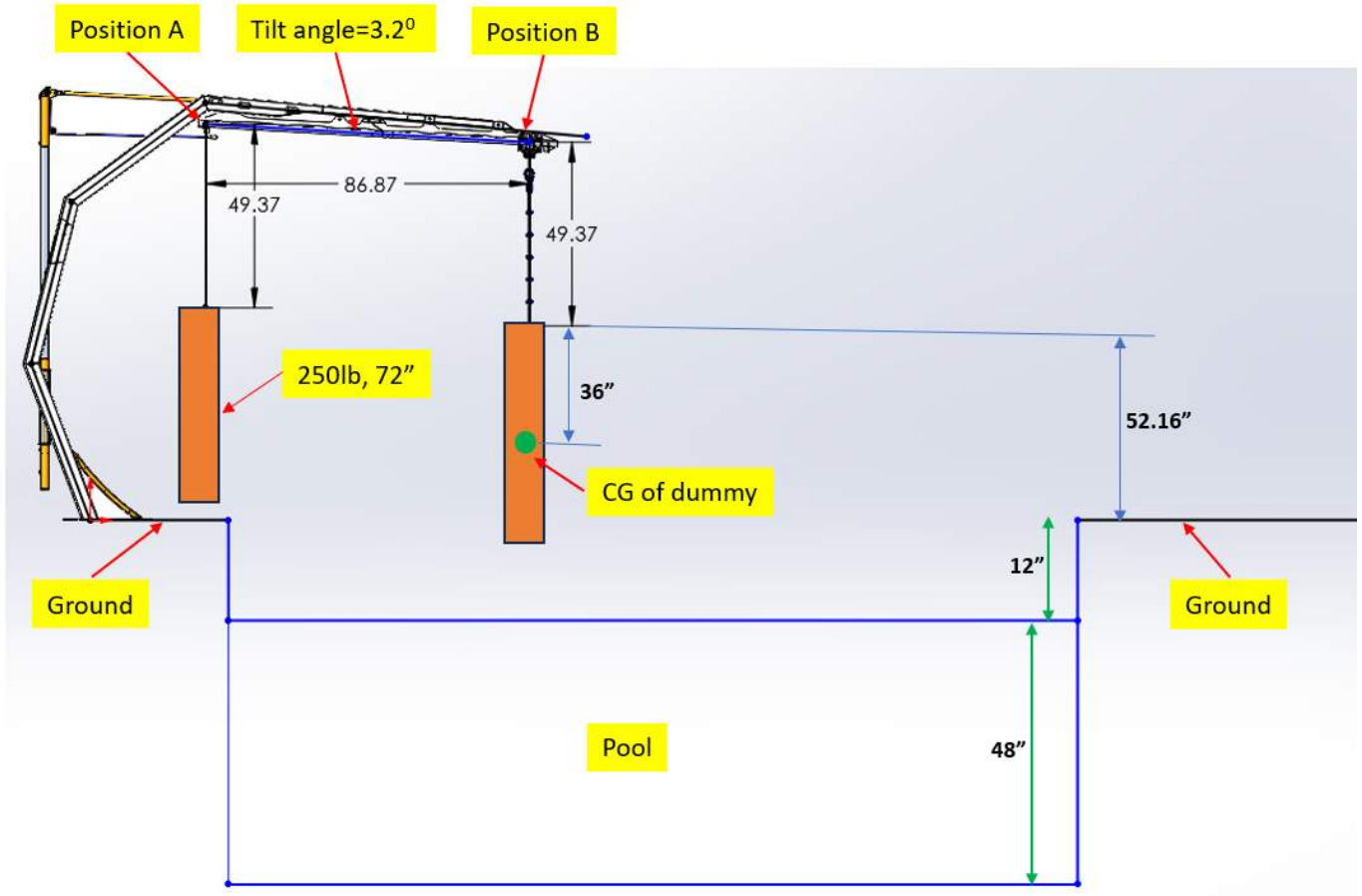
PROJECT DESCRIPTION

Project Description:

1. Perform hand calculations on the trolley system with the two cases.
2. The case #1 - Projectile Analysis: determine how far and how deep could a user go when launching from starting heights.
3. The case #2 - Forces on the user: determine the force on the user at beginning of ride and the end of ride.
4. The CAD model file for the calculation:
   - *Z0037C_V3.2 Master Assembly.SLDASM*
5. All related documents were received by 4/1/2024

CAD Model

1. The CAD model and the dimension information for calculation:



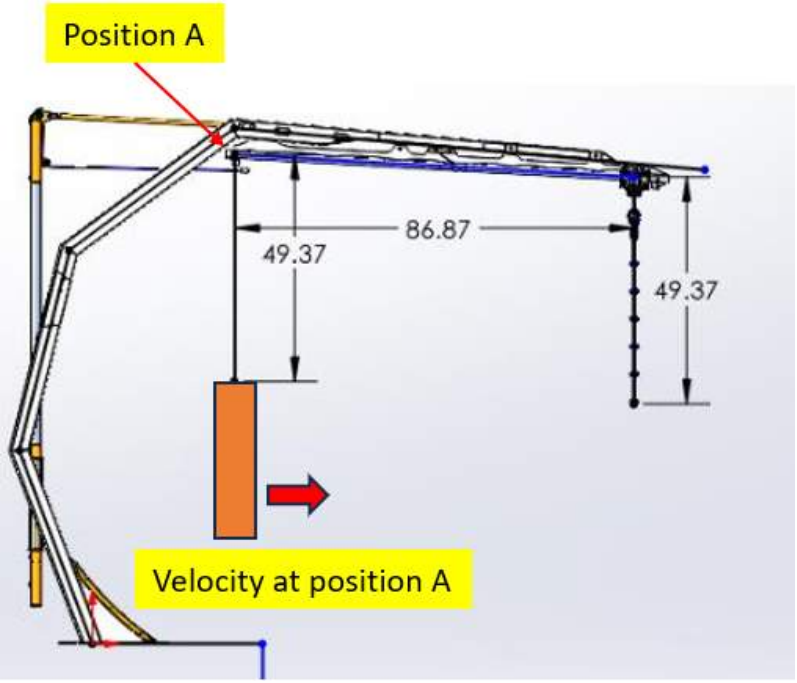
ASSUMPTIONS

Assumptions:

1. Assume a block/dummy on the rope with 250lbs mass and 6 feet height.
2. Assume the max jump forward distance is about 9.8 feet for a 250lbs adult from a standstill (worst case).
3. Considering the ideal condition, the person jumps at 45 degrees.
4. Assume it is frictionless contact at the top track rail.
5. Assume the 6 feet height dummy as a mass point at the CG (center of gravity).

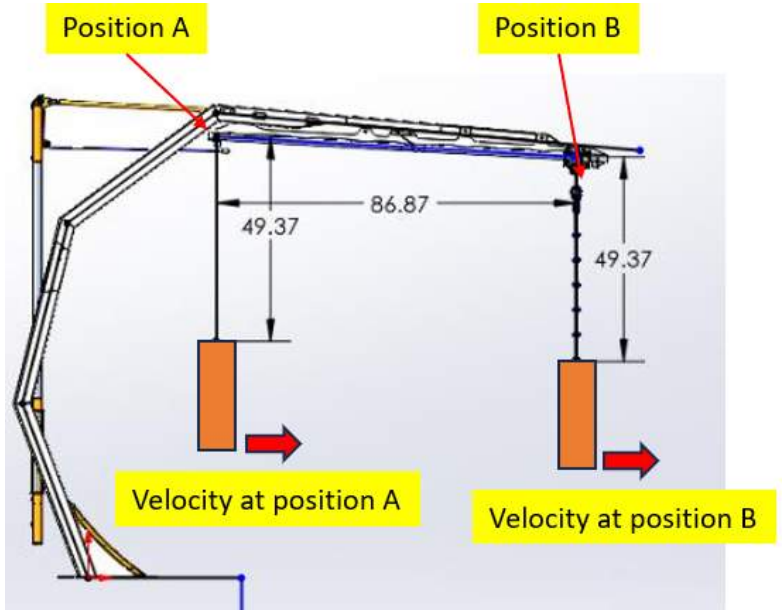
Calculation of initial velocity

1. Equations:
   - V x T = L
   - V = g x t / 2
   - In which: V is velocity, T is time, L is the length and g is the acceleration.
2. We have V = sqrt(L x g / 2), in which: L= 9.8 ft, g = 32 ft/s2
3. The calculated results:
   - The initial velocity at position A = sqrt(L x g / 2) = 12.56 ft/s



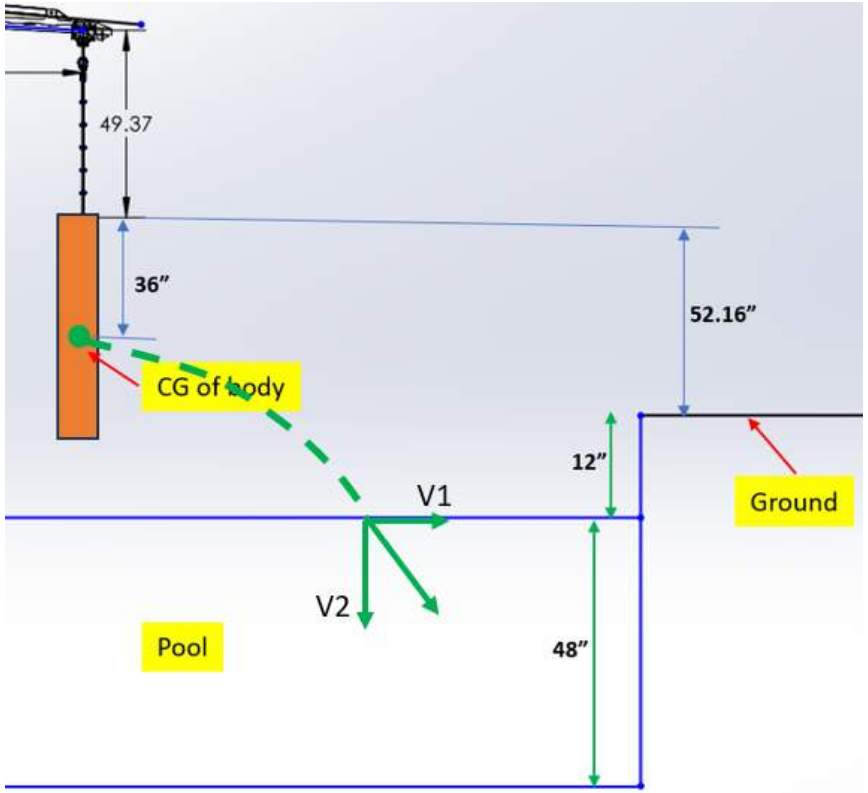
Item#1 – Projectile Analysis

1. Calculation#1 –velocity at position B:
   - Because of the frictionless contact and the tilt angle is only about 3 degrees between position A and B, we could assume the velocity at position B is the same as or very close to position A.
   - The velocity at position B = 12.56 ft/s
2. Calculation#2 – the moving distance before touch the water:
   - The initial horizontal speed V = 12.56 ft/s
   - The height above water (from CG of body to water) = 52.16+12-36 = 28.16 inch
   - The time before touch water t = sqrt(2L /g) = sqrt(2x28.16/32.15) = 0.38 s
   - The vertical velocity V2 = g x t = 12.33 ft/s
   - The horizontal velocity V1 = 12.57 ft/s
   - The moving distance before touch the water L = V1 x t = 4.75 ft



Item#1 – Projectile Analysis

3. Calculation#3 – the moving depth and distance in the water:
   - Equation: $F_d = 1/2 \cdot Cd \cdot \rho \cdot A \cdot v^2$
   - where:
   - F_d is the drag force, C_d is the drag coefficient, ρ is the density of the fluid (water is approximately 1000 kg/m3), A is the cross-sectional area of the object perpendicular to the flow of fluid, v is the velocity of the object relative to the fluid.
   - The drag coefficient (C_d) and the cross-sectional area (A) depend on the shape and orientation of the human body in the water. We'll need to make assumptions to proceed.



Item#1 – Projectile Analysis

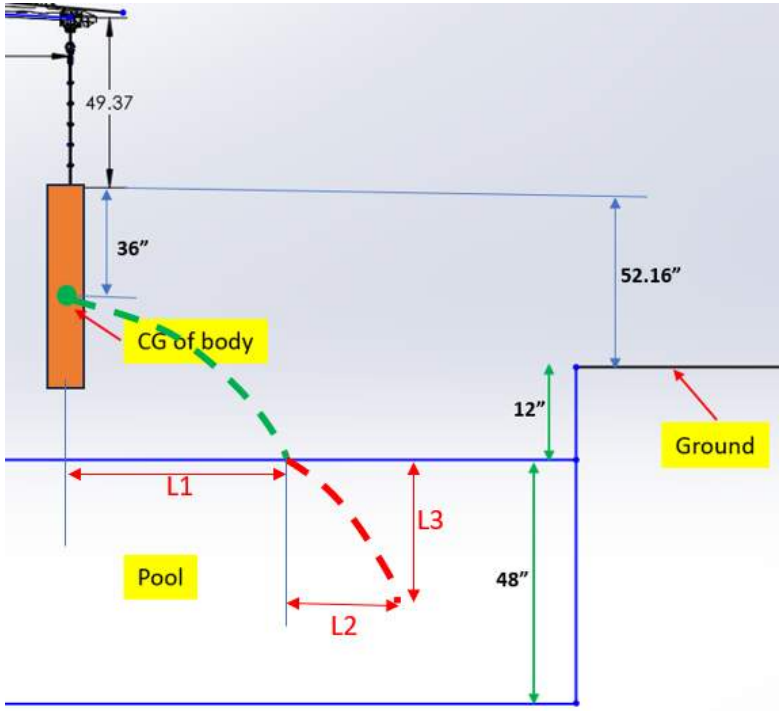
4. Calculation#4 – the moving depth and distance in the water:

- The depth and horizontal distance can be determined by integrating the motion equations under the influence of gravity and drag. However, the actual calculations can be very complex due to the non-linear drag force that depends on the velocity squared.
- Assume a constant average drag coefficient and ignoring buoyancy for the depth calculation, we can estimate the maximum depth and horizontal distance.
- Assume Cd=1.0 for a body position that is neither perfectly streamlined nor fully perpendicular to the flow. Assume cross-section area A=0.1 m^2, which is a rough estimate for a human body.
- Calculate the maximum depth and horizontal distance by considering the initial kinetic energy and the work done against the drag force. Distance = $\int_{V_i}^{0} \frac{1}{0.5Cd\rho Av} dv$ where v_i is the initial speed in the respective direction.
- The calculated maximum depth and horizontal distance the human can reach in water are approximately 0.84 meters.
- Note: these results are highly simplified. The actual values could differ significantly due to various factors such as the complex nature of drag in fluids, body orientation, and body shape effects.

Item#1 – Projectile Analysis

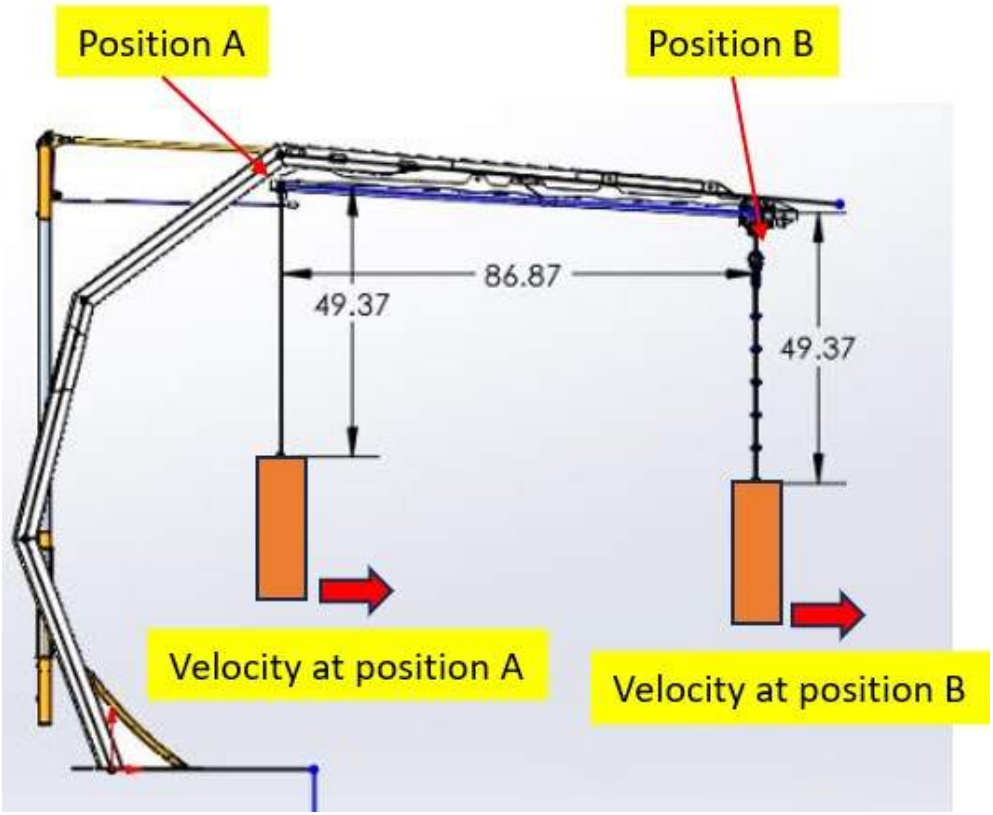
5. Calculation Results:

- Before touching the water, the body can move in horizontal direction L1 = 4.75 ft
- The max moving distance in horizontal direction in the water is about L2= 2.76 ft.
- The max depth in the water is about L3 = 2.76 ft.
- Note: if counting the body heigh 6ft, the max depth in the water would be 5.76 ft.



Item#2 – Forces on the user:

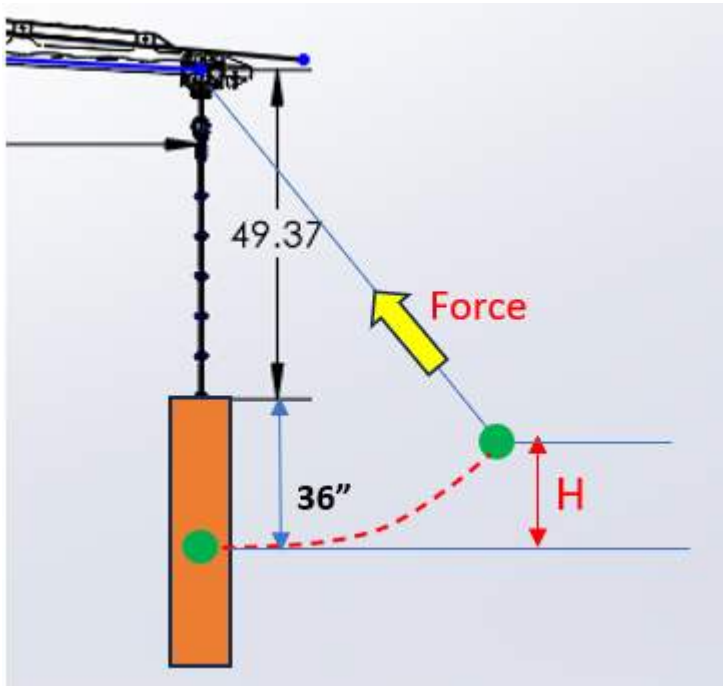
1. Calculation#1 – the max holding force on the user at position A:
   - Assume the body moves in horizontal direction, the initial holding force in vertical direction would be the same as the weight of user.
   - So, the max force on the user from rope at the beginning of ride (position A) is about 250 lbf.



Item#2 – Forces on the user:

2. Calculation#2 – the max holding force on the user at position B:

- Assume the user would hold the rope without release.
- The body would swing and cause higher force on the rope.
- Max force T_{max} = m x g + m x v^2 / r = 422 Lbf.
- The user swing height is about H = V^2 / 2g = 2.43 ft



3. Results:

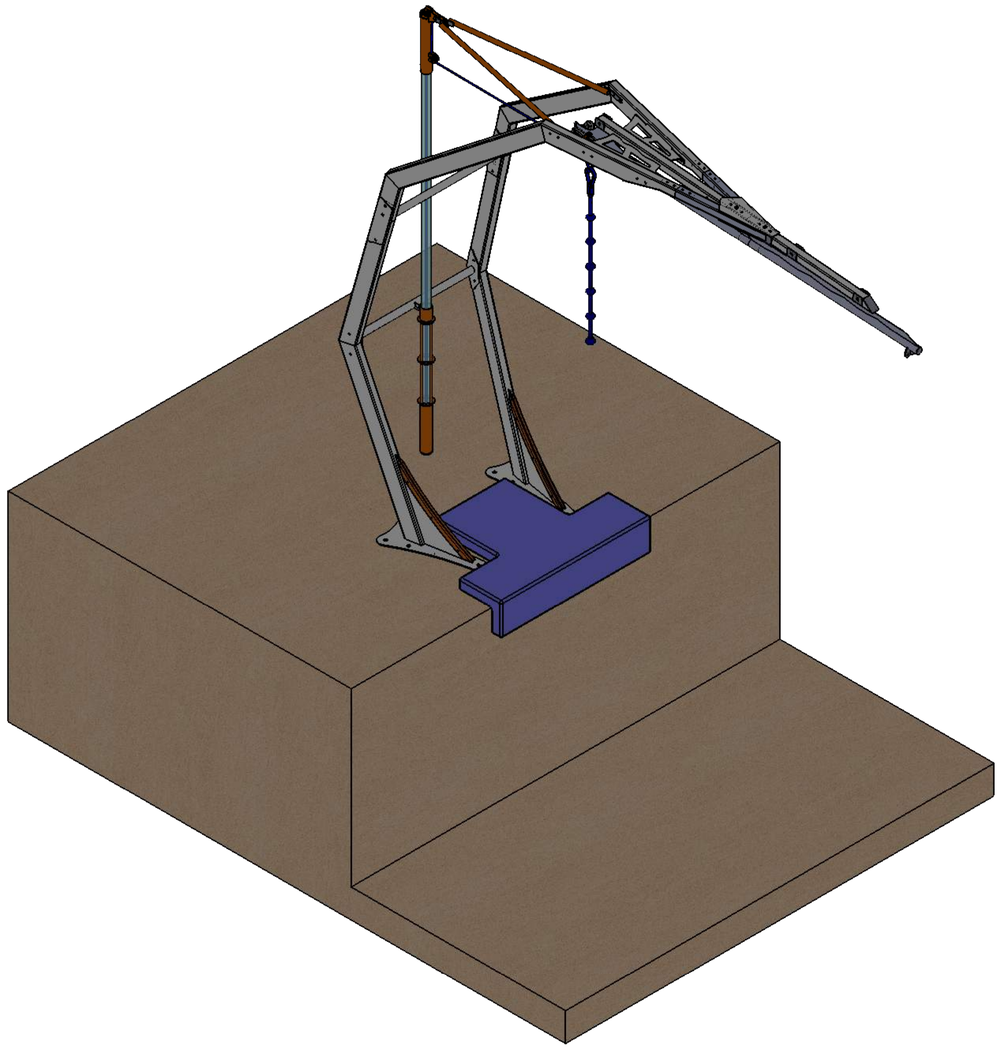
- The max force on the user (holding force on hands) from rope at the beginning of ride (position A) is about 250 Lbf.
- The max force on the user (holding force on hands) from rope at the end of ride (position B) is about 422 Lbf.
- The user can swing upward max height is about 2.43 ft.

REVISIONS			
REV.	DESCRIPTION	DATE	REV'D BY
A	Initial Release	7/27/2023	A. Salzman

Designed and engineered to the following standards:

- ASTM F2291-18 Amusement Rides and Devices
- ASTM F2461-18 Aquatic Play Equipment
- International Building Code (IBC) 2015 and ASCE 7, Minimum Design Loads for Building and Other Structures
- AISC Manual of Steel Contruction, 13th Edition
- ASD and Steel Design Guide 27 - Structural Stainless Steel

***Full structural analysis and stamped fabrication drawings available upon request



AquaZip'n V3.1 Architectural Guide

DESIGN

POOLSIDE ADVENTURES™

P.O. BOX 530
Frederick, MD 21705

PHONE: +1 800.956.6692
FAX: +1 240.575.6020
EMAIL: info@poolsideadventures.com

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Poolside Adventures
P.O. BOX 530
FREDERICK, MD 21705

UNLESS OTHERWISE SPECIFIED:
DIMENSIONS ARE IN INCHES [mm]
TOLERANCES:
FRACTIONAL ± 1/16
ANGULAR: MACH ± 1 BEND ± 1
TWO PLACE DECIMAL ± .03 [0.76]
THREE PLACE DECIMAL ± .005 [0.127]
DO NOT SCALE DRAWING

	NAME	DATE
DESIGNED		
DRAWN		
CHECKED		
ENGINEERED		

COMMENTS:
-NONE-

PROJECT:

TITLE:

SIZE	DWG. NO.	REV
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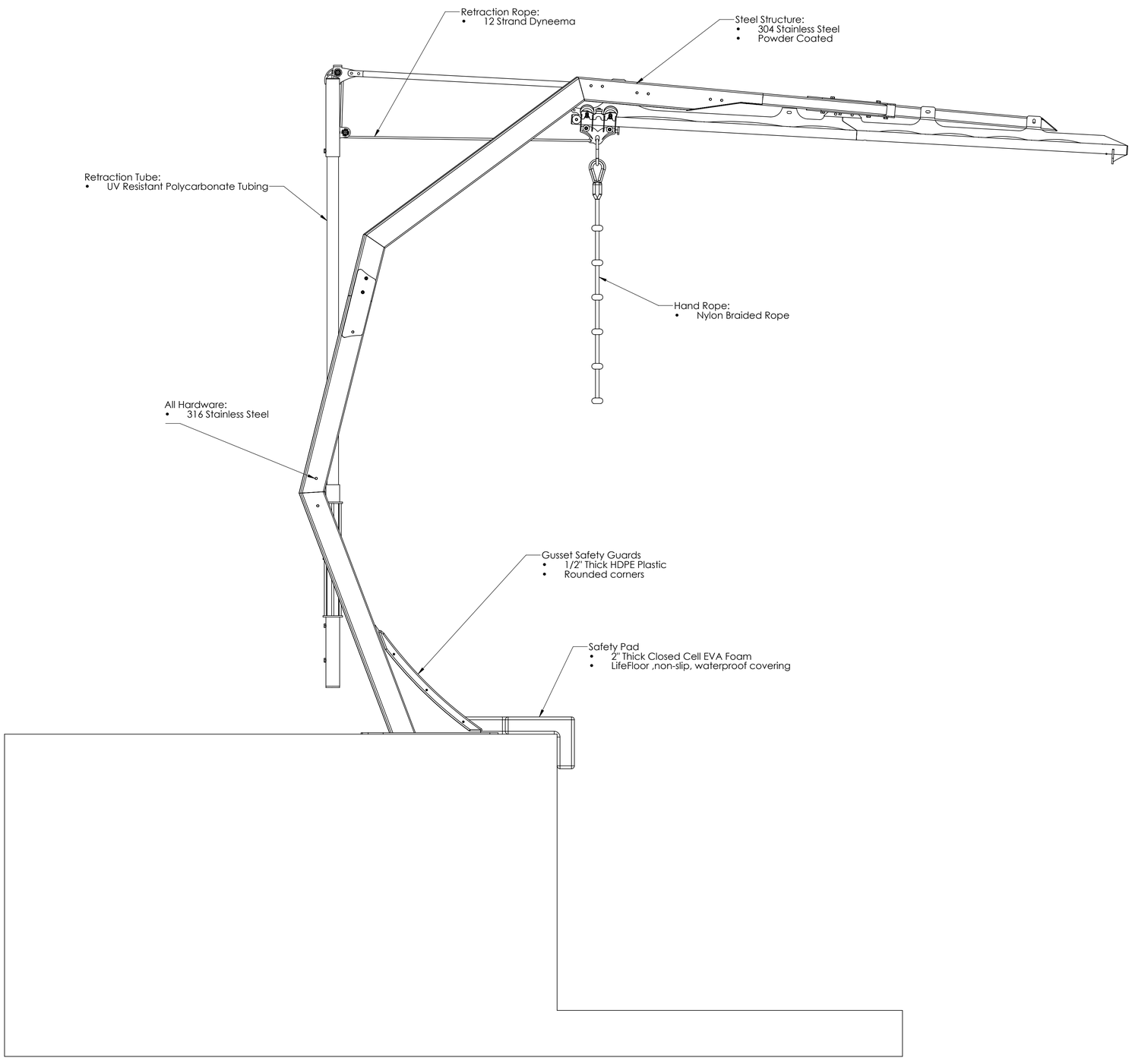

Material Specs

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TOLERANCES:
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ANGULAR: MACH ± 1 BEND ± 1
TWO PLACE DECIMAL ± .02 [0.51]
THREE PLACE DECIMAL ± .005 [0.127]
WELDS: 1/4

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TITLE:

SIZE	DWG. NO.	REV
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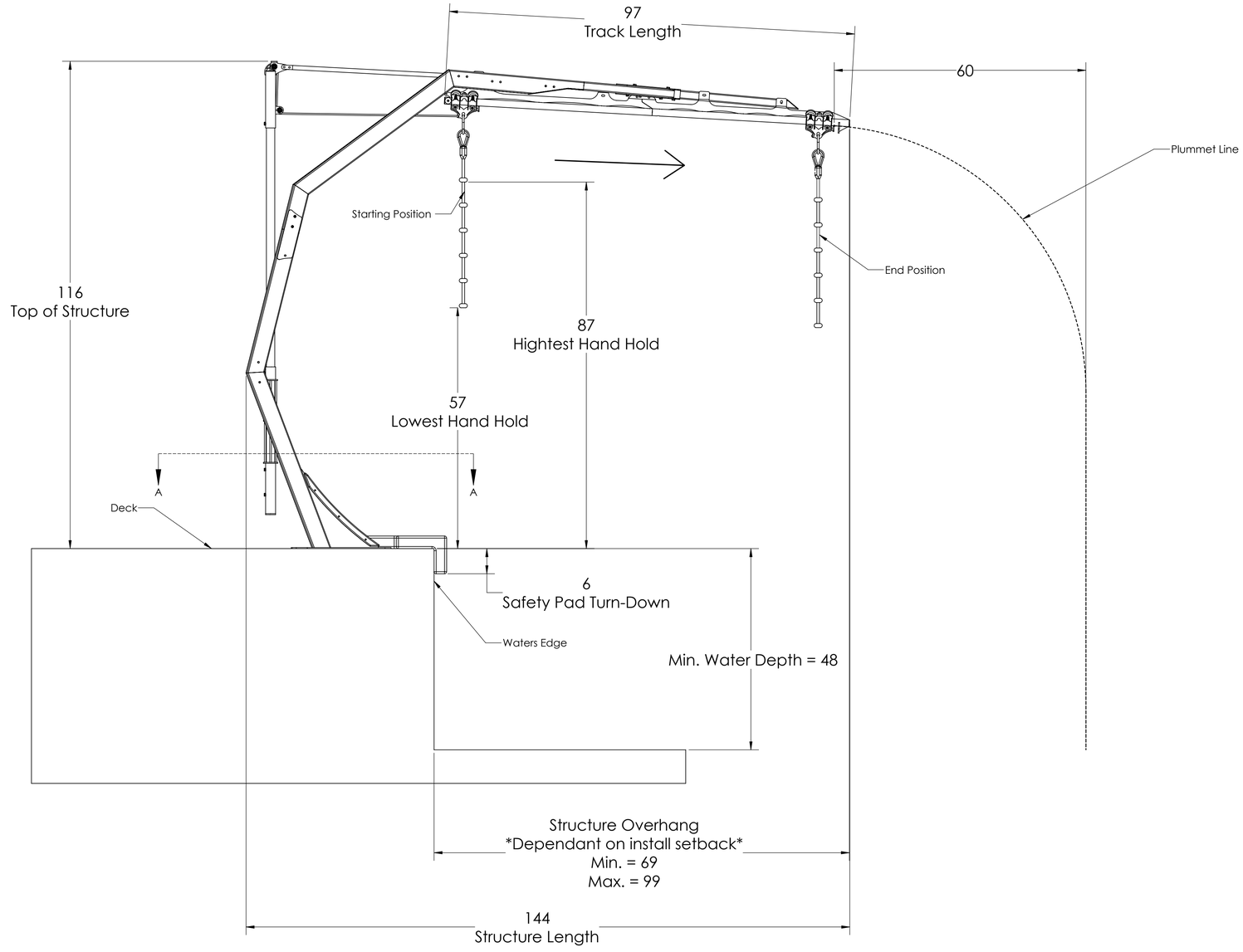
SCALE: 1:1 WEIGHT: 25799.23 SHEET 2 OF 7



Elevation View/Water Depth Req.

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ANGULAR: MACH ± 1 BEND ± 1
TWO PLACE DECIMAL ± .02 [0.51]
THREE PLACE DECIMAL ± .005 [0.127]
WELDS +1/16 -0

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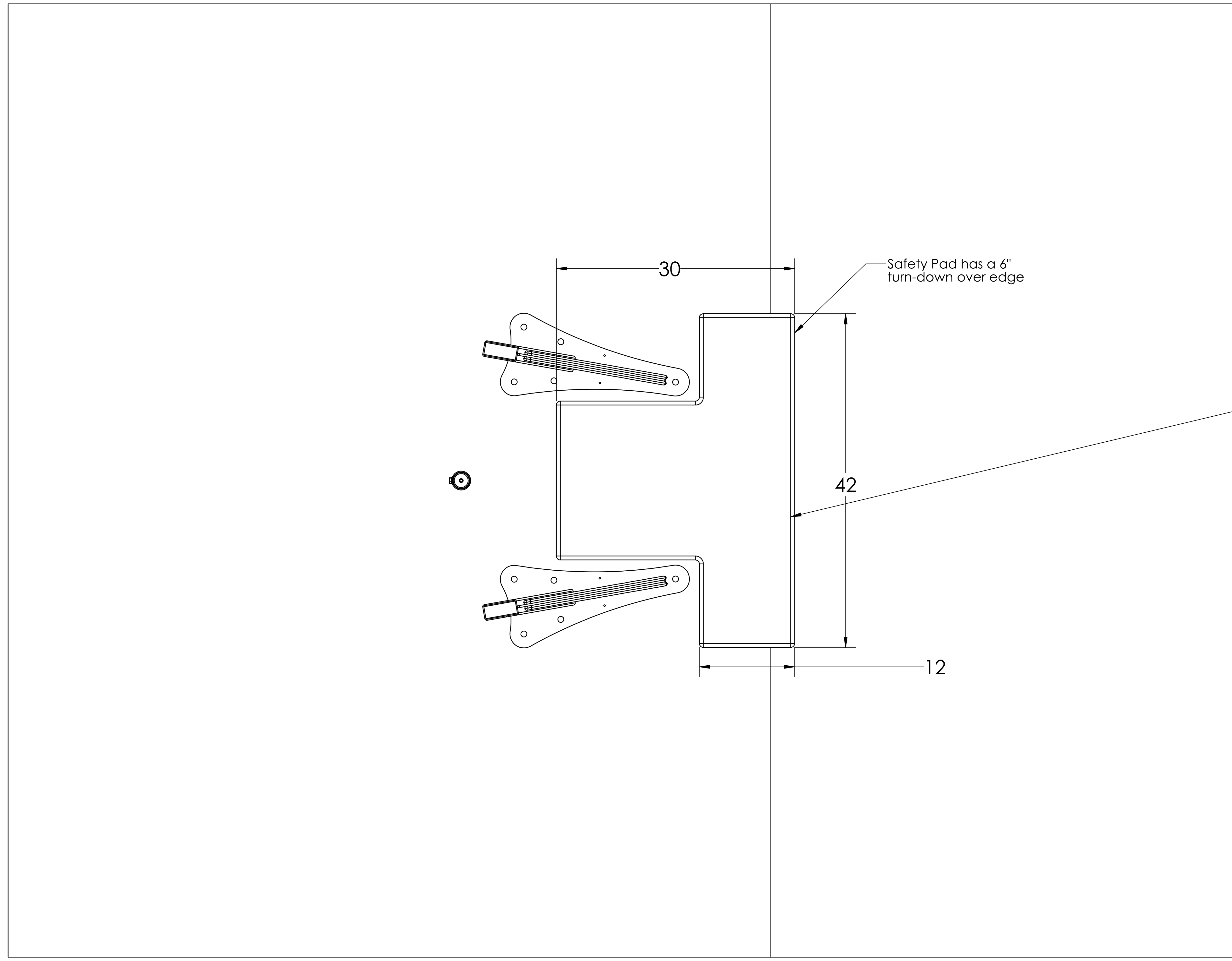
TITLE:

SIZE	DWG. NO.	REV
D	Z0037C_V3.1 Architectural	

SCALE: 1:1 WEIGHT: 25799.23 SHEET 3 OF 7



Safety Pad installs to deck using proprietary waterproof adhesive

SECTION A-A
SCALE 1 : 10

Safety Pad Dimensions

***Custome safety pads available upon request
to work with any gutter system***

Safety Pad Details

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DIMENSIONS ARE IN INCHES [mm]
TOLERANCES:
FRACTIONAL ± 1/16
ANGULAR: MACH ± 1 BEND ± 1
TWO PLACE DECIMAL ± .02 [0.51]
THREE PLACE DECIMAL ± .005 [0.127]
WELDS: $^{+1/16}_{-0}$

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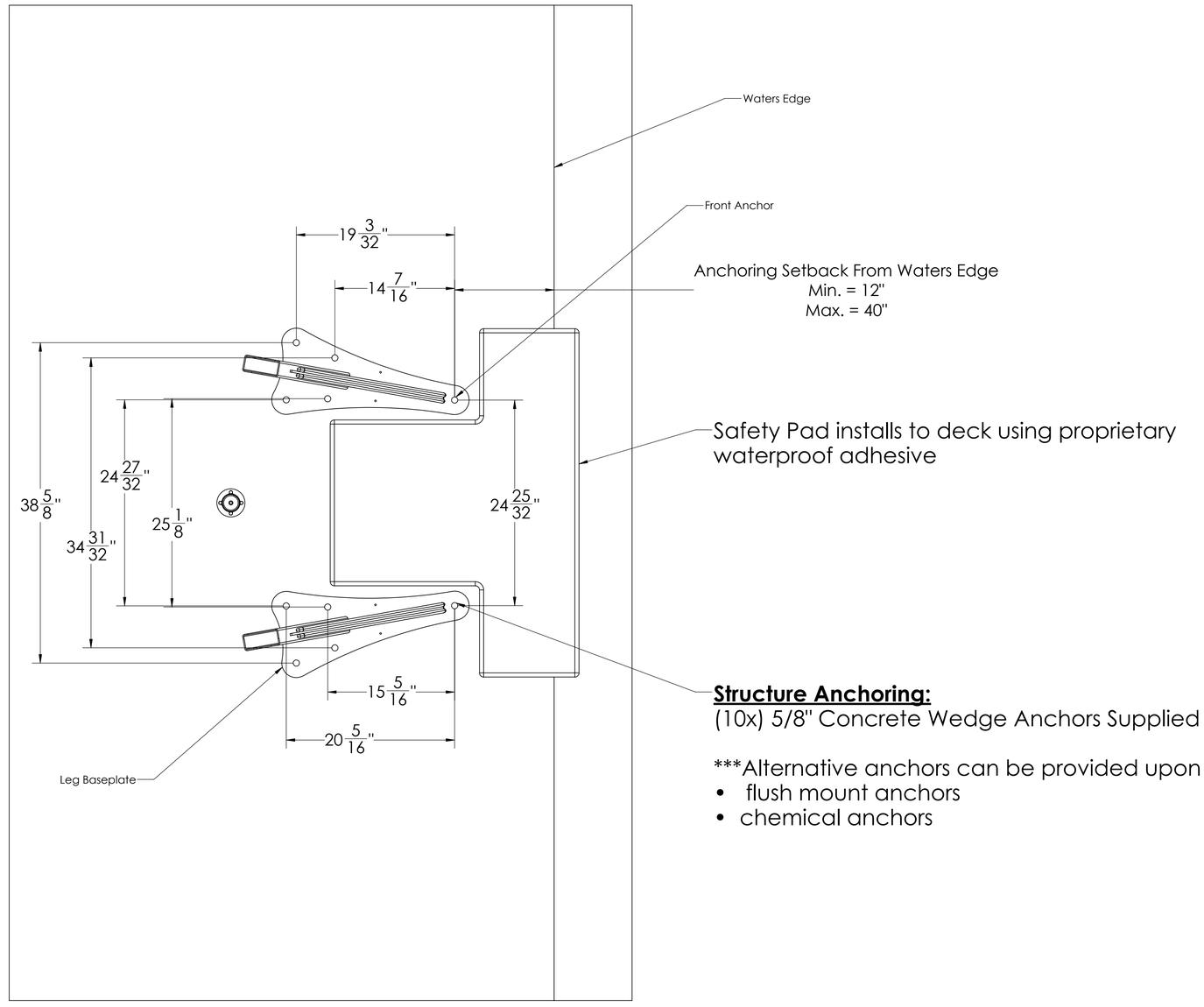
TITLE:

SIZE	DWG. NO.	REV
D	Z0037C_V3.1 Architectural	
SCALE: 1:1	WEIGHT: 25799.23	SHEET 4 OF 7



SECTION A-A
SCALE 1 : 8

Structure Anchoring:
(10x) 5/8" Concrete Wedge Anchors Supplied

***Alternative anchors can be provided upon request:
- flush mount anchors
- chemical anchors

*****Anchor dimensions are for reference only, not to be used for installation. Anchor installation is done by using the Leg Baseplates themselves as drilling templates.*****

Anchoring Details

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DIMENSIONS ARE IN INCHES [mm]
TOLERANCES:
FRACTIONAL ± 1/16
ANGULAR: MACH ± 1 BEND ± 1
TWO PLACE DECIMAL ± .02 [0.51]
THREE PLACE DECIMAL ± .005 [0.127]
WELDS: ±1/16 in

DO NOT SCALE DRAWING

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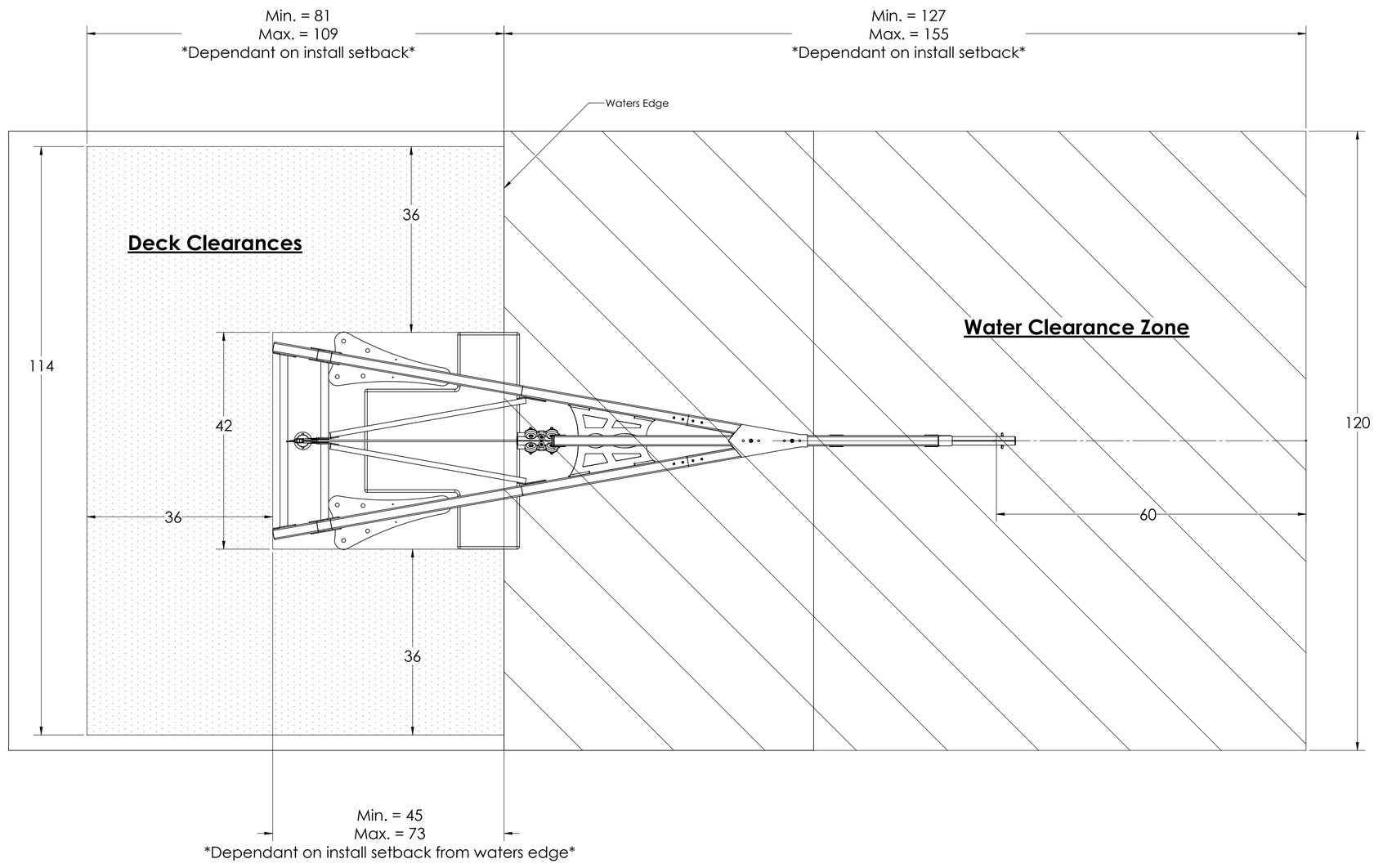
TITLE:

SIZE	DWG. NO.	REV
D	Z0037C_V3.1 Architectural	

SCALE: 1:1 WEIGHT: 25799.23 SHEET 5 OF 7



Water and Deck Clearances

THIS DRAWING HAS BEEN GENERATED AND IS MAINTAINED BY A CAD SYSTEM. CHANGES SHALL BE INCORPORATED AS DIRECTED BY THE DESIGN ACTIVITY

UNLESS OTHERWISE SPECIFIED:

DIMENSIONS ARE IN INCHES [mm]
TOLERANCES:
FRACTIONAL ± 1/16
ANGULAR: MACH ± 1 BEND ± 1
TWO PLACE DECIMAL ± .02 [0.51]
THREE PLACE DECIMAL ± .005 [0.127]
WELDS: ~1/4

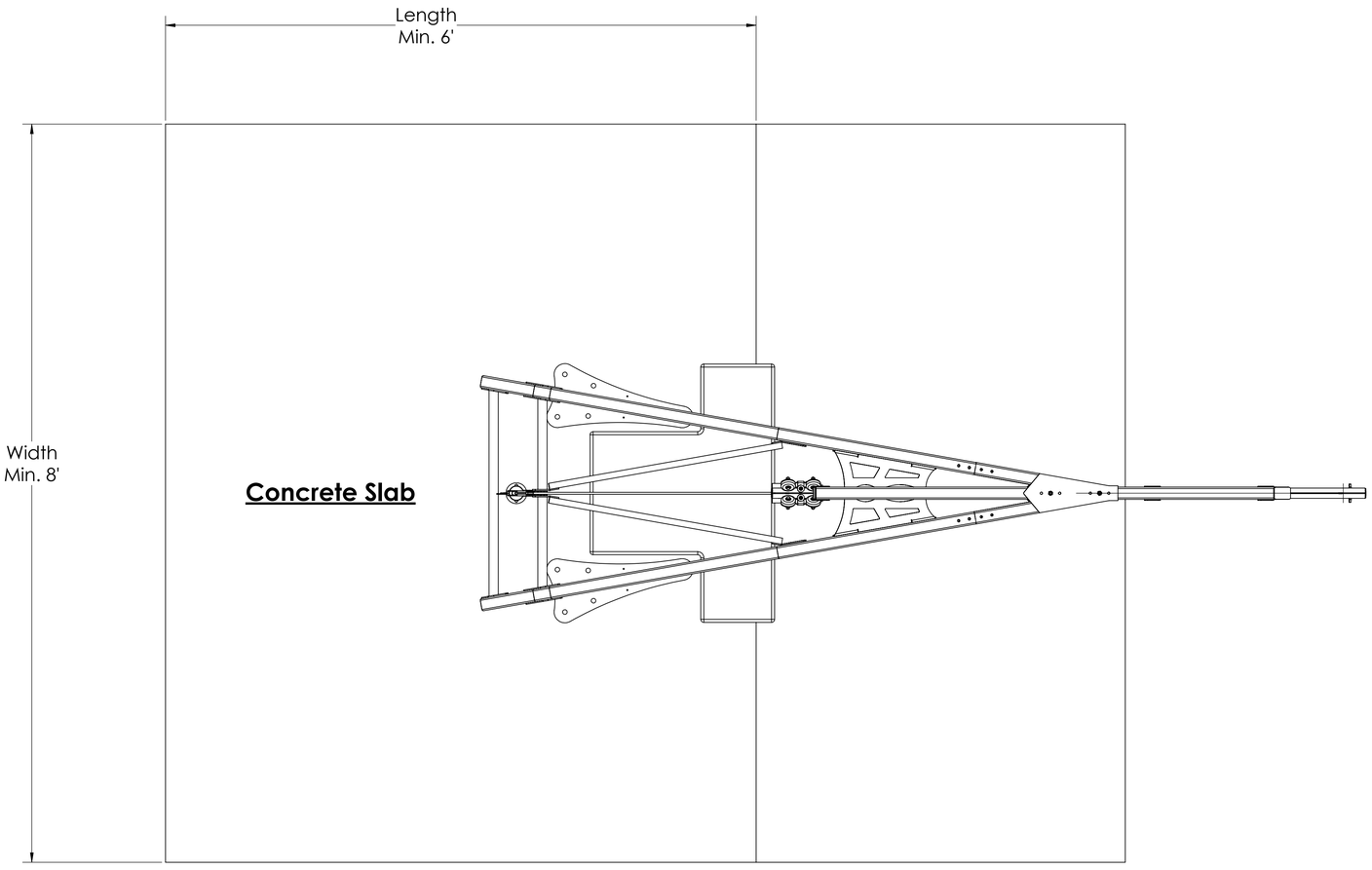
DO NOT SCALE DRAWING

Poolside Adventures
P.O. BOX 530
FREDERICK, MD 21705

TITLE:

SIZE	DWG. NO.	REV
D	Z0037C_V3.1 Architectural	

SCALE: 1:1 WEIGHT: 25799.23 SHEET 6 OF 7



Min. concrete thickness = 4"

Notes:
1. Location of front anchors no closer then 1' to front edge of pad.
2. Concrete dimensions shown are to acheive a min. required square footage. Alternative Lengths and widths can be accepted upon review.
3. Concrete width to be centered on AquaZip'n Frame.
4. Min. concrete thickness of 4" required, with 6x6 W2.0 welded wire mesh ASTM A185.
5. If concrete is new, minimum strength of 3000psi at 28 days is required.

Concrete Slab Requirements

THIS DRAWING HAS BEEN GENERATED AND IS MAINTAINED BY A CAD SYSTEM. CHANGES SHALL BE INCORPORATED AS DIRECTED BY THE DESIGN ACTIVITY

UNLESS OTHERWISE SPECIFIED:

DIMENSIONS ARE IN INCHES [mm]
TOLERANCES:
FRACTIONAL ± 1/16
ANGULAR: MACH ± 1 BEND ± 1
TWO PLACE DECIMAL ± .02 [0.51]
THREE PLACE DECIMAL ± .005 [0.127]
WELDS +1/16 -0

DO NOT SCALE DRAWING

Poolside Adventures
P.O. BOX 530
FREDERICK, MD 21705

TITLE:

SIZE D | DWG. NO. Z0037C_V3.1 Architectural | REV

SCALE: 1:1 WEIGHT: 25799.23 SHEET 7 OF 7

NAC
ARCHITECTURE

Patty Hayes, Board Chair
Washington State Board of Health
PO Box 47990
Olympia, WA 98504-7990

AQUATIC CENTER at MLK JR. PARK, Yakima

Variance Letter D ate: 2024.06.20

STATE IDENTIFICATION: State ID Facility #: F0476 Project #:2024003

Facility Information:
Aquatic Center at MLK Jr. Park (New outdoor pool facility with 5,300sf pool building and two leisure pools)
Plan Submittal: Drawing Plans have been submitted for review.

Aquatic Center at MLK Jr. Park, City of Yakima

Owner Contact:	Ken Wilkinson	Phone: 509-576-6416
Owner Address:	129 N 2nd street	Yakima, WA 98901
Facility Address:	610 S 9th Street	Yakima, WA 98901
Owner Representative:	Brooke Hanley (NAC Architecture) 509-838-8240	

Variance Request Contact:
NAC Architecture: Brooke Hanley Phone: 509-838-8240 Email: bhanley@nacarchitecture.com

Variance Request Citation:
WAC 246-262-160 states *the board may grant a variance from requirements of chapter **246-262** WAC if, in the sole discretion of the board, data and/or research provides sufficient evidence that the RWCF (attraction, device, equipment, procedure, etc.), will adequately protect public health and safety, as well as water quality.*

Variance Request: Code Related to Diving Envelope (WAC 246-262-010(21) & WAC 246-262-060(5)(vi)) for a **climbing wall** attraction.

Items noted in review letter include:

- **Climbing wall** attraction receiving pool shall meet the 2000-2001 FINA facility rules (depth application and setbacks)

In the Department of Health review response letter issued by Justin Law dated May 22, 2024, Justin requests NAC Architecture (NAC) and WaterTechnology, Inc. (WTI) to address important concerns regarding public safety related to the receiving pool for the proposed **climbing wall** attraction in Pool B. The concern is to address the minimum depth of the pool to be compliant with the WAC 246-262-010(21) & WAC 246-262-060(5)(c)(vi) regarding diving envelopes for features where users enter the water at 20” or higher above the water surface.

On behalf of the City of Yakima, WA; NAC & WTI respectfully requests your consideration of the current pool depth design at the climbing wall for the future Aquatic Center at MLK Jr. Park. To support this request we provide the attached information, engineering exhibits, and following commentary:

- The review letter states that the "diving envelope" from WAC 246-262-010(21) applies to **all attractions** where users enter above pool water level and therefore requires the CNCA (enter less than 20" above the water surface) or FINA (enter 20" or greater above the water surface) water depths. We submit that the attached engineering calculations for the **AquaClimb 3-Panel-High climbing wall** product will demonstrate that the manufacturer's required water depths and the designed water depths provided at the Yakima Aquatic Center are sufficient to protect the safety of the range of users allowed to participate in this attraction. Calculations were completed for a 48" tall, 50lbs person and a 78" tall, 250lbs person to show a range of sizes requested in the review letter. Please reference page 9 for the manufacturer's minimum depth requirements and pages 10-17 for the engineering calculations and associated notes. The Yakima design provides for 6" greater water depth than the minimum required by this engineering report. Please review the attached data in support of using the manufacturer's depth requirements in lieu of the CNCA or FINA diving envelope dimensions.

- WAC 246-262-060(5)(c)(vi) appears to apply specifically to "diving envelopes in pools or areas of pools designated for diving activities". The applicant submits that diving activities are generally defined as plunging into the water headfirst. Diving headfirst into water results in the need for deeper water to avoid a head & neck collision with the bottom of the pool which is different than a feet-first or tucked entry plunge where the body is significantly slowed in the first two feet of water. The **climbing wall** safety guidelines and standard operating procedures (provided in the exhibits) will note that users are required to re-enter the water in a feet-first manner. Diving from the unit is prohibited (and per the manufacturer data, bio-mechanically impossible). The engineering calculations completed also assumes a feet-first plummet into the water.

- The Model Aquatic Health Code also addresses the complexity of "other aquatic features" like **climbing walls** and would suggest that the manufacturer recommendations for design and operation would be adequate to install the feature.

  4.12.10[A] Other Aquatic Features Other AQUATIC FEATURES not otherwise addressed in the CODE, including but not limited to climbing walls, inflatables, and play structures, shall not be installed unless designed and operated in accordance with all manufacturer's installation and operations recommendations.

- 'A-frame' signs with all written safety guidelines will be publicly displayed near the **climbing wall** (see page 18 for example) to meet the criteria of WAC 246-262-070(10). The design team could also instruct AquaClimb to add a maximum height of 78" to the sign to correspond to the engineering calculations, if this would mitigate concerns over swimmers participating that do not fit within the engineering assumptions.

- See attached climbing wall diagram. The frame and panels of the wall tilt out over the water, ensuring the swimmer's descent is away from the wall and pool edge. The protective panels at the top do not have hand-holds and therefore prevents climbing over the top of the structure.

- This pool will be lifeguarded at all times while in operation and the lifeguard staff will be the first line of defense to screen bathers to make sure they are experienced swimmers, instruct swimmers on proper use of the attraction, and direct proper swimmer circulation to and from the activity within the pool to avoid congestion or collisions. The **climbing wall** will have a dedicated lifeguard to closely supervise the safety of swimmers when the attraction is open for use.

- Injury statistics requested by the review letter are not available from the manufacturer or another source, but the product literature, research paper, and testing tout the relative safety of the **climbing wall** compared to diving boards and slides. They also have over 1,000 installations across the world. See the provided letter from Aquatic Safety Research Group.

- The **AquaClimb** has also been designed and engineered to meet the following standards:
  - ASTM F24/F2291-21 Standard Practice for Design of Amusement Rides and Devices
  - ASTM F2461-20 Aquatic Play Equipment
  - European Standards EN17164 – Climbing walls for use in the water area
  - IBC 2018 & AISC Manual of Steel Construction
  - Other industry standards listed in the product data attached

- The City of Yakima specifically requested a pool design that would have a variety of intriguing activities for their patrons but would not need water deeper than 6-7ft. Pools deeper than 6-7ft come with their own safety risks and lifeguarding challenges. Shallow water is easier to supervise and guard. Rescues are much more likely to be needed in deep water where a bather in trouble cannot push off the bottom of the pool to bob back above the surface quickly until the lifeguard can assist them. Yakima is dedicated to making this facility fun while also as safe as possible for their community members and patrons.

- NAC submits that the design as described above and substantiated in the attached documentation meets the intent of providing a safe receiving pool for the **climbing wall** feature. NAC, WTI, and the City of Yakima respectfully requests a variance accordingly. If the State Board of Health has any follow-up conditions or actions required of the owner/operator, we are committed to implementing them.

NAC Architecture (NAC) has teamed with Water Technology (WTI) on numerous aquatic projects and so we have a history of producing these projects successfully. WTI has been designing Aquatic venues for over 40 years. WTI is widely known in the industry as one of the leading aquatic design firms in North America. As one of the industry's leaders, WTI has represented the waterpark industry during CPSC meetings on review of VGB rules and has also been involved in reviewing/editing sections of the MAHC.

They are also represented in the Washington DOH committee to update the existing administrative code to adopt a more comprehensive aquatic code like the MAHC. The NAC and WTI commitment to safe aquatic facilities is proven. The design of the receiving pool at the **climbing wall** for the Yakima Aquatic Center will not put the health and safety of the public at risk. The City of Yakima, having operated a public pool for many years is experienced and committed to the safety and the welfare of their patrons.

On behalf of the City of Yakima, NAC Architecture would like to thank you for your consideration of this Variance Request. Please feel free to contact me with any questions you may have regarding this request.

Thank you,

Brooke Hanley

Brooke Hanley, AIA, Principal Architect, NAC Architecture

Attachments:

- AquaClimb Safety and Fall Zone Engineering, including a floor plan and section of the receiving pool as designed for the Yakima Aquatic Center.

POOL B-ACTIVITY DATA

DESCRIPTION	QTY	UNITS		
POOL PERIMETER	314'-0"	FEET		
WATER SURFACE AREA	3,832	SQUARE FEET		
POOL WATER TEMPERATURE	84	°F		
POOL VOLUME	136,514	GALLONS		
SURGE TANK OPERATING VOLUME	7,415	GALLONS		
TOTAL VOLUME OF WATER	147,268	GALLONS		
CIRCULATION RATE	1,033	GPM		
TURNOVER/VOLUME/FLOW	60 MIN.		19,330 GAL.	322 GPM
TURNOVER/VOLUME/FLOW	180 MIN.		127,938 GAL.	711 GPM
FILTRATION RATE	12.66	GPM/FT²		
BACKWASH FLOW	306	GPM		
SURGE FACTOR	1.06	GAL/SQFT		
AVAILABLE SURGE CAPACITY IN SURGE TANK	4075	GALLONS		

SCHEDULE - BASIS OF DESIGN - POOL B

POOL ID	EQUIPMENT ID	EQUIPMENT	QTY	MANUFACTURER	DESCRIPTION
B	01	POOL LIFT	1	SR SMITH, AQUA CREEK, OR EQUAL	STANDARD ANCHORED, ROTATIONAL POOL LIFT, WITH 400 LB MINIMUM LIFTING CAPACITY. MUST MEET ALL APPLICABLE ADA REQUIREMENTS, WHILE MAINTAINING REQUIRED DECK CLEARANCE. PACKAGE TO INCLUDE ARMRESTS, ANCHOR, LIFT COVER, BATTERY CHARGER, AND CADDY
B	02	GRAB RAILS (PAIRS)	6	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	PRETZEL BEND STYLE, 1.50" OD x .120 WALL THICKNESS, 500 GRIT FINISH MIN.
B	03	ESCUTCHEON PLATE	34	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	STAINLESS STEEL ROUND ESCUTCHEON FOR 1.50" O.D. RAILS
B	04	WEDGE ANCHOR	34	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CAST BRONZE, 4-1/4" LONG, ACCEPTS 1.500" OD TUBING
B	05	IN-WALL STEPS	18	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	17-1/2" x 6", INJECTION MOLDED PLASTIC, PEBBLE TEXTURE, 1/4" WALL THICKNESS
B	09	LANE DIVIDERS	3	COMPETITOR SWIM PRODUCTS	4" WAVE QUELLING RACING LANE LINE, COLORS BY OWNER / ARCHITECT
B	10	DaiFLEX LANE LINE ANCHOR	6	DALDORADO	12" – NON-CORROSIVE PVC FLIP UP LANE LINE ANCHOR TO BE USED WITH DALDORADO PARALLEL GRATING. INCLUDES FLIP-UP HATCH, BASE UNIT, & SILICON COVERED SS BRAIDED STRAP EXTENSION WITH HOOK. CAN BE USED WITH THE DaiFLEX 8" OR 14" LANE LINE EXTENSION.
B	11	SAFETY ROPE	6	PARAGON AQUATICS	3/4" POLYETHYLENE ROPE WITH 5"x9" HANDI-LOCK FLOAT, VERIFY LENGTH WITH PLANS
B	12	CUP ANCHOR	10	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	4" SQUARE 304L SS ANCHOR AND 304L SS EYE BOLT
B	13	BASKETBALL HOOP	1	SR SMITH	STAINLESS STEEL BASKETBALL HOOP WITH ROCKSOLID ANCHOR
B	14	AQUA ZIPN	1	AQUACLIMB	DECK-MOUNTED OVERHEAD ROPE SWING, WITH SELF-RETRACTING TROLLEY, POWDER-COATED STAINLESS STEEL WITH HIGH TENACITY POLYESTER ROPE. INCLUDES SAFETY PAD-UNIVERSAL, WITH 316 SS HILTI FLUSH MOUNT CONCRETE ANCHORS.
B	15	AQUACLIMB	1	AQUACLIMB	2 WIDE X 3 HIGH AQUATIC CLIMBING WALL
B	16	LIFEGUARD CHAIR	2	TAILWIND, KEIFER, SPECTRUM AQUATICS, SR SMITH OR APPROVED EQUAL	RECYCLED PLASTIC WITH 304 SS HARDWARE, COLOR BY OWNER/ARCHITECT 40" SEAT HEIGHT (OWNER'S SAFETY CONSULTANT TO SPECIFY LOCATION.)
B	17	NINJACROSS	1	NINJACROSS	AQUATIC OBSTACLE COURSE
B	18	SAFETY PAD	3	PLAYTIME	WALL AND DECK SAFETY PAD AT NINJACROSS SYSTEM

SCHEDULE - CUSTOM RAILGOODS - POOL B

POOL ID	EQUIPMENT ID	EQUIPMENT	QTY	MANUFACTURER	DESCRIPTION
B	01	HAND RAIL	3	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CUSTOM FABRICATED, 316L SS, 1.50" OD x .120 WALL THICKNESS, 500 GRIT FINISH MIN.
B	02	HAND RAIL	2	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CUSTOM FABRICATED, 316L SS, 1.50" OD x .120 WALL THICKNESS, 500 GRIT FINISH MIN.

SCHEDULE - WATER FEATURE - POOL B

POOL ID	FEATURE ID	FEATURE	QTY	MANUFACTURER	DESCRIPTION	GPM (ea)	GPM (Total)
B	F01	DROP SLIDE	1	SPLASHTACULAR	FUTURE SLIDE PROVIDE PIPING CAPPED ONLY	500	500
B	F02	WATER SPRAY	2	WATERPLAY	PIPE DELUGE-FAN SPRAY FEATURE	60	120

INSTALL A 4" CONTRASTING VERTICAL PAINT STRIPE FROM 4" ABOVE FLOOR UP TO THE WATERLINE TILE. TYPICAL ALL INSIDE CORNERS. 3 / PL102

CLIMBING WALL IN ELEVATION VIEW, RECEIVING POOL IN SECTION VIEW

CLIMBING WALL IN PLAN, RECEIVING POOL IN PLAN VIEW

4" WIDE BAND @ 5'-0" WD CONTINUOUS ON POOL WALLS AND FLOOR. COLOR CONTRASTING TO POOL FINISH 2 / PL102

Pool Lower Deck Level 0"

Pool Static Water Level 0"

6' - 6" [1975] WD

FILL

5' - 0" [1525] | 8' - 6 3/4" [2600] | 5' - 0" [1525]

CLIMBING WALL DRAWINGS AT POOL B

PL120 POOL B - ACTIVITY PLAN
PLAN VIEW
1/4" = 1'-0"

N

REVISIONS

REV.NO.	DESCRIPTION	DATE
1	CHANGE PROPOSAL	04/16/2024

CONFORMED SET

CITY OF YAKIMA
YAKIMA POOL
YAKIMA WA

WTI WATER TECHNOLOGY, INC.
World Leaders in Aquatic Planning, Design and Engineering
100 Park Avenue | Beaver Dam, WI 53916
t 920.887.7375

NAC ARCHITECTURE
nacarchitecture.com
1203 WEST RIVERSIDE AVENUE
SPOKANE WA 99201
P:509.838.8240

NAC NO 111-22082
ISSUE DATE: 4/16/24
PROJECT NUMBER: 22314
DRAWN BY: T.ED
CHECKED BY: ACC

7893 REGISTERED ARCHITECT
MATTHEW W. FREEBY
STATE OF WASHINGTON
4/16/2024

POOL B - ACTIVITY POOL PLAN

PL120

Turn your pool into an ADVENTURE with AquaClimb®

For recreation centers, fitness facilities, camps, and private clubs, AquaClimb expands poolside programming with an easy addition that is safe, engaging, and fun. As the market leader, AquaClimb offers more benefits to its customers than any other climbing product:

Modular and Customizable

AquaClimb's height, width, and panel style can all be tailored to fit the size and design of your pool, with options for adding more panels at a later phase as your budget allows.

Challenging, Realistic Climbing

With 3D contoured panels, AquaClimb delivers a realistic rock-climbing experience that engages adolescents through adults to conquer the climb in different ways.

Top Safety Record

With best-in-class safety features to ensure climbers fall away from the wall, AquaClimb also has a proven performance history from 1,000 installations across the globe.

Activates the Deep End

As a safer alternative or enhancement to diving boards, AquaClimb attracts tweens and teens to those under-utilized, deep areas of a pool.

Easy to Install

Because AquaClimb is pre-assembled in the factory, no specialized skills or equipment are required for onsite installation at your facility on any pool gutter configuration.

Minimal Footprint

AquaClimb's small deck-mounted system saves clearance space and doesn't interfere with normal lap swimming. And with no water source required, it is an easy amenity to add.

AQUACLIMB® Four Unique Models

AquaClimb Krystal

- Budget-friendly and entry-level option
- Modular, flat panels in clear, blue, and green transparent tint
- Customizable up to four height options sized to pool's depth

AquaClimb 3D

- 3D contoured panels for realistic climbing available in translucent **Ice, Glacier,** or **Jade** colors, and solid painted color schemes
- Modular panels can be turned and flipped to change up the experience
- Translucent panels allow lifeguard visibility while giving privacy to the climber behind the wall

AquaClimb Kurve

- Sleek, curved frame that allows heights up to 20 feet
- 3D contoured panels available in color options of Ice or Glacier
- Translucent panels allow lifeguard visibility while giving privacy to the climber behind the wall

AquaClimb Luxe

- Completely customizable design to match your pool's aesthetics
- 3D contoured panels
- Deck mounted or Pool wall mounted

Take on the ADVENTURE with AquaClimb®

It's never been easier to add an exciting new amenity to your:

- Camp
- Country Club
- College/University
- Swim Club
- Recreation/Aquatic Facility
- Health/Fitness Center
- Military Wellness & Recreation
- Private Residence

Join thousands of other satisfied customers who love their AquaClimb:

"Our AquaClimb is spectacular. From the time we open the pool until the time we close, there is a line to make the climb. What an ingenious product and so much fun for the kids... and a few adults."

Mark Tiernan
General Manager at the Valley Country Club
Centennial, CO

"We had a great first year with the AquaClimb. Kids were constantly lined up for it, and everyone had a blast. AquaClimb was a big reason we saw a 40% increase in attendance over the last year."

Ted Davis
Southfield Parks and Recreation
Southfield, MI

To learn how you can bring the adventure of AquaClimb to your facility, contact us today:

POOLSIDE ADVENTURES™

PoolsideAdventures.com | 800.956.6692 | info@PoolsideAdventures.com

Building Courageous Kids for Life's Great Adventure

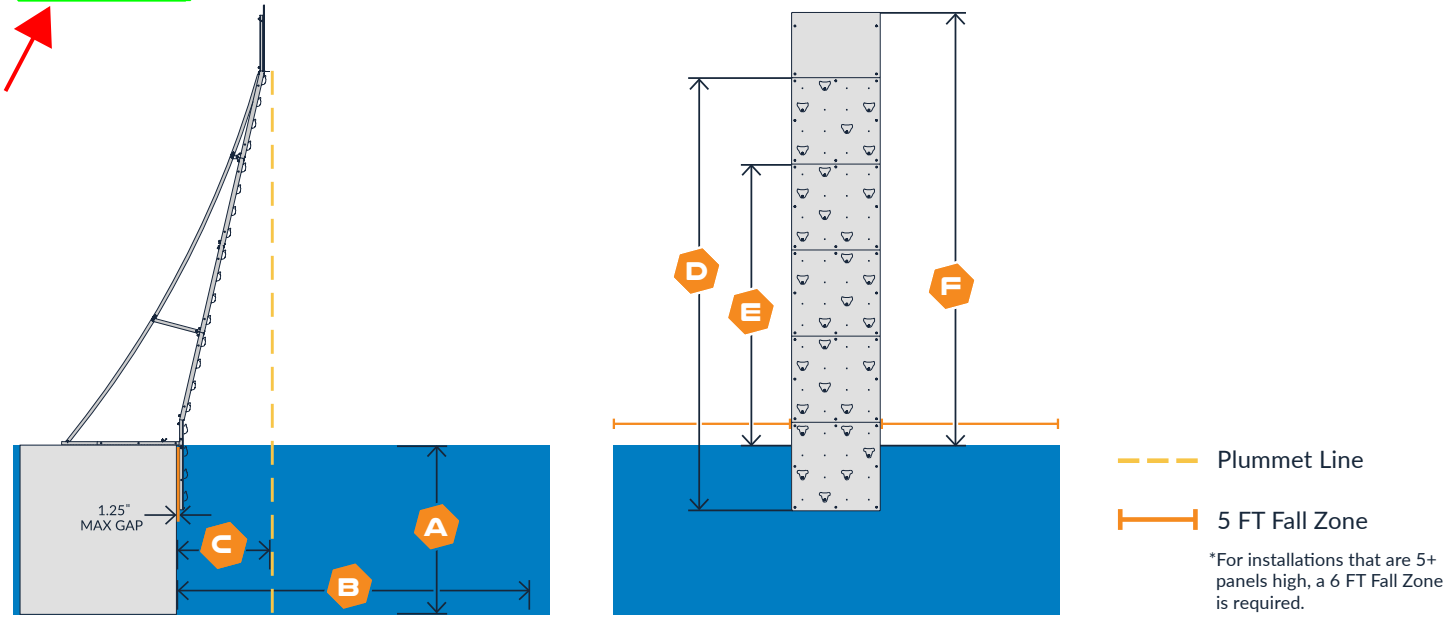
AQUACLIMB® Depth Requirements

Panel Options	A Minimum Pool Depth	B Drop Zone	C Plummet line from wall	D Available climbing height	E Height of top foothold*	F Above deck wall height
3 High Alt	5'	9'	1'9"	8'10"	4'5"	9'7"
3 High Yakima Product	6'	9'	1'9"	9'10"	5'5"	9'7"
4 High Alt	6'	10'	2'6"	12'1"	7'8"	12'10"
4 High	7'	10'	2'6"	13'1"	8'8"	12'10"
5 High Alt	8'	12'	3'3"	15'5"	11'	16'1"
5 High	9'	12'	3'3"	16'5"	12'	16'1"
6 High (Kurve Only)	10'	12'	3'3"	17'	12'5"	19'8"

*Based on climber's feet positioned at least 2' below highest hand grip

Alt - Alternate configurations will have the top row of handholds plugged for non-climbing terrain to meet pool depth requirements.

Important Safety Note: AquaClimb safety distances and pool depths are based upon a climber entering the water **feet first**. The AquaClimb was designed for a feet first entry at all times and supervision must be present when the AquaClimb is in use. To ensure the maximum level of safety, **there must be no diving at any time**.



To learn how you can bring the adventure of AquaClimb® to your facility, contact us today:

POOLSIDE ADVENTURES™

PoolsideAdventures.com | 800.956.6692 | info@poolsideadventures.com

Building Courageous Kids for Life's Great Adventure

FEAmax Report

AquaClimb Hand Calculation

“The information contained in this document is proprietary and confidential to FEAmax LLC. FEAmax submits this document with the understanding that it will be held in the strictest confidence and will not be disclosed, duplicated or used, in whole or in part [for any purpose other than evaluation of FEAmax qualifications] without the prior explicit written consent of FEAmax.”

FEAmax LLC.

PROJECT INFO.

Change History:

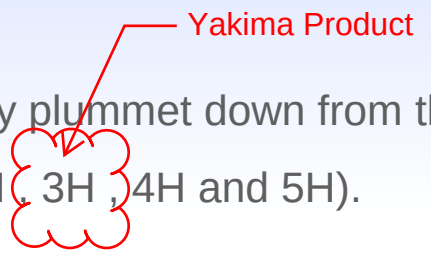
Version Number	Date	Summary	Author
V 1.0	2/2/2016	Initial release	Frank Wang

Client Information:

Contact name:	Laura Grandner
Email:	Laura@aquaclimb.com
Company name:	Pyramide USA
Address:	P.O. Box 530 Frederick, MD. 21705

PROJECT DESCRIPTION

- **Project Description**



1. Calculate the minimum depth required to safely plummet down from the highest foot hold point on the (4) levels of AquaClimb Walls (2H , 3H , 4H and 5H).
2. With the top climbing hold measurement provided – deduct 36” (3ft) down which would be the highest foot hold placement. Then with the following parameters calculate the minimum depth needed to safety let go and plummet straight down into the water without reaching the bottom floor of the pool.
3. Height: 48” minimum; 78” Maximum
4. Weight: 50 lbs minimum; 250 lbs maximum

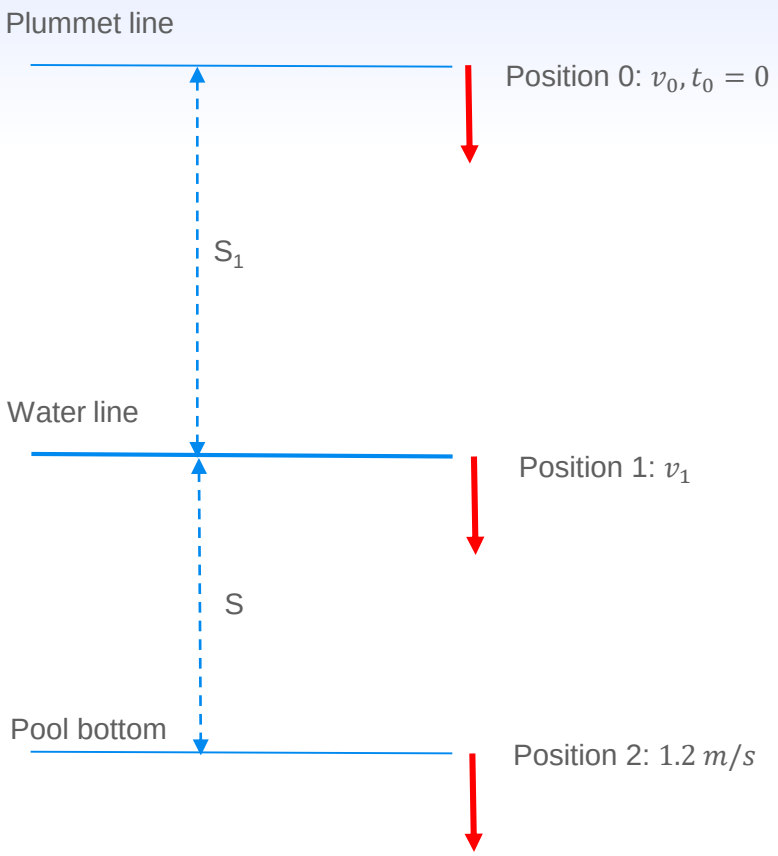
CALCULATION

Assumptions:

1. Minimum height of human body H_{human}= 48” = 1.2 meter
2. Water density ρ_{water}= 1.0 g/cm^3
3. Human body density ρ_{human}= 0.9 g/cm^3
4. The velocity enter the water = V_1
5. Water Resistance coefficient C_D = 1.0
6. Human body volume = V
7. Area of human body enter the water = A
8. Velocity of human body inside the water = V_x
9. The allowable decent velocity to the pool bottom = 1.2 m/s

Force applied to human body inside water:

1. Gravity $G = \rho_{human} g V$
2. Buoyancy (floating force) $\mathrm{F} = \rho_{water} g V$
3. Water resistance force $F_{resistance} = \frac{1}{2}\rho_{water} V_x^2 A C_D$



CALCULATION

According to Newton's second law, we have:

1. The acceleration in the water: $a = \frac{dV_x}{dt} = \frac{F}{m}$

2. $a = \frac{\rho_{human} gV - \rho_{water} gV - \frac{1}{2}\rho_{water} V_x^2 A C_D}{\rho_{human} V} = \frac{0.9\times9.8\times V - 1.0\times9.8\times V - 0.5\times1.0\times V_x^2 \times \frac{V}{1.2}\times1.0}{0.9\times V} = -(1.09 + 0.46V_x^2)$

3. $\frac{dV_x}{dt} = -(1.09 + 0.46V_x^2)$

4. $\mathrm{dt} = -\frac{dV_x}{(1.09+0.46V_x^2)}$

5. The max displacement of body moving in the water would be:

$$S = \int_0^t V_x \cdot dt = -\int_{1.2}^{V_1} V_x \cdot \frac{dV_x}{1.09 + 0.46V_x^2} = \cdots = -\int_{1.2}^{V_1} 0.46 \times \frac{1}{0.42} \times \frac{d(1 + 0.42 \times V_x^2)}{(1 + 0.42 \times V_x^2)}$$

$$= 1.09 \times [\ln(1 + 0.42 \times V_1^2) - \ln(1 + 0.42 \times 1.2^2)] = 1.09 \times [\ln(1 + 0.42 \times 2 \times 9.8 \times S_1) - 0.473]$$

6. The minimum depth of pool would be:

$$S = 1.09 \times \ln(1 + 8.23 \times S_1) - 0.52$$

CONCLUSION

If the body height is 48” (1.2 meter), we have:

$$S = 1.09 \times \ln(1 + 8.23 \times S_1) - 0.52$$

1. For 2H: S_1 = 1’ = 0.30 meter, we have the min pool depth:

   S = 0.84 meter = 2.8 feet

2. For 3H: S_1 = 1’9” = 0.53 meter, we have the min pool depth:

   S = 1.31 meter = 4.3 feet

3. For 4H: S_1 = 2’6” = 0.76 meter, we have the min pool depth:

   S = 1.64 meter = 5.4 feet

4. For 5H: S_1 = 3’3” = 1 meter, we have the min pool depth:

   S = 1.89 meter = 6.2 feet

Yakima Pool depth at climbing wall exceeds this recommendation and is 6'-6" deep

Yakima Product

Standard Height Options	Distance of plummet line from pool wall (A)	Minimum pool depth required (B)
2H	1'	4'
3H-5'	1' 9"	5'
3H	1' 9"	6'
4H-8'	2' 6"	8'
4H	2' 6"	9'
5H-11'	3' 3"	11'
5H	3' 3"	12'

CONCLUSION

If the body height is 78” (1.98 meter), the equation would be:

$$S = 1.78 \times \ln(1 + 5.49 \times S_1) - 0.60$$

1. For 2H: S_1 = 1’ = 0.30 meter, we have the min pool depth:

   S = 1.13 meter = 3.7 feet

2. For 3H: S_1 = 1’9” = 0.53 meter, we have the min pool depth:

   S = 1.83 meter = 6.0 feet

3. For 4H: S_1 = 2’6” = 0.76 meter, we have the min pool depth:

   S = 2.32 meter = 7.6 feet

4. For 5H: S_1 = 3’3” = 1 meter, we have the min pool depth:

   S = 2.73 meter = 8.9 feet

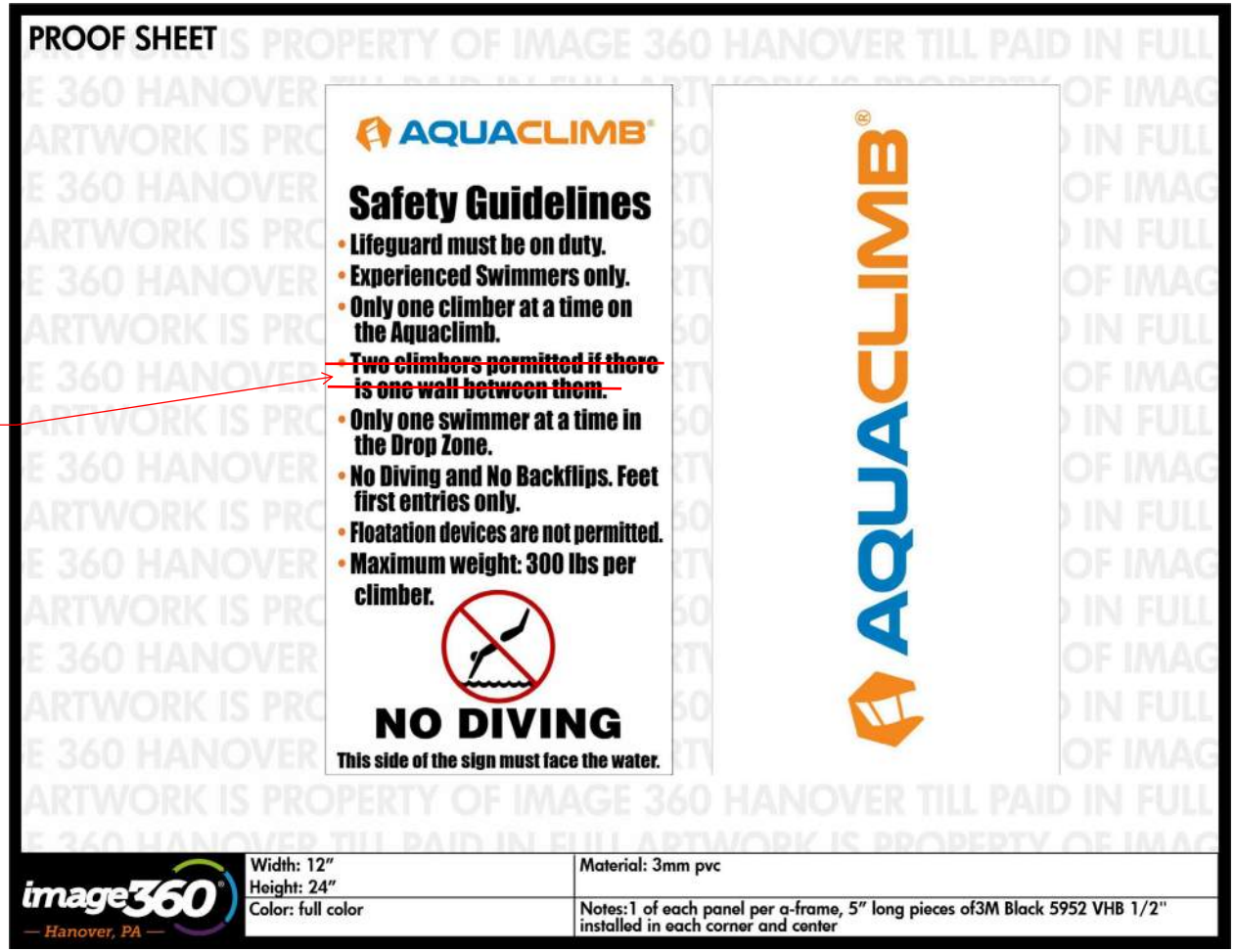
Yakima Pool depth at climbing wall exceeds this recommendation and is 6'-6" deep

Yakima Product

Standard Height Options	Distance of plummet line from pool wall (A)	Minimum pool depth required (B)
2H	1'	4'
3H-5'	1' 9"	5'
3H	1' 9"	6'
4H-8'	2' 6"	8'
4H	2' 6"	9'
5H-11'	3' 3"	11'
5H	3' 3"	12'

View proof for Printed PVC Panels for A-Frame

This rule does not apply to Yakima project since it is only 2 panels wide

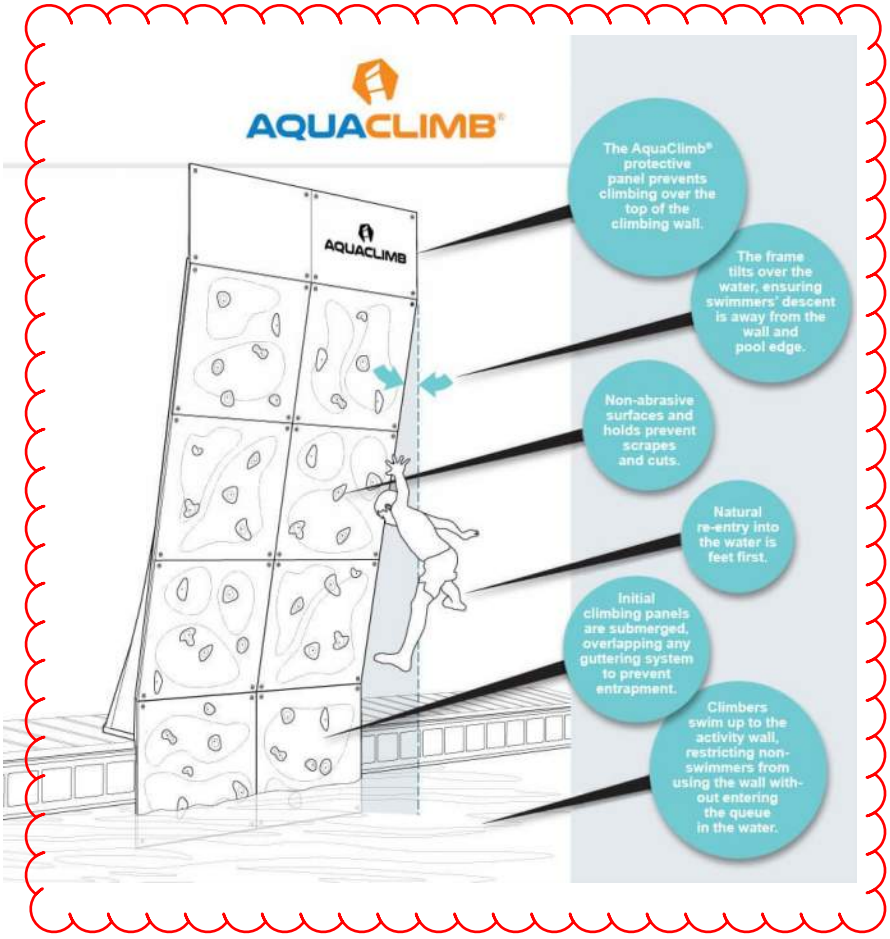


POOLSIDE ADVENTURES™

SAFETY

PROVIDE A SAFE WAY FOR POOL PLAY

AquaClimb® walls aren't just a fantastic poolside attraction. They're a safe way to play. They are specifically designed to eliminate the dangerous situations that can cause injury when sliding and diving. AquaClimb® is a safer alternative to diving boards and slides for both children and adults. Trust the brand that prioritizes you well being!



A SAFE WAY TO PLAY

- Each AquaClimb® comes complete with guidelines for safe use.
- AquaClimb® has clear protective panels to prevent climbers from climbing over the top of the wall.
- The AquaClimb® frame curves and hangs over the pool so that the natural re-entry into the water is feet first and the descent is away from the pool wall and edge.
- Non-abrasive surfaces and holds prevent scrapes and cuts.
- Natural re-entry into the water is feet first.
- Initial AquaClimb® climbing panels are submerged, overlapping any guttering system to prevent entrapment.
- Climbers swim up to the AquaClimb® activity wall, restricting non-swimmers from using the wall without entering the queue in the water.

Poolside Adventures products are recommended by the Aquatic Safety Research Group (ASRG) and are approved by state and

MEET OUR SAFETY TEAM

DR. TOM GRIFFITHS

Dr. Tom Griffiths is the President and Founder of Aquatic Safety Research Group, LLC. Recognized as an international leader in water safety, he has spent 38 years teaching, coaching and managing aquatics at three major universities. Griffiths has produced videos, textbooks, articles, and presentations in

local health departments throughout the USA, in addition to major health and safety organizations like PlaySafe LLC, a member of the International Play Equipment Manufacturers Association.

AquaClimbs are designed and engineered to the following standards:

- AISC Manual of Steel Construction, 15 th Edition, ASD
- IBC 2018
- ASCE/SEI 7-16
- ASTM F24/F2291- 21- Standard Practice for Design of Amusement Rides and Devices
- ASTM F2461-20 Aquatic Play Equipment
- European Standards EN17164 – Climbing Walls for Use in the Water Area

AquaZip'Ns are designed and engineered to the following standards:

- ASTM F2291-18 Amusement Rides and Devices
- ASTM F2461-18 Aquatic Play Equipment

CHECK OUT THESE ARTICLES ON THE BENEFITS OF ROCK CLIMBING FOR KIDS!

various areas of aquatics focusing his efforts on safety. He has also conducted hundreds of aquatic facility and beach inspections across the nation and abroad and teaches full day Aquatic Risk Management seminars. Perhaps his most significant contributions are the Five Minute Scanning Strategy©, Griff's Guard Stations©, Disappearing Dummies, his research on Shallow Water Blackout, and the National Note & Float program. He has been an aquatic safety expert for more than 40 years and shares his knowledge, expertise, and experience worldwide. Griffiths just released the 3rd

Why Rock Climbing is Such an Awesome Activity For Kids

5 Mental Health Benefits of Rock Climbing

Poolside Adventures stands on a history of providing a safe climbing experience. The recommended rules provided on our signage and advised during the sales and acquisition process are extremely important to operating a safe and fun activity for all.

We have recently viewed four YouTube videos which show our walls not being properly supervised, having the safe operation signage being displayed at the wall and the wall itself being used in a potentially unsafe manner. Though no accidents have been reported we strongly ask that all facilities please review the safe operation signage with staff and follow our guidelines.

Thank you!

edition of the popular The Complete Swimming Pool Reference.

Read Dr. Tom Griffiths 10-Year Review of the AquaClimb (PDF)

RACHEL GRIFFITHS

Rachel Griffiths, M.A. is the Communication Director for Aquatic Safety Research Group. Rachel conducts water safety research to help prevent drowning and provides water safety education to the public. She is also the President of Note and Float Life Jacket Fund,

AQUATIC SAFETY
Research Group

We Take Water Safety Seriously

DATE: April 9, 2015
TO: Laura Grandner
FROM: Dr. Tom Griffiths
RE: AquaClimb

Ten Year Review

As you know, nearly ten years ago, we placed an AquaClimb climbing wall in the diving well on the Penn State University Campus to test and analyze your product. I was pleased to learn how attractive it was to our students, and how it promoted fun and fitness in the pool with a new and exciting activity that was safe.

Since that time, Rachel and I have inspected hundreds of aquatic facilities and discovered that AquaClimb Walls are a safer alternative to many other poolside recreational products, primarily because swimmers do not have to climb a ladder in a wet environment over a concrete swimming pool deck. Because AquaClimb is accessed from the water inside the swimming pool, rather the swimming pool deck, there is very little chance of a child falling and hitting the deck. Further, the AquaClimb is angled out over the water, and as a result it is very improbable, if not impossible, that a child can fall to the deck.

As an expert witness in courts of law, I see many horrific accidents involving diving boards and slides, but I have never heard of an accident of any kind, minor or major, involving an AquaClimb. As we travel around this country and abroad teaching our full day Aquatic Risk Management Seminars, promoting AquaClimb as a safe, fun, and fitness alternative to other pool products is an essential part of our program. As you recall, AquaClimb is particularly valuable as a replacement for diving boards which no longer meet the depth and distance requirement or because of inadequate protective railings. I might also add that I have never seen a pool product installed as quickly in a swimming pool as an AquaClimb. I truly believe in your product and remain available to answer any questions you and others may have concerning AquaClimb Climbing Walls.

1632 Glenwood Circle, State College, PA 16803 **T** 814.883.0058 **F** 814.234.0313 TomGriffiths@AquaticSafetyGroup.com
AquaticSafetyGroup.com

Regards,

Tom Griffiths
President and Founder
Aquatic Safety Research Group, LLC

Rachel Griffiths
Communication Director
Aquatic Safety Research Group, LLC

[redacted]

I. INTRODUCTION

The AquaClimb is an exciting new recreational and fitness component that offers new programming opportunities to aquatic facilities. Because the AquaClimb extends below the surface of the water, participants can easily swim up to the climbing wall and begin to traverse it without leaving the pool itself. Even those individuals without use of their legs can utilize the AquaClimb to exercise the upper body in a fun, challenging, and non-threatening way. Perhaps the most meritorious application of the AquaClimb is an alternative to a diving board in a swimming pool which no longer meets safe diving depth and distance requirements.

Climbers who fall from the AquaClimb will enter the water feet-first. To enter the water head-first from the climbing wall structure is almost a biomechanical impossibility. Prior to purchasing and installing an AquaClimb, aquatic facilities should contact their local regulatory agency (e.g. Health Department) to determine whether regulations, recommendations or suggestions regarding the safe installation and use of the AquaClimb exist. **AQUATIC SAFETY RESEARCH GROUP, LLC**, an independent and objective water safety consultant firm, remains available to assist facilities in answering questions concerning the safe use of the AquaClimb.

II. STANDARD OPERATING PROCEDURES

A. LIFEGUARDS

Whenever the AquaClimb is in use, it is recommended that a properly trained and certified lifeguard be assigned exclusively to the AquaClimb. The lifeguard should be strategically placed to supervise and control use of the structure and to minimize climber

misbehavior. Because the apparatus will be positioned in deep water, a lifeguard with deep water skills and qualifications is needed. This lifeguard must also be trained for the proper use and monitoring of the in-water climbing structure. The lifeguard should be positioned close to the wall with a full and unobstructed view of the climbing wall and drop zone, with the ability to see underwater in the drop zone. The lifeguard must stay focused on the climbing wall whenever in use and attention should not be diverted to other areas of the pool. Lifeguard orientations, in-service trainings and emergency action plans should include the AquaClimb and should be reviewed and practiced regularly but at least monthly. In many pools, the best vantage point for proper surveillance may be directly across the pool facing the wall. However, each facility should determine where to best position supervisory staff to ensure a full and unobstructed view of the climbing wall and the drop zone.

The aquatic facility should also establish an entrance and exit pattern (left to right and right to left) to avoid congestion of swimmers waiting to swim into the drop zone to begin their ascent on the wall. This pattern can be changed daily or hourly. For larger installations allowing two or more climbers, additional safety precautions must be implemented to minimize the risk of a climber falling onto someone swimming into or out of the drop zone. One such approach is to direct climbers, once they have fallen from the wall, to swim to the closest edge of the drop zone so as to avoid swimming underneath a second climber.

B. DEPTH REQUIREMENTS

While most competitive swim agencies, including the National Collegiate Athletic Association (NCAA), require a minimum water depth of five (5) feet to dive headfirst from starting platforms, the AquaClimb, which promotes only feet-first entries, takes a more conservative approach, requiring a minimum water depth of five (5) feet for installation of its shortest three-panel wall. As panels are added vertically to the structure, minimum water depth requirements increase. To ensure safety of climbers, AquaClimb has applied commonly accepted safe head-first diving depths to feet-first entries from the structure.

We recognize that these depths are very conservative given that they are intended to minimize the risk of injury from head-first entries rather than from feet-first entries, but

[redacted] absent additional research we cannot safely recommend alternative water depths which deviate from these nationally-accepted standards.

MINIMUM DEPTH REQUIREMENTS FOR AQUACLIMB INSTALLATION			
Panel Height* - standard	3 panels (lowered)	4 panels (lowered)	5 panels (lowered)
Minimum Water Depth	5 feet	7 feet	8 feet

* Each panel measures approximately $3ft^2$ or $1m^2$

MINIMUM DEPTH REQUIREMENTS FOR AQUACLIMB INSTALLATION			
Panel Height* - standard	3 panels	4 panels	5 panels
Minimum Water Depth	6 feet	8 feet	9 feet

C. DECK CLEARANCES

Whenever possible, four feet of deck space should be maintained between the end of the support structure and the perimeter pool wall or fence. If less than four feet is available, a combination of pedestrian control stanchions and traffic cones should be used to direct patrons around the support system. To best accommodate persons with disabilities, a minimum of three feet (36”) clearance around the support structures should be maintained. Even with spacious decks, stanchions and cones always come highly recommended, as they minimize the risk of someone coming into contact with the structure. Customers are advised to check building and fire codes to determine whether support structures can permissibly block access to the pool deck, particularly in cases where the support structure would come within three feet of a wall.

[redacted]

D. NUMBER OF CLIMBERS

With a one panel or two panel wide AquaClimb, it is ***highly recommended*** that only one climber use the AquaClimb at a time. With a three panel or wider AquaClimb, however, there is an opportunity to allow more than one climber on the wall at the same time. Multiple climbers should only be allowed when there is no possibility of one climber either interfering with or falling on top of another climber. Multiple climbers should be instructed to climb the wall vertically rather than to traverse the wall horizontally. Climbers should also maintain a distance of at least one panel from other climbers to minimize the risk of climber interference, horseplay and accidental concurrent falls.

E. VERIFIED SWIMMERS ONLY

Because the AquaClimb is installed in deep water (see minimum depth requirements above), this climbing attraction is to be used only by "swimmers" – persons with verified swimming ability. The attractive colors and the fun activity that the structure provides, are likely to draw younger, weaker swimmers to the climbing wall. These persons should be properly screened to ensure they possess the requisite deep-water skills necessary for using the structure. Following standard aquatic safety practices, anyone wishing to enter deep water to use the AquaClimb should be given a swim test. A recommended swim test would be to have the swimmer/climber jump into ***chest-deep*** water, surface, swim the equivalent length of the buffer zone and return to the starting point. Requiring climbers to tread water for 30 – 60 seconds comes highly recommended. Swim tests should be conducted in chest-deep water to maximize swimmer safety.

[redacted]

F. DROP ZONE

Climbers will fall from the wall into the water. It is therefore imperative to keep people from entering the "drop zone" where they would risk being struck by a falling climber. No other swimmers should be allowed into the drop zone when a climber is on the wall.

3 panel high:

4 panel high:

5 panel high:

G. FEET-FIRST ENTRIES ONLY

While head-first entries, including dives, are improbable to perform from the face of the climbing wall, and although the depth requirements for the various climbing wall configurations are extremely safe and tend to be conservative, climbers must be warned that all entries into the water from the AquaClimb should be feet-first. Climbers who intentionally violate this safety rule should be prohibited from using the AquaClimb.

[redacted]

H. UNDERWATER ACTIVITIES

Participants should not be allowed to play with the structure itself, particularly while submerged. While there are no hidden hazards or entrapment potentials inherent in the AquaClimb, it is intended for above-water use. It is not intended or designed for underwater use by climbers. Playing underwater around the structure makes it more difficult for the lifeguard to properly supervise the activity. This could lead to injury should a climber fall onto someone who was playing underwater in the drop zone.

[redacted]

III. SUGGESTIONS FOR SAFETY SIGNAGE

Perhaps the most appropriate place to place caution/warning signs would be on the side. The three most important warnings should include:

- "Swimmers Only"
- "No Head First Entries"
- "Only One Climber at a Time unless there are 1-2 clear panel between climbers"

These three warnings can be placed together on the same sign in the appropriate colors (red/white, black/yellow, orange/black). Additional signs/warnings may be mounted on the rear of the support structure.

WASHINGTON STATE BOARD OF HEALTH

Date: August 7, 2024

To: Washington State Board of Health Members

From: Kate Dean, Board Member

Subject: Variance Request (Cheney) – Chapter 246-262-010(21) WAC, Definition of Diving Envelope, & 246-262-060(5)(vi) Diving envelope Requirements

Background and Summary:

RCW 70.90.120 authorizes the State Board of Health (Board) to adopt rules governing safety, sanitation, and water quality of water recreation facilities. WAC 246-262-160 sets the process for variance requests. The Board has the sole discretion to approve variance requests, if the Board determines the data and our research provides sufficient evidence that the variance will adequately protect public health and safety. Upon receipt of a request, the Board will strive to make a determination on the variance request within sixty days.

On July 17, 2024, the Board received a variance request from Brooke Hanley requesting the approval of 3 separate pieces of equipment under sections 246-262-010(21) WAC, definition of diving envelope, & 246-262-060(5)(vi) diving envelope requirements. The equipment includes a NinjaCross Obstacle Course, AquaZip'n Rope Swing, and a climbing wall.

Today, Board and Department of Health staff will introduce the variance requests for consideration by the Board. Due to the large size of supporting documentation, staff need additional time to complete the review and to determine compliance with the health and safety standards as outlined in Chapter 246-262 WAC.

Staff recommends that the Board hold a special meeting in late August to allow for additional time to review the requests and to provide a timely response to the requester.

Recommended Board Actions

This is an information briefing.

Staff

Andrew Kamali

PO Box 47990 • Olympia, WA 98504-7990
360-236-4110 • wsboh@sboh.wa.gov • sboh.wa.gov

Washington State Board of Health
Policy & Procedure

Policy Number:	**2018-001**
Subject:	**Handling Variances, Exemptions, and Waivers in State Board of Health Rules**
Approved Date:	**August 8, 2018**

Background

The State Board of Health (Board) has broad authority to adopt rules on a number of public health and safety topics. These rules may include provisions regarding variances, exemptions, or waivers allowed under the rules, which may be granted by the Washington Department of Health (Department), local health jurisdictions, or the Board.

Variances, exemptions, and waivers are different types of exceptions that support flexible and reasonable application of Board rules depending on the particular situation. The terms are not defined in the regulations referenced below, but the general dictionary definitions of these words can be used to understand the distinctions between them:

- **Variance** means a modified means of meeting a rule requirement.
- **Exemption** means relief from a rule requirement.
- **Waiver** means the setting aside of a rule requirement.

As outlined in Table 1 of this policy, one or more of these exception provisions are used in twelve Board rules. In addition, state rules on reclaimed water administered by the Washington Department of Ecology reference Board waiver authority in chapter 246-290 WAC, *Group A Public Water Supplies*, for approval of direct potable reuse of reclaimed water.

In most cases, authority to grant exceptions is assigned to the Department, local board of health, or local health officer. Only three rules directly involve the Board. Two rules assign decision-making authority to the Board and a third provides the Board with optional approval authority:

1) WAC 246-262-160: Authorizes the Board to act on variance requests to requirements of chapter 246-262 WAC, *Recreational Water Contact Facilities*.
2) WAC 246-290-060: Authorizes the Board to act on requests for variances, exemptions, or waivers to requirements of chapter 246-290 WAC, *Group A Public Water Supplies*.
3) WAC 246-260-201: Authorizes the Department or local health officer to act on variance requests to requirements of chapter 246-260 WAC, *Water Recreation Facilities*. However, the Board may require that variance requests be submitted for Board review and approval.

Policy Statement

Variances, exemptions, and waivers are valuable tools in Board rules. The Board plays a limited role directly granting such exceptions in implementing the rules. Where required in rules, the Board will consider requests for variances, exemptions, and waivers under the procedure outlined below.

New or revised Board rules can help refine the Board's limited role granting these exceptions and help align provisions for variances, exemptions, and waivers across Board rules. The following should be taken into consideration as Board rules containing these provisions are next updated:

- Variances, exemptions, and waivers should be clearly defined and correctly applied in all Board rules.
- Approval authority for variances, exemptions, and waivers should rest with the health agency where it best protects public health and safety, ensures accountability, and is most easily administered.
- Unless it provides needed flexibility, rules granting variances, exemptions, or waivers should avoid listing multiple or optional approval authorities and should instead authorize one agency.
- For ease of administration, rules authorizing local health jurisdictions to approve variances, exemptions, or waivers should identify local health officers rather than local boards of health as the approval authority.
- Provisions in chapter 246-260 WAC and chapter 246-262 WAC should be aligned—or combined if the rules are consolidated—and should assign approval authority to either the Department or local health officer.
- Where meaningful, annual reporting to the Board on activity related to variances, exemptions, and waivers can be required. If required, such reporting should occur consistently.

Board Procedure

Where required in rule, the Board will consider requests for variances, exemptions, and waivers. As noted previously, two rules require Board action: chapter 246-262 WAC, *Recreational Water Contact Facilities*, and chapter 246-290 WAC, *Group A Public Water Supplies*. Chapter 246-262 WAC lacks any process requirements, so the following procedures apply in full. In contrast, WAC 246-290-060 and Policy J.28 of the Department's Office of Drinking Water outline a few process requirements that should be applied to dovetail with Board process requirements starting at the point of application to the Department. Variance and exemption requests under WAC 246-290-060 must be considered in accordance with 40 CFR s. 141.4 (variances and exemptions to National Primary Drinking Water Regulations).

Submittal of Requests

Requests should be addressed to the Board Chair and signed by an authorized agent of the owner/operator of the facility or utility (not a third-party agent). With applications to the Department of Health under WAC 246-290-060, the Board Chair should be copied. The request should include and describe the following:

- name and address of the facility or utility, name of the owner/operator, and name and information for the lead contact;
- rule citation authorizing Board action;
- the specific rule or rules for which a variance, exemption, or waiver is sought;
- the situation, need, and justification for the request;
- supporting documentation and technical analysis developed or used to assess the request and meet the intent of the regulation to ensure health and safety;
- steps taken to mitigate concerns or risks; and
- commitment to carry out conditions or follow–up actions that may be applied to the request.

Receipt and Notification

Upon receipt of a request, Board staff, in consultation with the Executive Director, will respond to the requester within five business days acknowledging receipt of the request. The Executive Director or staff will notify Board members that a request has been received and will be brought to the Board for consideration at the next regularly scheduled Board meeting. The Board will strive to complete its

work and respond to a request within 60 days. If no regular meeting is scheduled within 60 days of receipt, or if the agenda for the regular meeting cannot accommodate review of the request, or if staff need more time to complete its review, the request may be addressed at the following Board meeting. The Executive Director or staff will notify the requester of dates and times that the Board is scheduled to meet and consider the request. As part of its initial review, the Board will determine whether a request falls within its authority to review. If the Board determines that a request falls outside the scope of its authority, staff will notify the requester of this and close the request.

Review and Board Action

The Board may identify a sponsoring Board member and will direct staff to review the request on the basis of relevant laws, industry standards, health and safety guidelines, and other relevant material. Board staff will coordinate and consult with the Department and other subject matter experts as appropriate in reviewing the request.

The sponsor and Board staff assigned to review the request will present their findings and recommendation to the Board. The Board may ask a Department representative to provide a recommendation or technical analysis to help inform Board discussions. The Board may invite the requester to present the request and respond to questions from the Board at its meeting.

Following review, the Board may grant the request, grant the request with conditions, deny the request, or ask for additional information before acting on the request. The Board may grant a variance, exemption, or waiver from rule requirements if it meets the substantive requirements of the rule allowing a variance, exemption, or waiver. Variances and exemptions granted to public water systems must be conditioned on a compliance schedule in accordance with WAC 246-290-060(6). The decision will be made by the Board in public meeting. Once the Board has made its decision, Board staff will follow up with a written notice to the requester. If the Board denies a request, the notice will contain information about how the requester may appeal the decision.

Water Recreation Variance Request, Cheney Aquatic Center

Chapter 246-262 WAC

Andrew Kamali, Policy Advisor - August 7, 2024

WASHINGTON STATE BOARD OF HEALTH

David DeLong,

Water Recreation Program Lead

Cheney Aquatic Center Variances

- Purpose
- Review variance requirements
- Cheney Variance Request(s)
  - Aqua Climb Climbing Wall
  - Aqua Zip’N Rope Swing
  - Ninja Cross Obstacle Course

Background

WAC 246-262-160

Variance.

The board may grant a variance from requirements of chapter 246-262 WAC if, in the sole discretion of the board, data and/or research provides sufficient evidence that the recreational water contact facility (attraction, device, equipment, procedure, etc.), will adequately protect public health and safety, as well as water quality.

Background

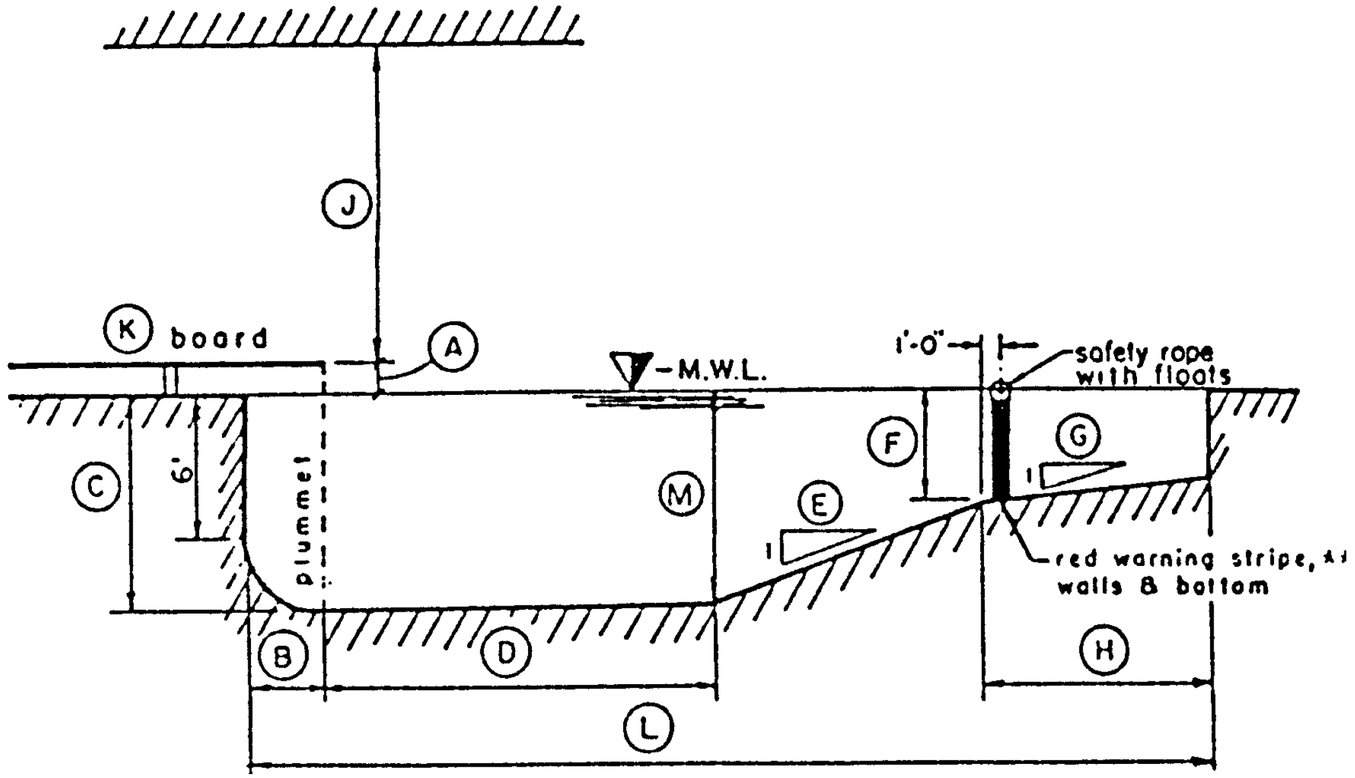
- **WAC 246-262-010(21)** "Diving envelope" means the minimum dimensions of an area within the pool necessary to provide entry from a diving board, platform, or attraction segment where users enter above pool water level.

- **WAC 246-262-060(5)(vi)** *describes diving envelope requirements*

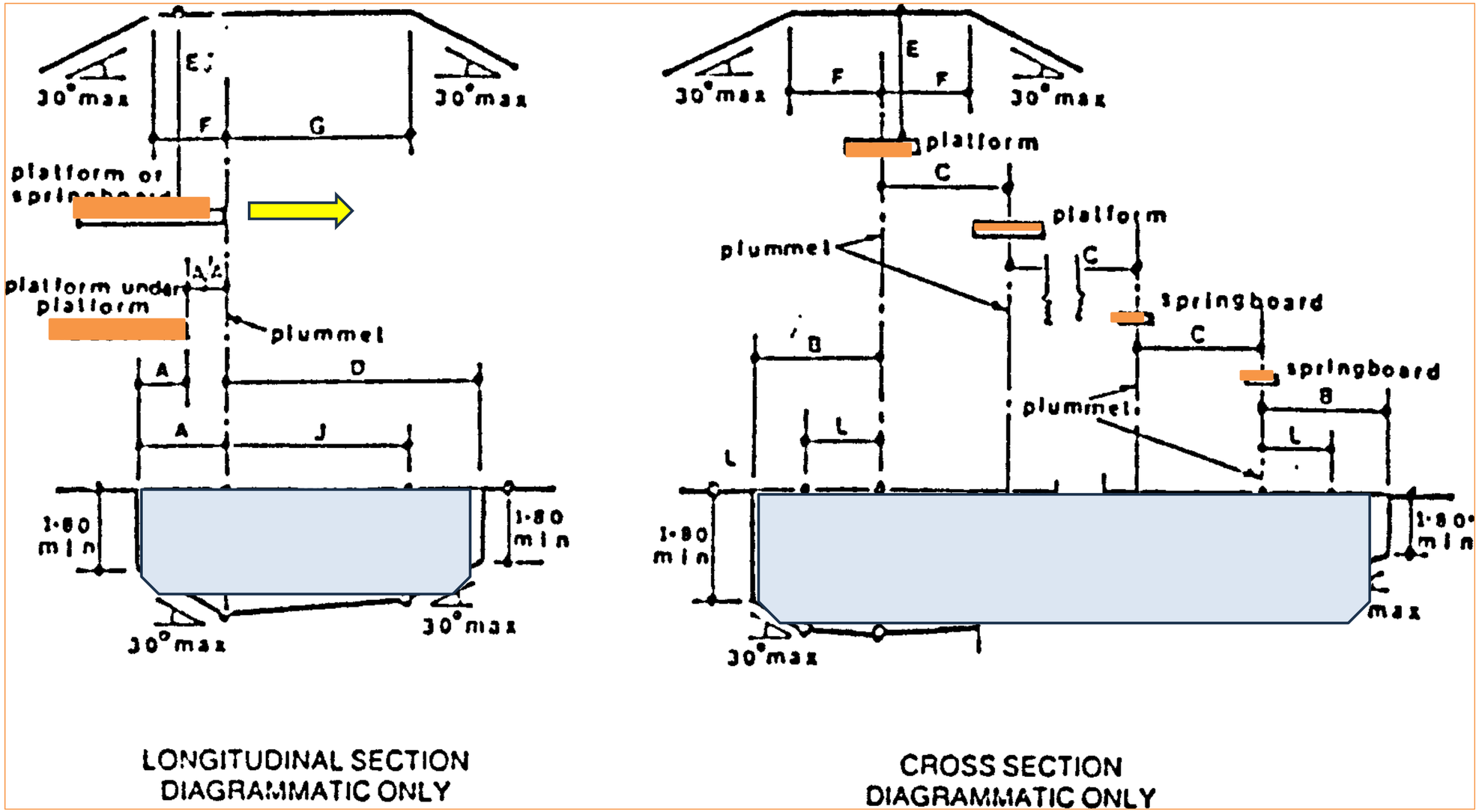
Diving Envelopes

Non-competitive Diving

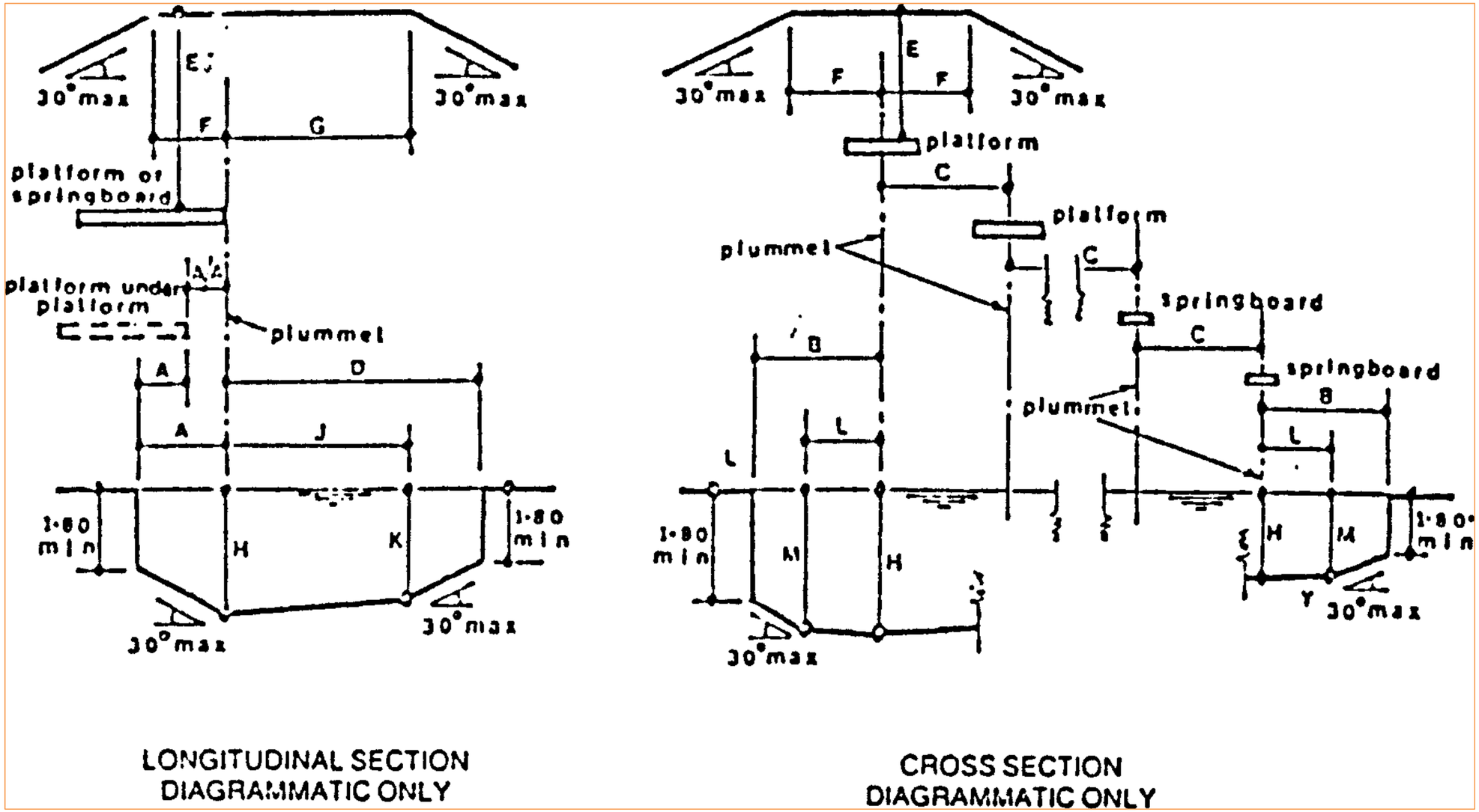
- Council for National Cooperation in Aquatics Standards
- 20-inch board height
- Depth at plummet 9-10 feet
- Length 16 feet



Competitive Diving Envelopes

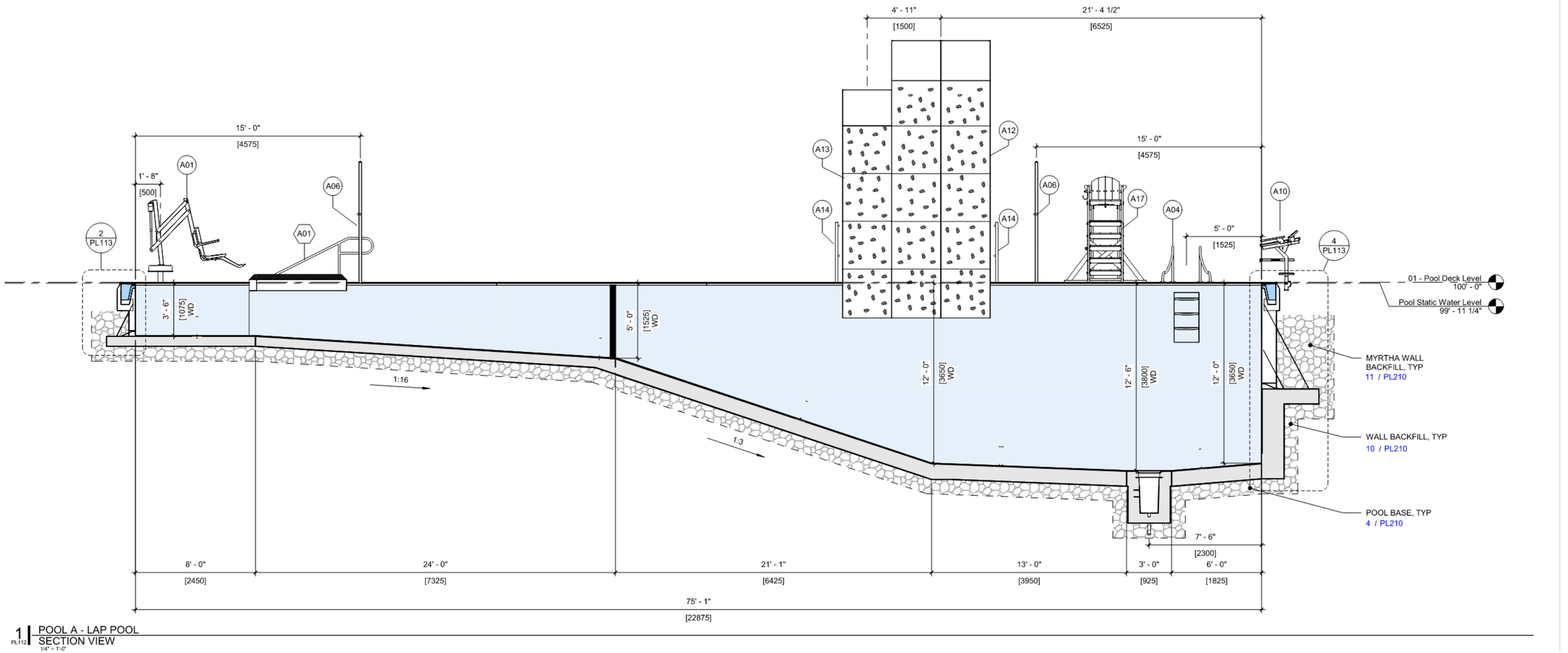


Competitive Diving Envelopes



Cheney Pool Climbing Wall

Cheney Pool – Climbing Wall



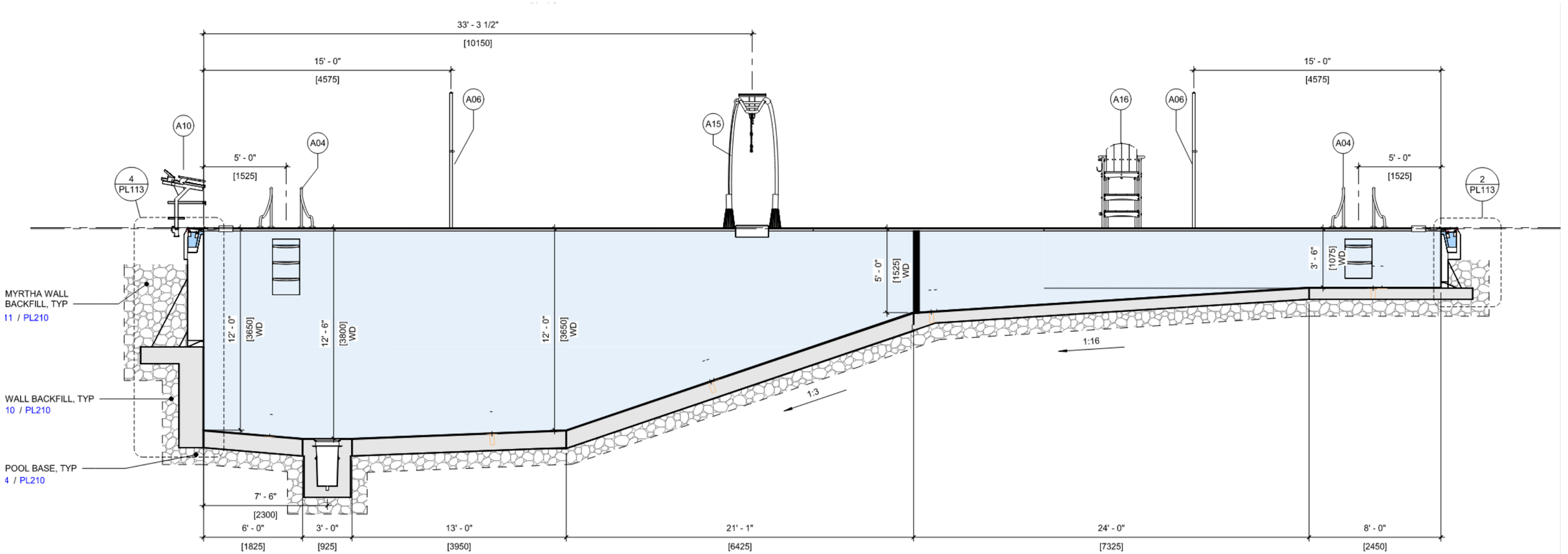
1 PL112 POOL A - LAP POOL
SECTION VIEW
1/4" = 1'-0"

Cheney Pool
Aqua Zip

AQUAZIP'N®

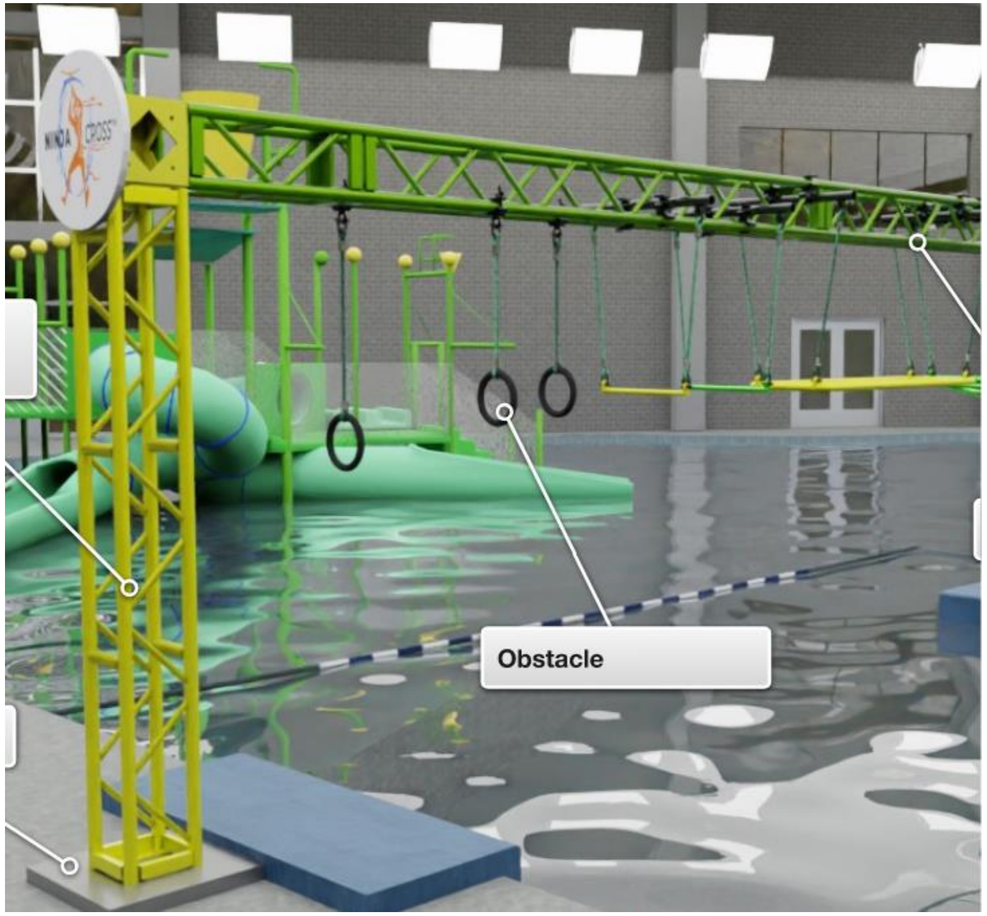
Combining the thrill of a zip line with the fun of a rope swing

Cheney Pool – Aqua Zip



3 PL112 POOL A - LAP POOL
SECTION VIEW
1/4" = 1'-0"

Cheney Pool
Ninja Cross



Cheney Pool Variances Status

- Department of Health, State Board of Health, and Spokane County Regional Health District staff are still reviewing the data, arguments, and mitigations proposed by the facility and are not prepared to give an evaluation of or recommendation to the Board.

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

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- 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:
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We will make every effort to provide you the information requested and correct any compliance issues on our website.

NAC
ARCHITECTURE

Patty Hayes, Board Chair
Washington State Board of Health
PO Box 47990
Olympia, WA 98504-7990

CHENEY AQUATIC CENTER Variance Letter Date: 2024.06.25

PROJECT IDENTIFICATION: Lap Pool #: SR009200 Leisure Pool #: SR009201

On Behalf of:
Cheney Aquatic Center, City of Cheney
Owner Contact: Dan Curley Phone: 509-498-9293
Owner Address: 609 2nd Street Cheney, WA 99004
Facility Address: 115 North 8th Street (formerly 711 Cedar Street), Cheney, WA 99004

Owner Representative: Brooke Hanley (NAC Architecture) 509-838-8240

Variance Request Contact:
NAC Architecture: Brooke Hanley Phone: 509-838-8240 Email: bhanley@nacarchitecture.com

Facility Information:
Cheney Aquatic Center - Project includes an outdoor 6-lane 25-yard lap pool & separate leisure pool with zero-entry, spray features, & lazy river. The pool building with locker rooms, lifeguard offices, party room, and mechanical spaces is about 5000sf. The entire facility is lifeguarded and enclosed securely.

Plan Submittal: Drawing Plans have been submitted for review.

Variance Request Citation:
WAC 246-262-160 states *the board may grant a variance from requirements of chapter **246-262** WAC if, in the sole discretion of the board, data and/or research provides sufficient evidence that the RWCF (attraction, device, equipment, procedure, etc.), will adequately protect public health and safety, as well as water quality.*

Variance Request: Code Related to Diving Envelope (WAC 246-262-010(21) & WAC 246-262-060(5)(vi)) for a **climbing wall** attraction.

Items noted in review letter include:

- **Climbing wall** attraction receiving pool shall meet the 2000-2001 FINA facility rules (depth application and setbacks)

In the Spokane Regional Health District review response issued by Steve Main dated May 24, 2024, Steve requests NAC Architecture (NAC) and WaterTechnology, Inc. (WTI) address important concerns regarding public safety related to the receiving pool for the proposed **climbing wall** attraction in Pool B. The

concern is to address the minimum depth of the pool to be compliant with the WAC 246-262-010(21) & WAC 246-262-060(5)(c)(vi) regarding diving envelopes for features where users enter the water at 20" or higher above the water surface.

On behalf of the City of Cheney; NAC & WTI respectfully request your consideration of the current pool depth design at the climbing wall for the future Cheney Aquatic Center. To support this request we provide the attached information, engineering exhibits, and following commentary:

- The review letter states that the "diving envelope" from WAC 246-262-010(21) applies to **all attractions** where users enter above pool water level and therefore requires the CNCA (enter less than 20" above the water surface) or FINA (enter 20" or greater above the water surface) water depths. We submit that the attached engineering calculations for the **AquaClimb 5-Panel-High & 5-Panel-High-Alt climbing wall** products will demonstrate that the manufacturer's required water depths and the designed water depths provided at the Cheney Aquatic Center are sufficient to protect the safety of the range of users allowed to participate in this attraction. Calculations were completed for a 48" tall, 50lbs person and a 78" tall, 250lbs person to show a range of sizes requested in the review letter. Please reference page 9 for the manufacturer's minimum depth requirements and pages 10-17 for the engineering calculations and associated notes. The Cheney design provides for greater water depth than the minimum required by this engineering report as noted in the attached information. Please review the attached data in support of using the manufacturer's depth requirements in lieu of the CNCA or FINA diving envelope dimensions.

- WAC 246-262-060(5)(c)(vi) appears to apply specifically to "diving envelopes in pools or areas of pools designated for diving activities". The applicant submits that diving activities are generally defined as plunging into the water headfirst. Diving headfirst into water results in the need for deeper water to avoid a head & neck collision with the bottom of the pool which is different than a feet-first or tucked entry plunge where the body is significantly slowed in the first two feet of water. The **climbing wall** safety guidelines and standard operating procedures (provided in the exhibits) will note that users are required to re-enter the water in a feet-first manner. Diving from the unit is prohibited (and per the manufacturer data, bio-mechanically improbable). The engineering calculations completed also assume a feet-first plummet into the water.

- The Model Aquatic Health Code also addresses the complexity of "other aquatic features" like **climbing walls** and would suggest that the manufacturer recommendations for design and operation would be adequate to install the feature.

  4.12.10[A] Other Aquatic Features Other AQUATIC FEATURES not otherwise addressed in the CODE, including but not limited to climbing walls, inflatables, and play structures, shall not be installed unless designed and operated in accordance with all manufacturer's installation and operations recommendations.

- 'A-frame' signs with all written safety guidelines will be publicly displayed near the **climbing wall** (see page 18 for example) to meet the criteria of WAC 246-262-070(10). The design

team could also instruct AquaClimb to add a maximum height of 78" to the sign to correspond to the engineering calculations, if this would mitigate concerns over swimmers participating that do not fit within the engineering assumptions.

- See attached climbing wall diagram. The frame and panels of the wall tilt out over the water, ensuring the swimmer's descent is away from the wall and pool edge. The protective panels at the top do not have hand-holds and therefore prevents climbing over the top of the structure. The "Alt" panel climbing wall does not provide hand holds as high as the full 5 panel system and therefore requires less minimum water depth per the manufacturer's recommendations.

- This pool will be lifeguarded at all times while in operation and the lifeguard staff will be the first line of defense to screen bathers to make sure they are experienced swimmers, instruct swimmers on proper use of the attraction, and direct proper swimmer circulation to and from the activity within the pool to avoid congestion or collisions. The **climbing wall** will have a dedicated lifeguard to closely supervise the safety of swimmers when the attraction is open for use. Cheney is dedicated to making this facility fun while also as safe as possible for their community members and patrons.

- The product literature, research paper, and testing tout the relative safety of the **climbing wall** compared to diving boards and slides. They also have over 1,000 installations across the world. See the provided letter from Aquatic Safety Research Group.

- The **AquaClimb** has also been designed and engineered to meet the following standards:
  - ASTM F24/F2291-21 Standard Practice for Design of Amusement Rides and Devices
  - ASTM F2461-20 Aquatic Play Equipment
  - European Standards EN17164 – Climbing walls for use in the water area
  - IBC 2018 & AISC Manual of Steel Construction
  - Other industry standards listed in the product data attached

- NAC submits that the design as described above and substantiated in the attached documentation meets the intent of providing a safe receiving pool for the **climbing wall** feature. NAC, WTI, and the City of Cheney respectfully requests a variance accordingly. If the State Board of Health has any follow-up conditions or actions required of the owner/operator, we are committed to reviewing them for implementation.

NAC Architecture (NAC) has teamed with Water Technology (WTI) on numerous aquatic projects and so we have a history of producing these projects successfully. WTI has been designing Aquatic venues for over 40 years. WTI is widely known in the industry as one of the leading aquatic design firms in North America. As one of the industry's leaders, WTI has represented the waterpark industry during CPSC meetings on review of VGB rules and has also been involved in reviewing/editing sections of the MAHC. They are also represented in the Washington DOH committee to update the existing administrative code to adopt a more comprehensive aquatic code like the MAHC. The NAC and WTI commitment to safe aquatic facilities is proven. The design of the receiving pool at the **climbing wall** for the Cheney Aquatic

Center will not put the health and safety of the public at risk. The City of Cheney, having operated a public pool for many years is experienced and committed to the safety and the welfare of their patrons.

On behalf of the City of Cheney, NAC Architecture would like to thank you for your consideration of this Variance Request. Please feel free to contact me with any questions you may have regarding this request.

Thank you,

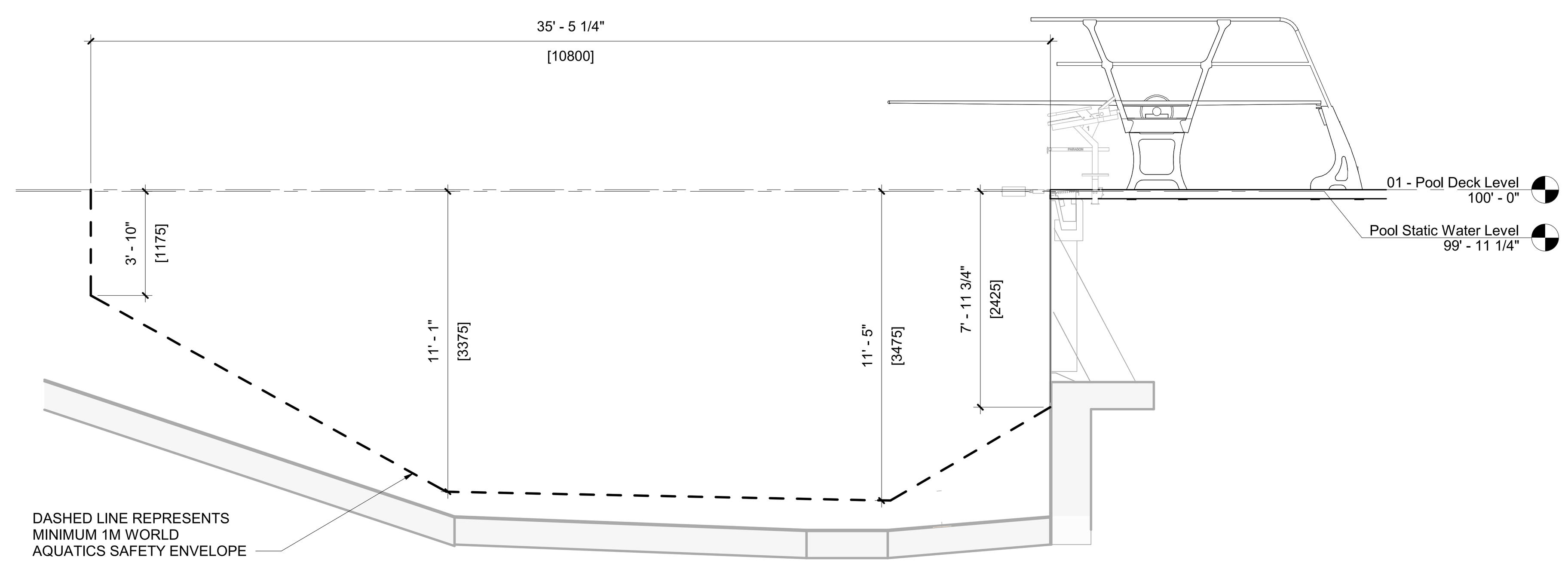
Brooke Hanley

Brooke Hanley, AIA, Principal Architect, NAC Architecture

Attachments:

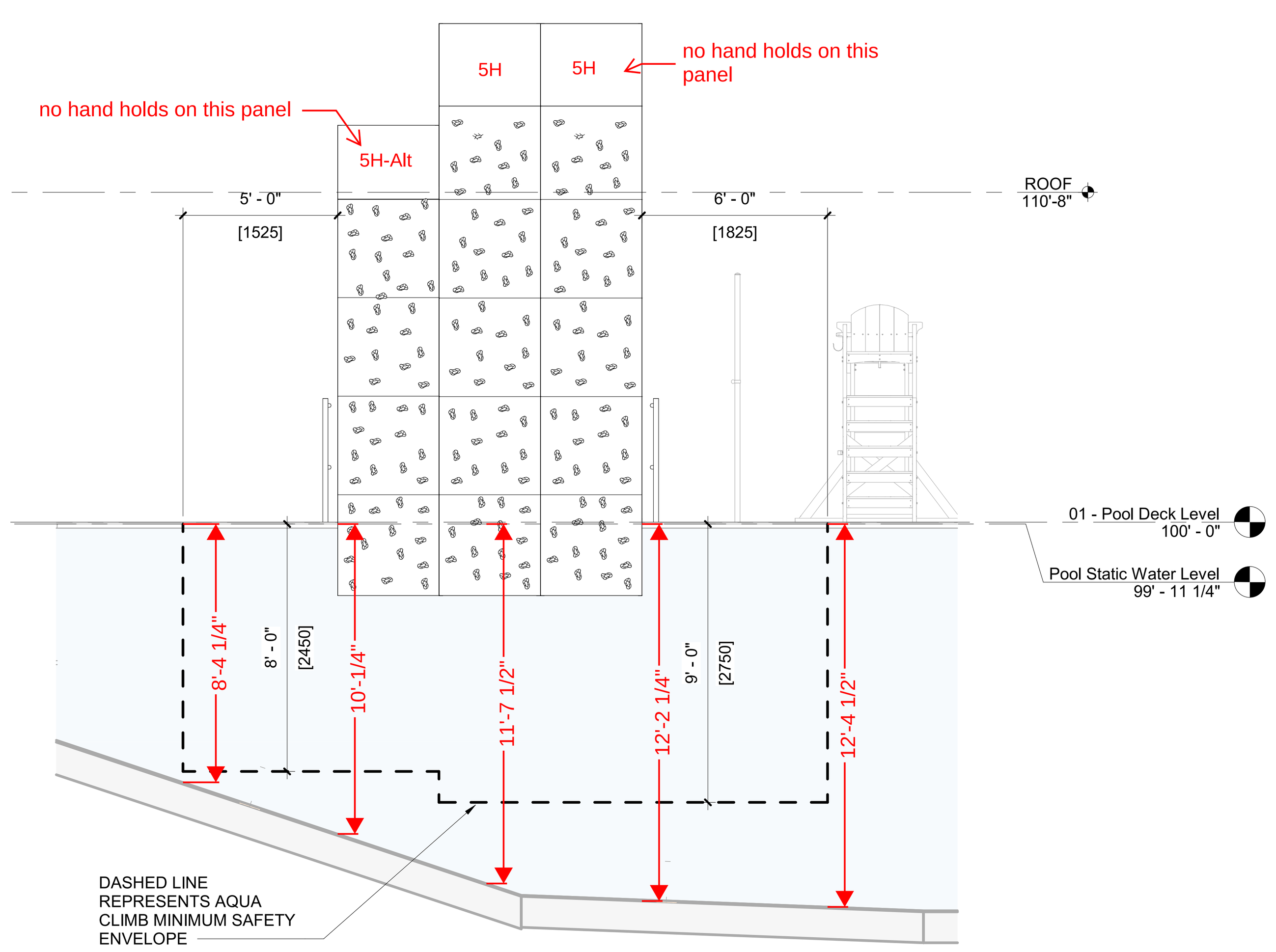
- AquaClimb Safety and Fall Zone Engineering, including a floor plan and section of the receiving pool as designed for the Cheney Aquatic Center.

NOTE:
ALL LANE DIVIDERS AND BACKSTROKE FLAGS MUST
BE REMOVED WHILE DIVING BOARD IS IN USE.



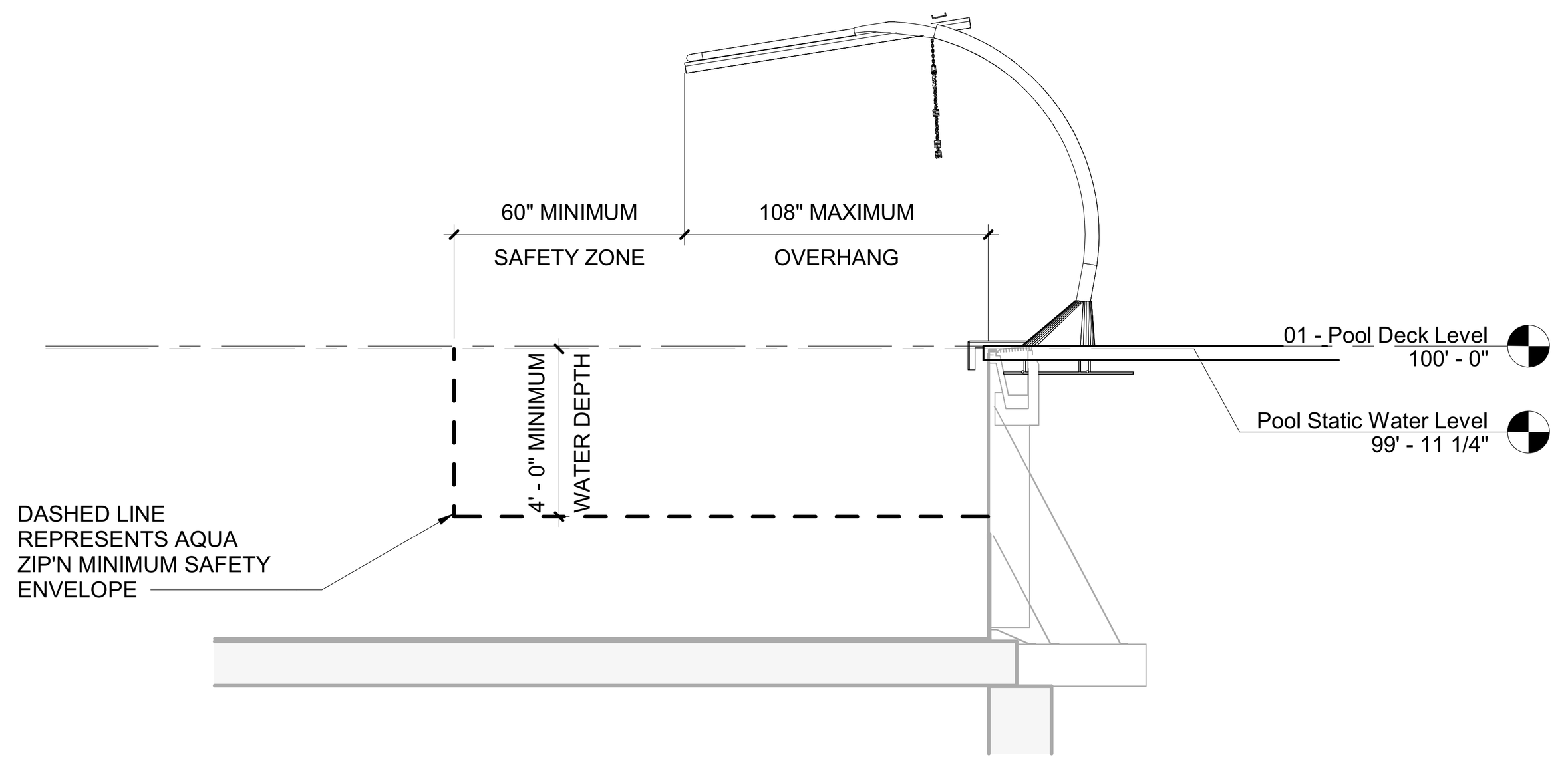
2 POOL A - LAP POOL DIVING SAFETY ENVELOPE
PL115 SECTION VIEW
1/4" = 1'-0"

NOTE:
ALL LANE DIVIDERS AND BACKSTROKE FLAGS MUST
BE REMOVED WHILE CLIMBING WALLS ARE IN USE.



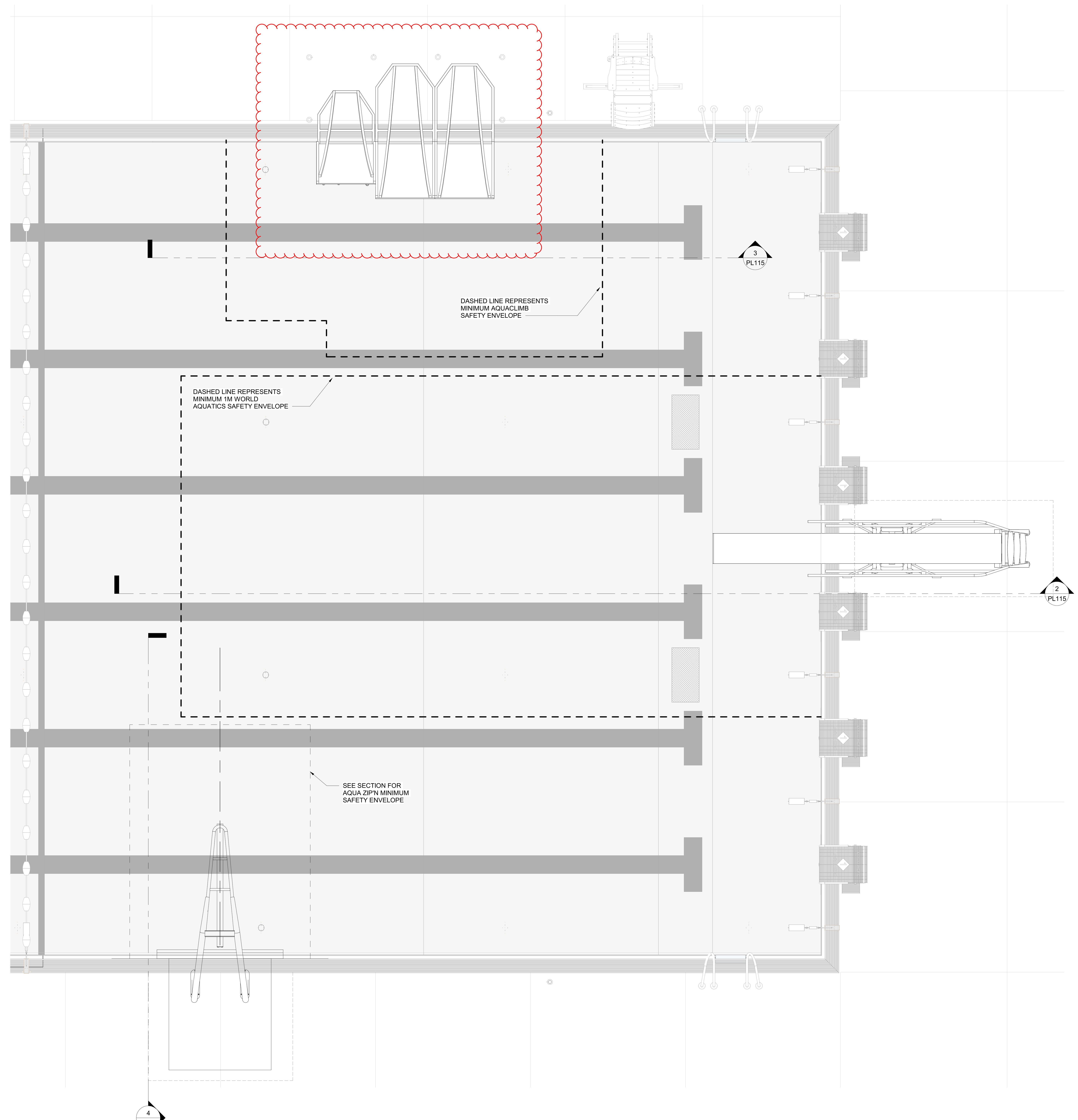
3 POOL A - LAP POOL CLIMBING WALL SAFETY ENVELOPE
PL115 SECTION VIEW
1/4" = 1'-0"

NOTE:
ALL LANE DIVIDERS AND BACKSTROKE FLAGS
MUST BE REMOVED WHILE AQUA ZIP'N IS IN USE.



4 POOL A - LAP POOL AQUA ZIP'N SAFETY ENVELOPE
PL115 SECTION VIEW
1/4" = 1'-0"

NOTE:
ALL LANE DIVIDERS AND BACKSTROKE FLAGS MUST BE
REMOVED WHILE CLIMBING WALLS, DIVING BOARD AND
AQUA ZIP'N ARE IN USE.



1 POOL A - LAP POOL SAFETY ENVELOPES
PL115 PLAN VIEW
3/8" = 1'-0"

REVISIONS

BID SET

WTI WATER TECHNOLOGY INC
World Leaders in Aquatic Planning, Design and Engineering
100 Park Avenue | Beaver Dam, WI 53916
| 920.887.7375 | #23458

CITY OF CHENEY
CHENEY AQUATIC CENTER
711 CEDAR ST, CHENEY, WA 99004

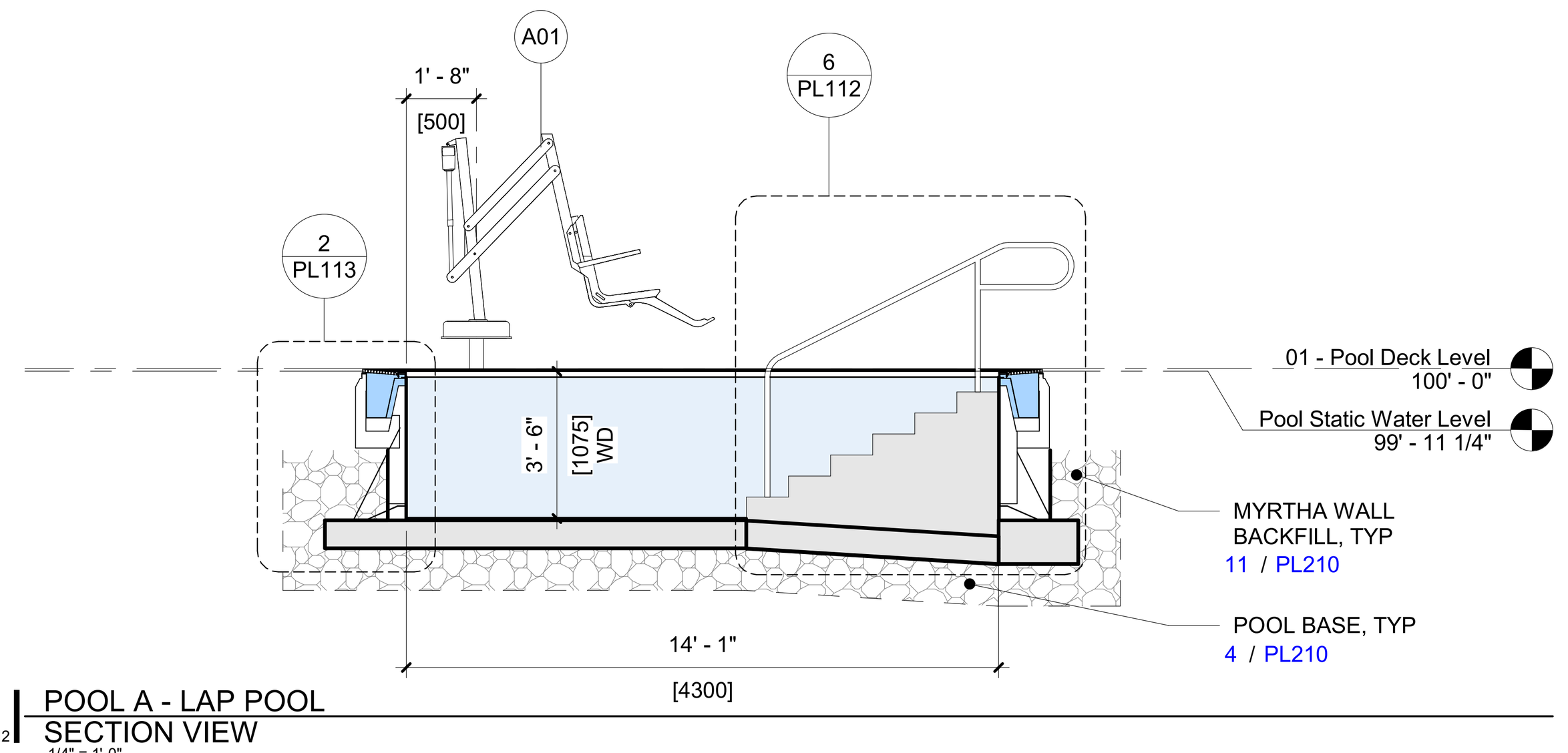
REGISTERED ARCHITECT
MATTHEW W. FRISBY
STATE OF WASHINGTON
4/16/2024

NAC
ARCHITECTURE
nacarchitecture.com
1203 WEST RIVERSIDE AVE
SPOKANE WA 99201
P:509.838.8240

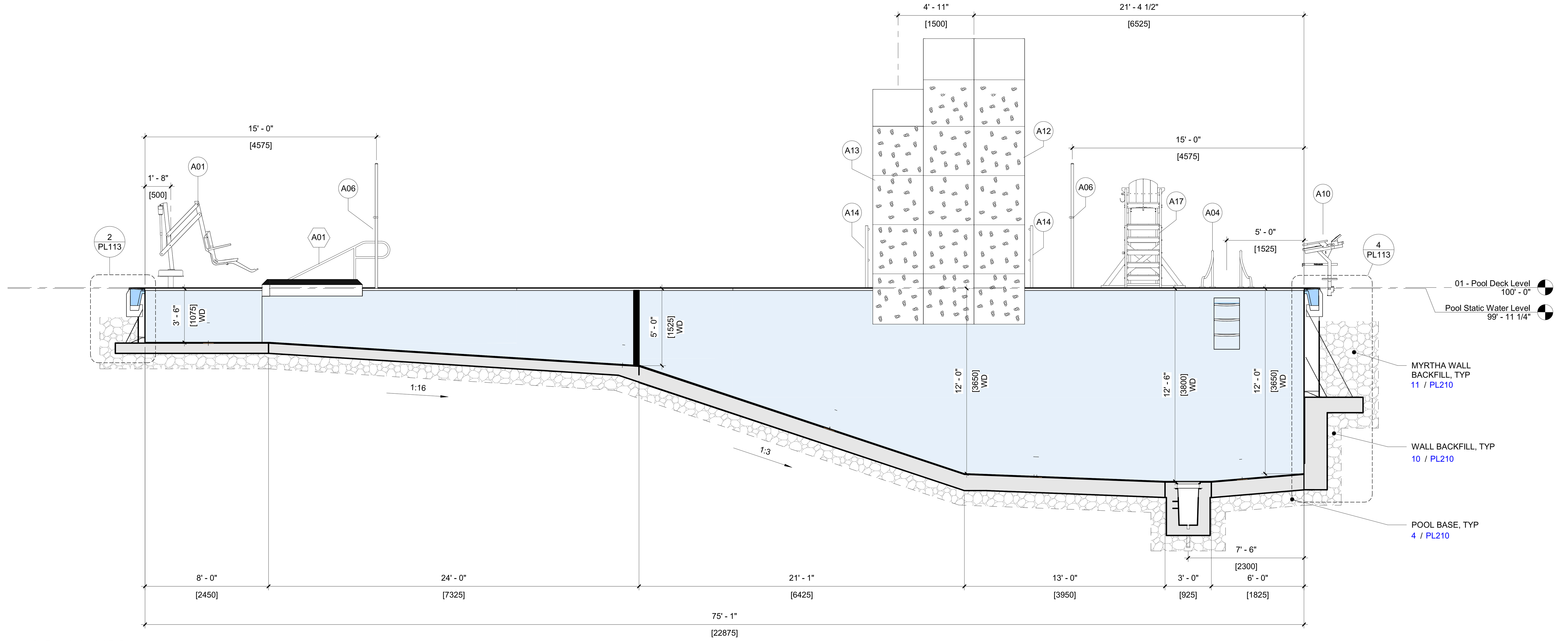
NAC NO	23458
DRAWN	MJC
CHECKED	GGA
DATE	04/16/2024

POOL A - LAP POOL
SAFETY
ENVELOPES

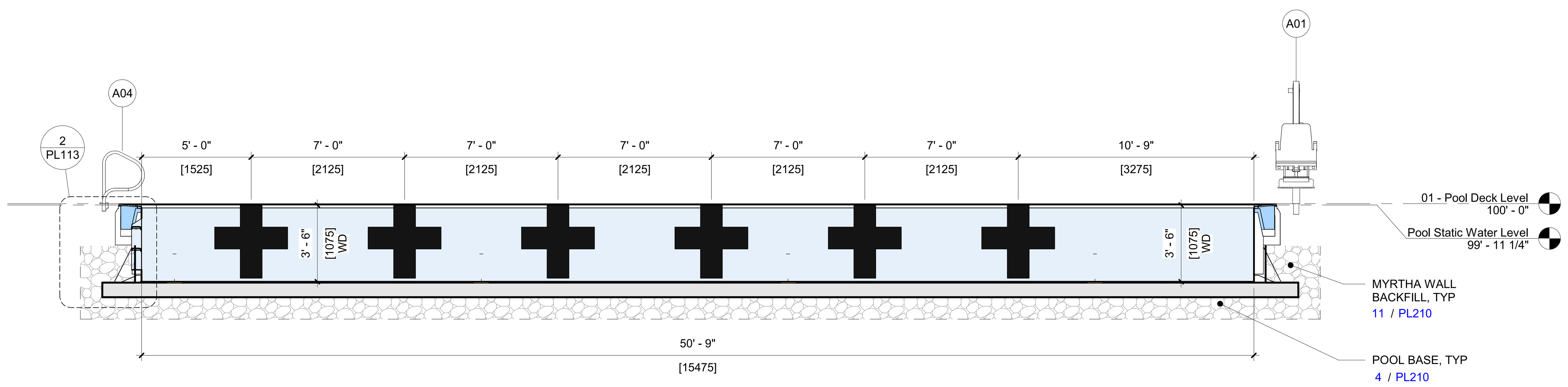
PL115



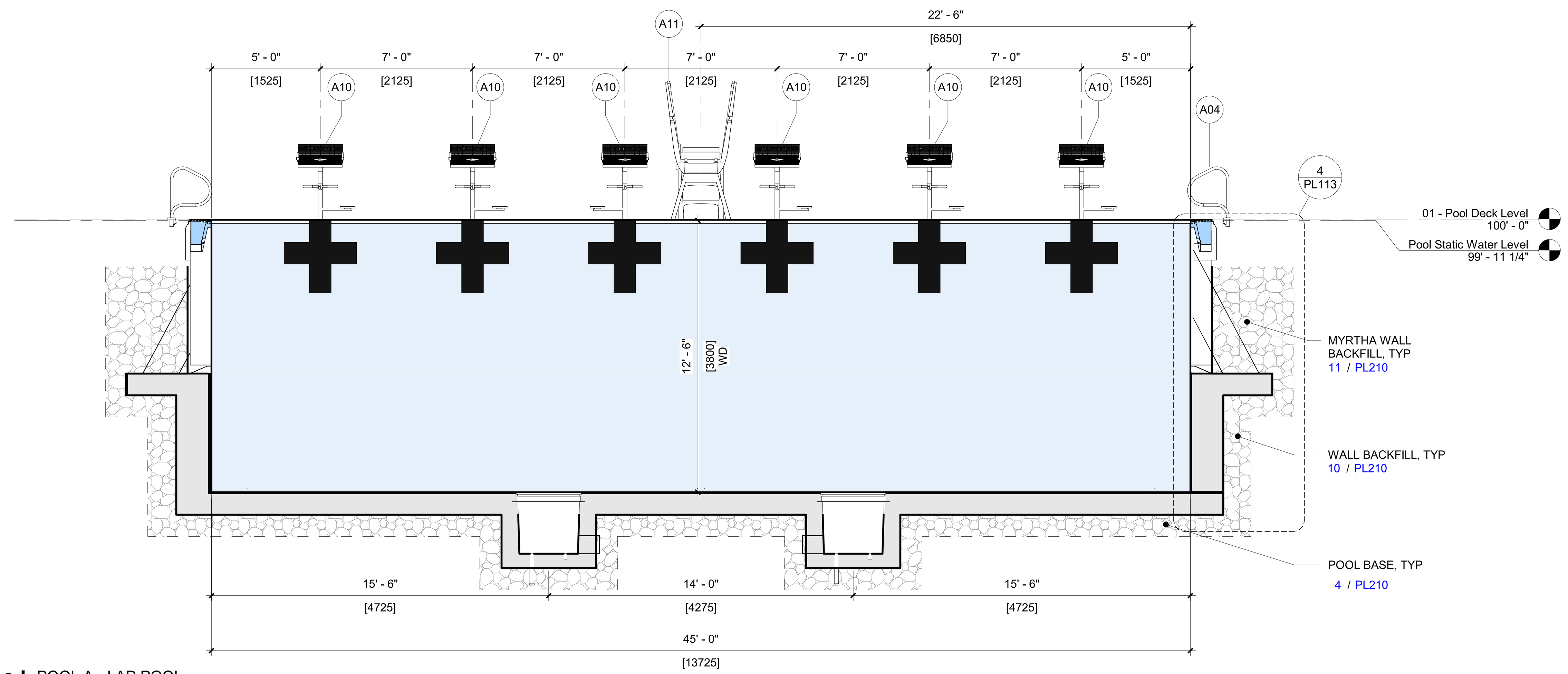
4 POOL A - LAP POOL SECTION VIEW
1/4" = 1'-0"



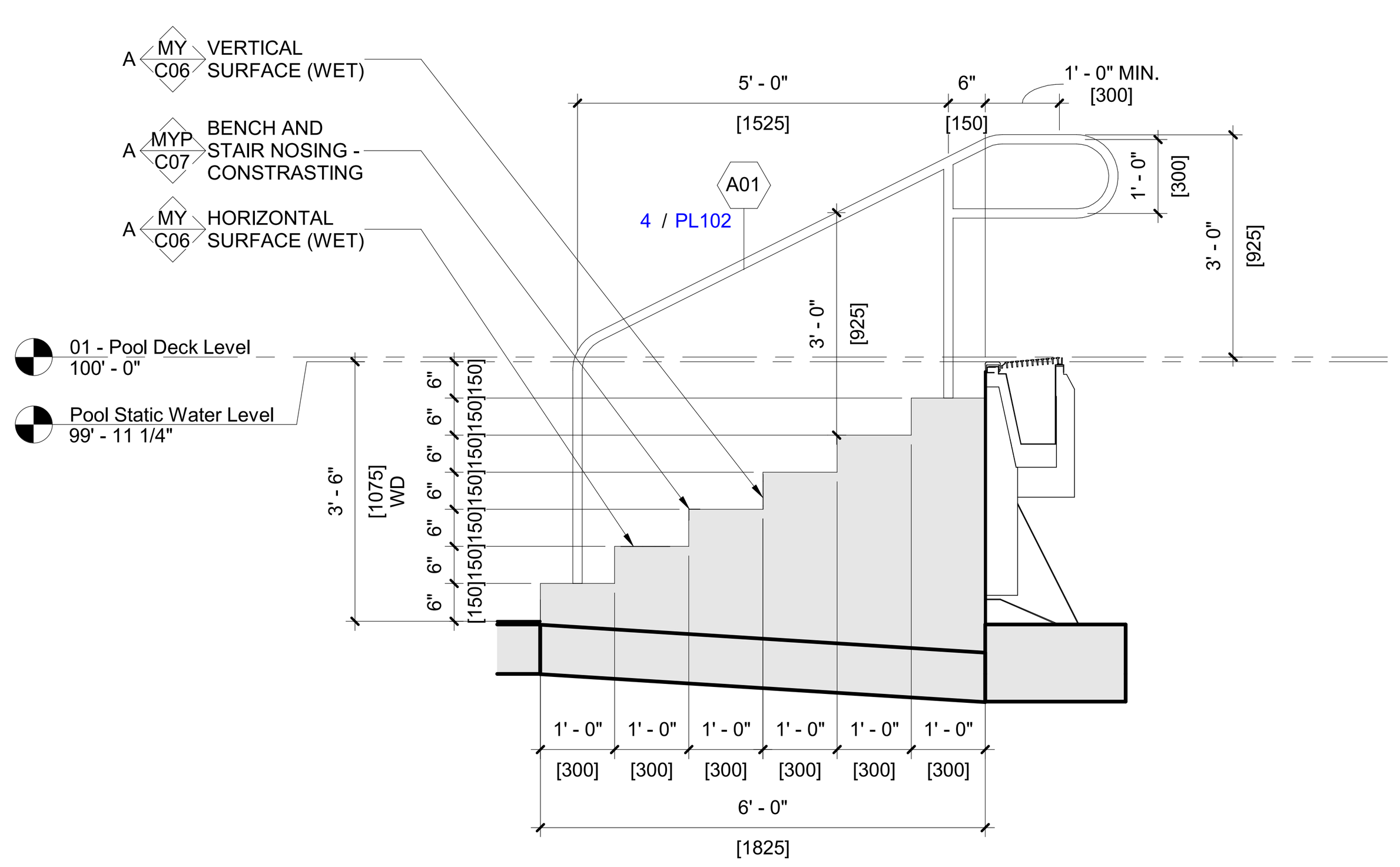
1 POOL A - LAP POOL SECTION VIEW
1/4" = 1'-0"



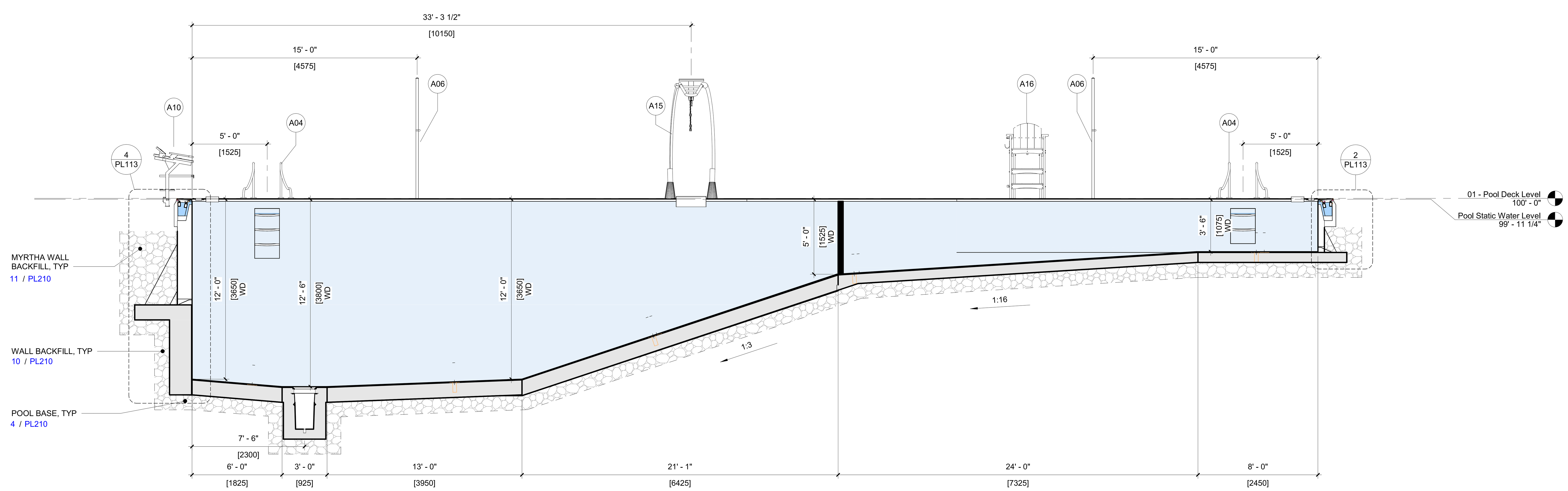
5 POOL A - LAP POOL SECTION VIEW
1/4" = 1'-0"



2 POOL A - LAP POOL SECTION VIEW
1/4" = 1'-0"



6 POOL A - LAP POOL STAIRS SECTION VIEW
1/2" = 1'-0"



3 POOL A - LAP POOL SECTION VIEW
1/4" = 1'-0"

REVISIONS

BID SET

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NAC NO	23458
DRAWN	MJC
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DATE	04/16/2024

POOL A - LAP POOL SECTIONS

PL112

4/12/2024 10:10:05 AM Autodesk Docs://111-23084 Cheney Aquatic Center/Cheney_Aquatic-Pool-24.rvt

Turn your pool into an ADVENTURE with AquaClimb®

For recreation centers, fitness facilities, camps, and private clubs, AquaClimb expands poolside programming with an easy addition that is safe, engaging, and fun. As the market leader, AquaClimb offers more benefits to its customers than any other climbing product:

Modular and Customizable

AquaClimb's height, width, and panel style can all be tailored to fit the size and design of your pool, with options for adding more panels at a later phase as your budget allows.

Challenging, Realistic Climbing

With 3D contoured panels, AquaClimb delivers a realistic rock-climbing experience that engages adolescents through adults to conquer the climb in different ways.

Top Safety Record

With best-in-class safety features to ensure climbers fall away from the wall, AquaClimb also has a proven performance history from 1,000 installations across the globe.

Activates the Deep End

As a safer alternative or enhancement to diving boards, AquaClimb attracts tweens and teens to those under-utilized, deep areas of a pool.

Easy to Install

Because AquaClimb is pre-assembled in the factory, no specialized skills or equipment are required for onsite installation at your facility on any pool gutter configuration.

Minimal Footprint

AquaClimb's small deck-mounted system saves clearance space and doesn't interfere with normal lap swimming. And with no water source required, it is an easy amenity to add.

AQUACLIMB® Four Unique Models

AquaClimb Krystal

- Budget-friendly and entry-level option
- Modular, flat panels in clear, blue, and green transparent tint
- Customizable up to four height options sized to pool's depth

AquaClimb 3D

- 3D contoured panels for realistic climbing available in translucent **Ice, Glacier,** or **Jade** colors, and solid painted color schemes
- Modular panels can be turned and flipped to change up the experience
- Translucent panels allow lifeguard visibility while giving privacy to the climber behind the wall

AquaClimb Kurve

- Sleek, curved frame that allows heights up to 20 feet
- 3D contoured panels available in color options of Ice or Glacier
- Translucent panels allow lifeguard visibility while giving privacy to the climber behind the wall

AquaClimb Luxe

- Completely customizable design to match your pool's aesthetics
- 3D contoured panels
- Deck mounted or Pool wall mounted

AQUACLIMB® Depth Requirements

Panel Options	A Minimum Pool Depth	B Drop Zone	C Plummet line from wall	D Available climbing height	E Height of top foothold*	F Above deck wall height
3 High Alt	5'	9'	1'9"	8'10"	4'5"	9'7"
3 High	6'	9'	1'9"	9'10"	5'5"	9'7"
4 High Alt	6'	10'	2'6"	12'1"	7'8"	12'10"
4 High	7'	10'	2'6"	13'1"	8'8"	12'10"
5 High Alt	8'	12'	3'3"	15'5"	11'	16'1"
5 High	9'	12'	3'3"	16'5"	12'	16'1"
6 High (Kurve Only)	10'	12'	3'3"	17'	12'5"	19'8"

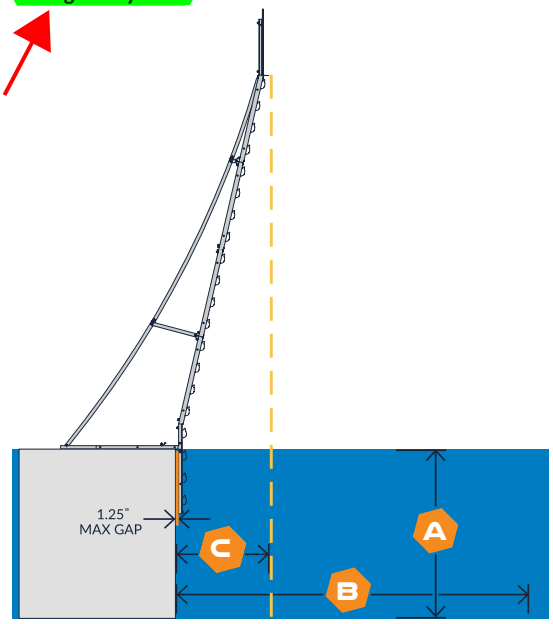
Cheney Products - see below for definition of Alt

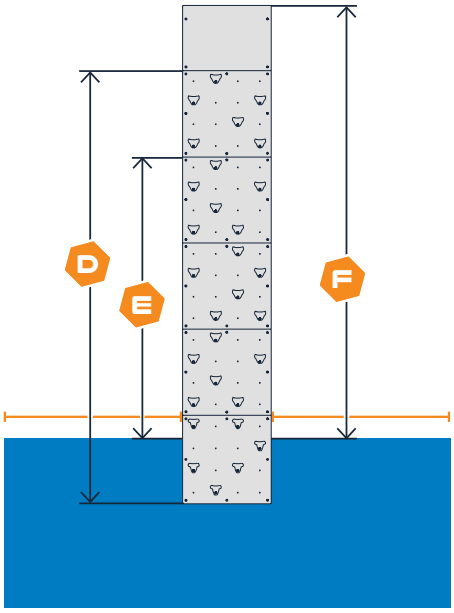
The Cheney project has a combination of a 5 high alt panel and (2) 5 high panels

*Based on climber's feet positioned at least 2' below highest hand grip

Alt - Alternate configurations will have the top row of handholds plugged for non-climbing terrain to meet pool depth requirements.

Important Safety Note: AquaClimb safety distances and pool depths are based upon a climber entering the water **feet first**. The AquaClimb was designed for a feet first entry at all times and supervision must be present when the AquaClimb is in use. To ensure the maximum level of safety, **there must be no diving at any time**.





Plummet Line

5 FT Fall Zone

*For installations that are 5+ panels high, a 6 FT Fall Zone is required.

To learn how you can bring the adventure of AquaClimb® to your facility, contact us today:

POOLSIDE ADVENTURES™

PoolsideAdventures.com | 800.956.6692 | info@poolsideadventures.com

Building Courageous Kids for Life's Great Adventure

FEAmax Report

AquaClimb Hand Calculation

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FEAmax LLC.

PROJECT INFO.

Change History:

Version Number	Date	Summary	Author
V 1.0	2/2/2016	Initial release	Frank Wang

Client Information:

Contact name:	Laura Grandner
Email:	Laura@aquaclimb.com
Company name:	Pyramide USA
Address:	P.O. Box 530 Frederick, MD. 21705

PROJECT DESCRIPTION

- **Project Description**

1. Calculate the minimum depth required to safely plummet down from the highest foot hold point on the (4) levels of AquaClimb Walls (2H , 3H , 4H and 5H).
2. With the top climbing hold measurement provided – deduct 36” (3ft) down which would be the highest foot hold placement. Then with the following parameters calculate the minimum depth needed to safety let go and plummet straight down into the water without reaching the bottom floor of the pool.
3. Height: 48” minimum; 78” Maximum
4. Weight: 50 lbs minimum; 250 lbs maximum

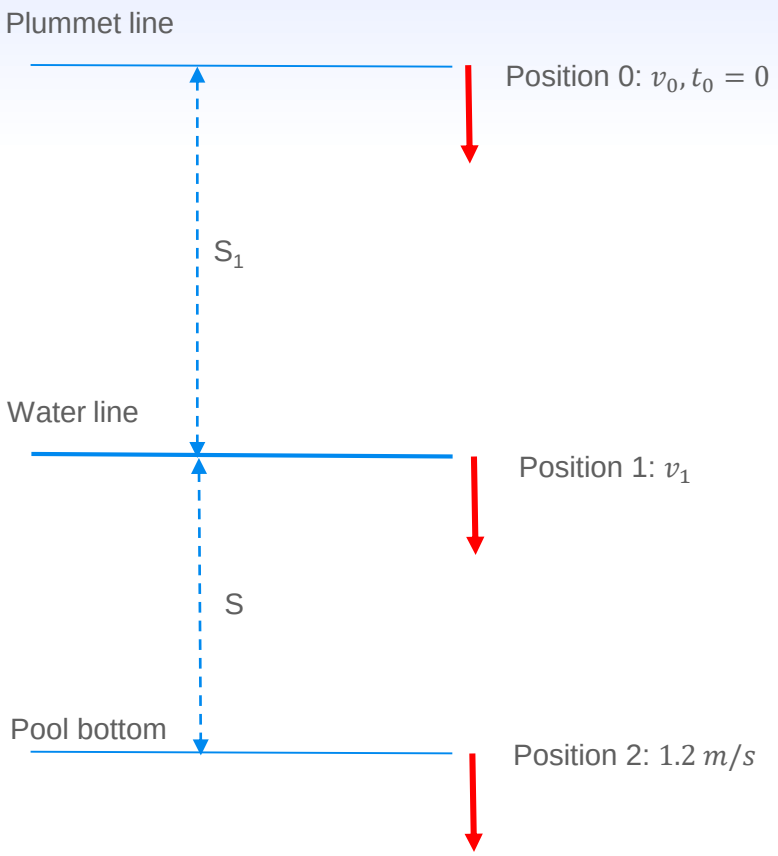
CALCULATION

Assumptions:

1. Minimum height of human body H_{human}= 48” = 1.2 meter
2. Water density ρ_{water}= 1.0 g/cm^3
3. Human body density ρ_{human}= 0.9 g/cm^3
4. The velocity enter the water = V_1
5. Water Resistance coefficient C_D = 1.0
6. Human body volume = V
7. Area of human body enter the water = A
8. Velocity of human body inside the water = V_x
9. The allowable decent velocity to the pool bottom = 1.2 m/s

Force applied to human body inside water:

1. Gravity $G = \rho_{human} g V$
2. Buoyancy (floating force) $\mathrm{F} = \rho_{water} g V$
3. Water resistance force $F_{resistance} = \frac{1}{2}\rho_{water} V_x^2 A C_D$



CALCULATION

According to Newton's second law, we have:

1. The acceleration in the water: $a = \frac{dV_x}{dt} = \frac{F}{m}$

2. $a = \frac{\rho_{human} gV - \rho_{water} gV - \frac{1}{2}\rho_{water} V_x^2 A C_D}{\rho_{human} V} = \frac{0.9\times9.8\times V - 1.0\times9.8\times V - 0.5\times1.0\times V_x^2 \times \frac{V}{1.2}\times1.0}{0.9\times V} = -(1.09 + 0.46V_x^2)$

3. $\frac{dV_x}{dt} = -(1.09 + 0.46V_x^2)$

4. $\mathrm{dt} = -\frac{dV_x}{(1.09+0.46V_x^2)}$

5. The max displacement of body moving in the water would be:

$$S = \int_0^t V_x \cdot dt = -\int_{1.2}^{V_1} V_x \cdot \frac{dV_x}{1.09 + 0.46V_x^2} = \cdots = -\int_{1.2}^{V_1} 0.46 \times \frac{1}{0.42} \times \frac{d(1 + 0.42 \times V_x^2)}{(1 + 0.42 \times V_x^2)}$$

$$= 1.09 \times [\ln(1 + 0.42 \times V_1^2) - \ln(1 + 0.42 \times 1.2^2)] = 1.09 \times [\ln(1 + 0.42 \times 2 \times 9.8 \times S_1) - 0.473]$$

6. The minimum depth of pool would be:

$$S = 1.09 \times \ln(1 + 8.23 \times S_1) - 0.52$$

CONCLUSION

If the body height is 48” (1.2 meter), we have:

$$S = 1.09 \times \ln(1 + 8.23 \times S_1) - 0.52$$

1. For 2H: S_1 = 1’ = 0.30 meter, we have the min pool depth:

   S = 0.84 meter = 2.8 feet

2. For 3H: S_1 = 1’9” = 0.53 meter, we have the min pool depth:

   S = 1.31 meter = 4.3 feet

3. For 4H: S_1 = 2’6” = 0.76 meter, we have the min pool depth:

   S = 1.64 meter = 5.4 feet

4. For 5H: S_1 = 3’3” = 1 meter, we have the min pool depth:

   S = 1.89 meter = 6.2 feet

Panel Options	A Minimum Pool Depth
3 High Alt	5'
3 High	6'
4 High Alt	6'
4 High	7'
5 High Alt	8'
5 High	9'
6 High (Kurve Only)	10'

CONCLUSION

If the body height is 78” (1.98 meter), the equation would be:

$$S = 1.78 \times \ln(1 + 5.49 \times S_1) - 0.60$$

1. For 2H: S_1 = 1’ = 0.30 meter, we have the min pool depth:

   S = 1.13 meter = 3.7 feet

2. For 3H: S_1 = 1’9” = 0.53 meter, we have the min pool depth:

   S = 1.83 meter = 6.0 feet

3. For 4H: S_1 = 2’6” = 0.76 meter, we have the min pool depth:

   S = 2.32 meter = 7.6 feet

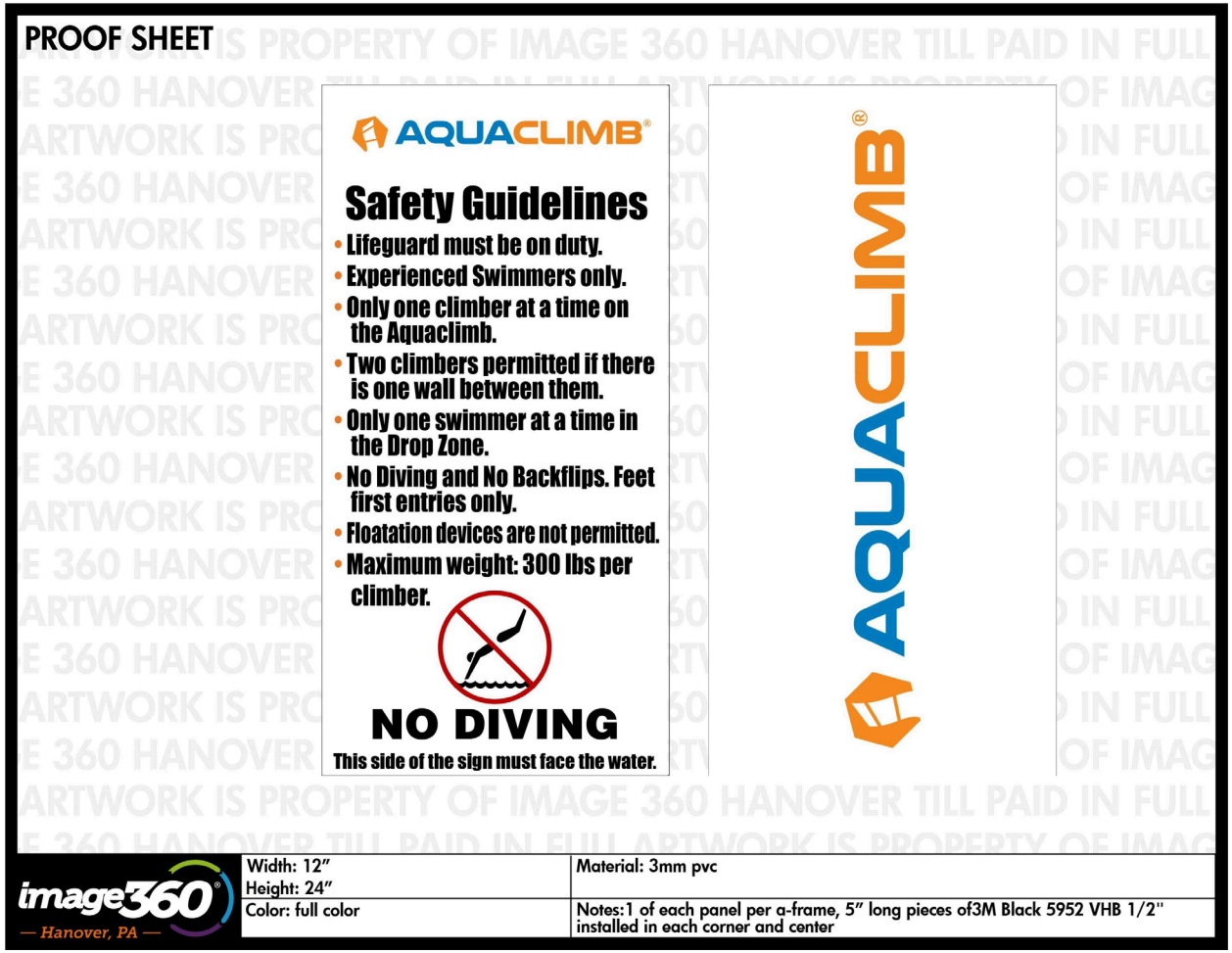
4. For 5H: S_1 = 3’3” = 1 meter, we have the min pool depth:

   S = 2.73 meter = 8.9 feet

Panel Options	A Minimum Pool Depth
3 High Alt	5'
3 High	6'
4 High Alt	6'
4 High	7'
5 High Alt	8'
5 High	9'
6 High (Kurve Only)	10'

Cheney pool depth at climbing walls exceeds this calculation and ranges from 9'-1" to 12'-4 1/2" at the 5H panel drop zones and 8'-4" to 9'-8" at the 5H Alt panel drop zones. The Alt panels do not have hand holds available at the highest points and therefore reduces the water depth minimum because the potential fall height has been reduced.

View proof for Printed PVC Panels for A-Frame

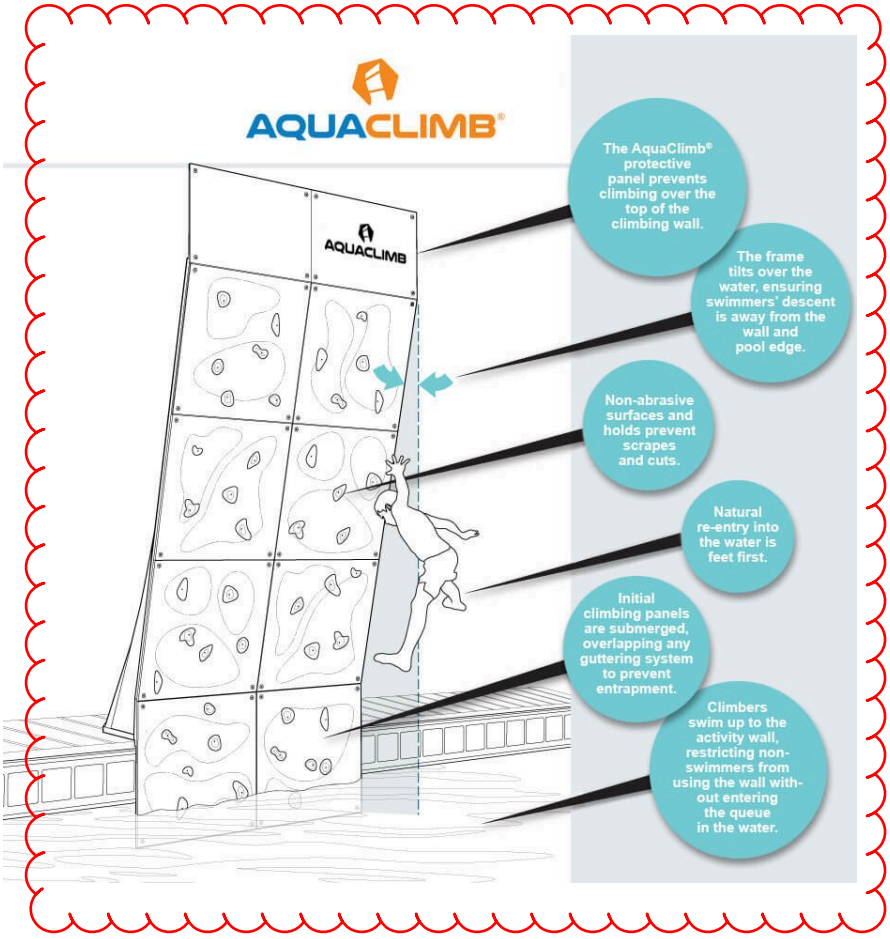


POOLSIDE ADVENTURES™

SAFETY

PROVIDE A SAFE WAY FOR POOL PLAY

AquaClimb® walls aren't just a fantastic poolside attraction. They're a safe way to play. They are specifically designed to eliminate the dangerous situations that can cause injury when sliding and diving. AquaClimb® is a safer alternative to diving boards and slides for both children and adults. Trust the brand that prioritizes you well being!



A SAFE WAY TO PLAY

- Each AquaClimb® comes complete with guidelines for safe use.
- AquaClimb® has clear protective panels to prevent climbers from climbing over the top of the wall.
- The AquaClimb® frame curves and hangs over the pool so that the natural re-entry into the water is feet first and the descent is away from the pool wall and edge.
- Non-abrasive surfaces and holds prevent scrapes and cuts.
- Natural re-entry into the water is feet first.
- Initial AquaClimb® climbing panels are submerged, overlapping any guttering system to prevent entrapment.
- Climbers swim up to the AquaClimb® activity wall, restricting non-swimmers from using the wall without entering the queue in the water.

Poolside Adventures products are recommended by the Aquatic Safety Research Group (ASRG) and are approved by state and

MEET OUR SAFETY TEAM

DR. TOM GRIFFITHS

Dr. Tom Griffiths is the President and Founder of Aquatic Safety Research Group, LLC. Recognized as an international leader in water safety, he has spent 38 years teaching, coaching and managing aquatics at three major universities. Griffiths has produced videos, textbooks, articles, and presentations in

local health departments throughout the USA, in addition to major health and safety organizations like PlaySafe LLC, a member of the International Play Equipment Manufacturers Association.

AquaClimbs are designed and engineered to the following standards:

- AISC Manual of Steel Construction, 15 th Edition, ASD
- IBC 2018
- ASCE/SEI 7-16
- ASTM F24/F2291- 21- Standard Practice for Design of Amusement Rides and Devices
- ASTM F2461-20 Aquatic Play Equipment
- European Standards EN17164 – Climbing Walls for Use in the Water Area

AquaZip'Ns are designed and engineered to the following standards:

- ASTM F2291-18 Amusement Rides and Devices
- ASTM F2461-18 Aquatic Play Equipment

CHECK OUT THESE ARTICLES ON THE BENEFITS OF ROCK CLIMBING FOR KIDS!

various areas of aquatics focusing his efforts on safety. He has also conducted hundreds of aquatic facility and beach inspections across the nation and abroad and teaches full day Aquatic Risk Management seminars. Perhaps his most significant contributions are the Five Minute Scanning Strategy©, Griff's Guard Stations©, Disappearing Dummies, his research on Shallow Water Blackout, and the National Note & Float program. He has been an aquatic safety expert for more than 40 years and shares his knowledge, expertise, and experience worldwide. Griffiths just released the 3rd

Why Rock Climbing is Such an Awesome Activity For Kids

5 Mental Health Benefits of Rock Climbing

Poolside Adventures stands on a history of providing a safe climbing experience. The recommended rules provided on our signage and advised during the sales and acquisition process are extremely important to operating a safe and fun activity for all.

We have recently viewed four YouTube videos which show our walls not being properly supervised, having the safe operation signage being displayed at the wall and the wall itself being used in a potentially unsafe manner. Though no accidents have been reported we strongly ask that all facilities please review the safe operation signage with staff and follow our guidelines.

Thank you!

edition of the popular The Complete Swimming Pool Reference.

Read Dr. Tom Griffiths 10-Year Review of the AquaClimb (PDF)

RACHEL GRIFFITHS

Rachel Griffiths, M.A. is the Communication Director for Aquatic Safety Research Group. Rachel conducts water safety research to help prevent drowning and provides water safety education to the public. She is also the President of Note and Float Life Jacket Fund,

AQUATIC SAFETY
Research Group

We Take Water Safety Seriously

DATE: April 9, 2015
TO: Laura Grandner
FROM: Dr. Tom Griffiths
RE: AquaClimb

Ten Year Review

As you know, nearly ten years ago, we placed an AquaClimb climbing wall in the diving well on the Penn State University Campus to test and analyze your product. I was pleased to learn how attractive it was to our students, and how it promoted fun and fitness in the pool with a new and exciting activity that was safe.

Since that time, Rachel and I have inspected hundreds of aquatic facilities and discovered that AquaClimb Walls are a safer alternative to many other poolside recreational products, primarily because swimmers do not have to climb a ladder in a wet environment over a concrete swimming pool deck. Because AquaClimb is accessed from the water inside the swimming pool, rather the swimming pool deck, there is very little chance of a child falling and hitting the deck. Further, the AquaClimb is angled out over the water, and as a result it is very improbable, if not impossible, that a child can fall to the deck.

As an expert witness in courts of law, I see many horrific accidents involving diving boards and slides, but I have never heard of an accident of any kind, minor or major, involving an AquaClimb. As we travel around this country and abroad teaching our full day Aquatic Risk Management Seminars, promoting AquaClimb as a safe, fun, and fitness alternative to other pool products is an essential part of our program. As you recall, AquaClimb is particularly valuable as a replacement for diving boards which no longer meet the depth and distance requirement or because of inadequate protective railings. I might also add that I have never seen a pool product installed as quickly in a swimming pool as an AquaClimb. I truly believe in your product and remain available to answer any questions you and others may have concerning AquaClimb Climbing Walls.

1632 Glenwood Circle, State College, PA 16803 **T** 814.883.0058 **F** 814.234.0313 TomGriffiths@AquaticSafetyGroup.com
AquaticSafetyGroup.com

Regards,

Tom Griffiths
President and Founder
Aquatic Safety Research Group, LLC

Rachel Griffiths
Communication Director
Aquatic Safety Research Group, LLC

AQUATIC SAFETY RESEARCH GROUP, LLC
CONSULTING, TRAINING AND EXPERT WITNESS SERVICES

I. INTRODUCTION

The AquaClimb is an exciting new recreational and fitness component that offers new programming opportunities to aquatic facilities. Because the AquaClimb extends below the surface of the water, participants can easily swim up to the climbing wall and begin to traverse it without leaving the pool itself. Even those individuals without use of their legs can utilize the AquaClimb to exercise the upper body in a fun, challenging, and non-threatening way. Perhaps the most meritorious application of the AquaClimb is an alternative to a diving board in a swimming pool which no longer meets safe diving depth and distance requirements.

Climbers who fall from the AquaClimb will enter the water feet-first. To enter the water head-first from the climbing wall structure is almost a biomechanical impossibility. Prior to purchasing and installing an AquaClimb, aquatic facilities should contact their local regulatory agency (e.g. Health Department) to determine whether regulations, recommendations or suggestions regarding the safe installation and use of the AquaClimb exist. **AQUATIC SAFETY RESEARCH GROUP, LLC**, an independent and objective water safety consultant firm, remains available to assist facilities in answering questions concerning the safe use of the AquaClimb.

II. STANDARD OPERATING PROCEDURES

A. LIFEGUARDS

Whenever the AquaClimb is in use, it is recommended that a properly trained and certified lifeguard be assigned exclusively to the AquaClimb. The lifeguard should be strategically placed to supervise and control use of the structure and to minimize climber

misbehavior. Because the apparatus will be positioned in deep water, a lifeguard with deep water skills and qualifications is needed. This lifeguard must also be trained for the proper use and monitoring of the in-water climbing structure. The lifeguard should be positioned close to the wall with a full and unobstructed view of the climbing wall and drop zone, with the ability to see underwater in the drop zone. The lifeguard must stay focused on the climbing wall whenever in use and attention should not be diverted to other areas of the pool. Lifeguard orientations, in-service trainings and emergency action plans should include the AquaClimb and should be reviewed and practiced regularly but at least monthly. In many pools, the best vantage point for proper surveillance may be directly across the pool facing the wall. However, each facility should determine where to best position supervisory staff to ensure a full and unobstructed view of the climbing wall and the drop zone.

The aquatic facility should also establish an entrance and exit pattern (left to right and right to left) to avoid congestion of swimmers waiting to swim into the drop zone to begin their ascent on the wall. This pattern can be changed daily or hourly. For larger installations allowing two or more climbers, additional safety precautions must be implemented to minimize the risk of a climber falling onto someone swimming into or out of the drop zone. One such approach is to direct climbers, once they have fallen from the wall, to swim to the closest edge of the drop zone so as to avoid swimming underneath a second climber.

B. DEPTH REQUIREMENTS

While most competitive swim agencies, including the National Collegiate Athletic Association (NCAA), require a minimum water depth of five (5) feet to dive headfirst from starting platforms, the AquaClimb, which promotes only feet-first entries, takes a more conservative approach, requiring a minimum water depth of five (5) feet for installation of its shortest three-panel wall. As panels are added vertically to the structure, minimum water depth requirements increase. To ensure safety of climbers, AquaClimb has applied commonly accepted safe head-first diving depths to feet-first entries from the structure.

We recognize that these depths are very conservative given that they are intended to minimize the risk of injury from head-first entries rather than from feet-first entries, but

absent additional research we cannot safely recommend alternative water depths which deviate from these nationally-accepted standards.

MINIMUM DEPTH REQUIREMENTS FOR AQUACLIMB INSTALLATION			
Panel Height* - standard	3 panels (lowered)	4 panels (lowered)	5 panels (lowered)
Minimum Water Depth	5 feet	7 feet	8 feet

* Each panel measures approximately $3ft^2$ or $1m^2$

MINIMUM DEPTH REQUIREMENTS FOR AQUACLIMB INSTALLATION			
Panel Height* - standard	3 panels	4 panels	5 panels
Minimum Water Depth	6 feet	8 feet	9 feet

C. DECK CLEARANCES

Whenever possible, four feet of deck space should be maintained between the end of the support structure and the perimeter pool wall or fence. If less than four feet is available, a combination of pedestrian control stanchions and traffic cones should be used to direct patrons around the support system. To best accommodate persons with disabilities, a minimum of three feet (36”) clearance around the support structures should be maintained. Even with spacious decks, stanchions and cones always come highly recommended, as they minimize the risk of someone coming into contact with the structure. Customers are advised to check building and fire codes to determine whether support structures can permissibly block access to the pool deck, particularly in cases where the support structure would come within three feet of a wall.

D. NUMBER OF CLIMBERS

With a one panel or two panel wide AquaClimb, it is ***highly recommended*** that only one climber use the AquaClimb at a time. With a three panel or wider AquaClimb, however, there is an opportunity to allow more than one climber on the wall at the same time. Multiple climbers should only be allowed when there is no possibility of one climber either interfering with or falling on top of another climber. Multiple climbers should be instructed to climb the wall vertically rather than to traverse the wall horizontally. Climbers should also maintain a distance of at least one panel from other climbers to minimize the risk of climber interference, horseplay and accidental concurrent falls.

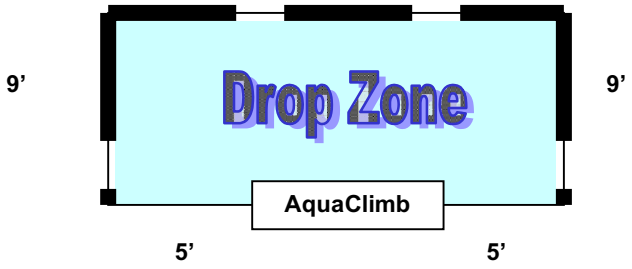
E. VERIFIED SWIMMERS ONLY

Because the AquaClimb is installed in deep water (see minimum depth requirements above), this climbing attraction is to be used only by "swimmers" – persons with verified swimming ability. The attractive colors and the fun activity that the structure provides, are likely to draw younger, weaker swimmers to the climbing wall. These persons should be properly screened to ensure they possess the requisite deep-water skills necessary for using the structure. Following standard aquatic safety practices, anyone wishing to enter deep water to use the AquaClimb should be given a swim test. A recommended swim test would be to have the swimmer/climber jump into ***chest-deep*** water, surface, swim the equivalent length of the buffer zone and return to the starting point. Requiring climbers to tread water for 30 – 60 seconds comes highly recommended. Swim tests should be conducted in chest-deep water to maximize swimmer safety.

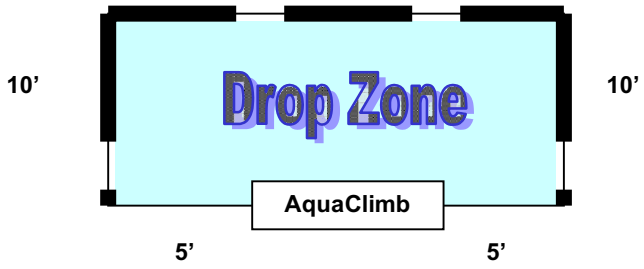
F. DROP ZONE

Climbers will fall from the wall into the water. It is therefore imperative to keep people from entering the "drop zone" where they would risk being struck by a falling climber. No other swimmers should be allowed into the drop zone when a climber is on the wall.

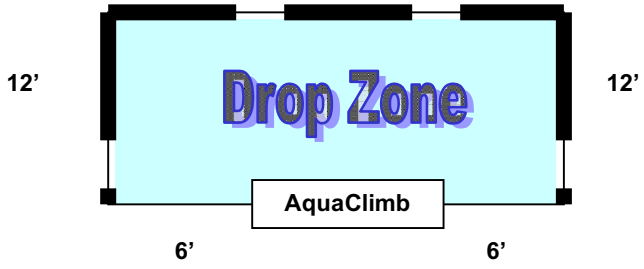
3 panel high:



4 panel high:



5 panel high:



G. FEET-FIRST ENTRIES ONLY

While head-first entries, including dives, are improbable to perform from the face of the climbing wall, and although the depth requirements for the various climbing wall configurations are extremely safe and tend to be conservative, climbers must be warned that all entries into the water from the AquaClimb should be feet-first. Climbers who intentionally violate this safety rule should be prohibited from using the AquaClimb.

H. UNDERWATER ACTIVITIES

Participants should not be allowed to play with the structure itself, particularly while submerged. While there are no hidden hazards or entrapment potentials inherent in the AquaClimb, it is intended for above-water use. It is not intended or designed for underwater use by climbers. Playing underwater around the structure makes it more difficult for the lifeguard to properly supervise the activity. This could lead to injury should a climber fall onto someone who was playing underwater in the drop zone.

III. SUGGESTIONS FOR SAFETY SIGNAGE

Perhaps the most appropriate place to place caution/warning signs would be on the side. The three most important warnings should include:

- "Swimmers Only"
- "No Head First Entries"
- "Only One Climber at a Time unless there are 1-2 clear panel between climbers"

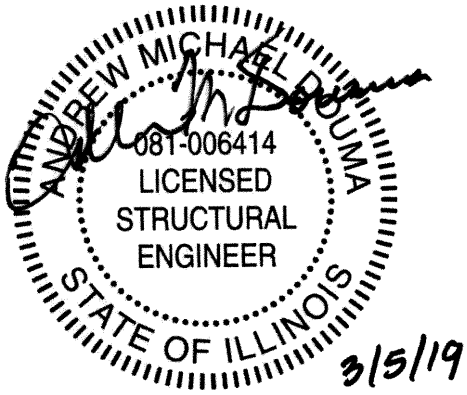
These three warnings can be placed together on the same sign in the appropriate colors (red/white, black/yellow, orange/black). Additional signs/warnings may be mounted on the rear of the support structure.

ASTM F2291-18 & ASTM F2461-18 STAMPED REVIEW

AQUACLIMB CLIMBING WALL

Project: Park Department
City of Decatur, Illinois

FRAMING AND COMPONENT DESIGN



Prepared For:
Pyramide USA, Inc.
8 East 2ND Street
Frederick, MD 21701

WBCM PROJECT NO. 19.0056.00
Date: 3/05/19

WHITNEY, BAILEY, COX & MAGNANI, LLC
100 Sterling Parkway – Suite 108 Mechanicsburg, PA 17050
MAIN (717) 691-4708 FAX (717) 691-4749

AQUACLIMB CLIMBING WALL

Design Criteria:

Loading:

- **Live Load** = 300lbs MAX Point Load
  Deflection Limit L/360
- Per ASTM F229- Consider Load Combination of Min 34 mph wind plus climber (section attached)
- Wind Load-
  Basic- 100mph
  Reduced for Combination- 35mph
  Exposure = B
  Importance Factor = 0.87
  Kzt = 1.0
  Kd = 0.85

Material:

- Tubes – A304 Stainless Steel, Fy=30 ksi, Fu = 75 ksi
- plates – A304 Stainless Steel, Fy=40 ksi, Fu = 88 ksi
- Bolts - ASTM F593 Type 304 Stainless steel bolts

References:

- AISC Manual of Steel Construction, 13th Edition, ASD
- IBC 2015
- ASCE/SEI 7-05
- ASTM F2291-18 Amusement Rides and Devices
- ASTM F2461-18 Aquatic Play Equipment

Note – Design of panels, hand-holds and anchorage to panel, and panel anchorage to frame is not included in our scope of services

NAC
ARCHITECTURE

Patty Hayes, Board Chair
Washington State Board of Health
PO Box 47990
Olympia, WA 98504-7990

CHENEY AQUATIC CENTER Variance Letter Date: 2024.06.25

PROJECT IDENTIFICATION: Lap Pool #: SR009200 Leisure Pool #: SR009201

On Behalf of:
Cheney Aquatic Center, City of Cheney
Owner Contact: Dan Curley Phone: 509-498-9293
Owner Address: 609 2nd Street Cheney, WA 99004
Facility Address: 115 North 8th Street (formerly 711 Cedar Street), Cheney, WA 99004

Owner Representative: Brooke Hanley (NAC Architecture) 509-838-8240

Variance Request Contact:
NAC Architecture: Brooke Hanley Phone: 509-838-8240 Email: bhanley@nacarchitecture.com

Facility Information:
Cheney Aquatic Center - Project includes an outdoor 6-lane 25-yard lap pool & separate leisure pool with zero-entry, spray features, & lazy river. The pool building with locker rooms, lifeguard offices, party room, and mechanical spaces is about 5000sf. The entire facility is lifeguarded and enclosed securely.

Plan Submittal: Drawing Plans have been submitted for review.

Variance Request Citation:
WAC 246-262-160 states *the board may grant a variance from requirements of chapter **246-262** WAC if, in the sole discretion of the board, data and/or research provides sufficient evidence that the RWCF (attraction, device, equipment, procedure, etc.), will adequately protect public health and safety, as well as water quality.*

Variance Request: Code language related to Diving Envelope (WAC 246-262-010(21) & WAC 246-262-060(5)(vi)) for the **AquaZip'N Rope Swing** attraction.

Items noted in review letter include:

- **Aqua Zip'N Rope swing** attraction receiving pool shall conform to the CNCA or FINA standards (depth application and setbacks)

In the Spokane Regional Health District review response issued by Steve Main dated May 24, 2024, Steve requests NAC Architecture (NAC) and WaterTechnology, Inc. (WTI) address important concerns regarding public safety related to the receiving pool for the proposed **AquaZip'N Rope Swing** attraction in Pool B.

The concern is to address the minimum depth of the pool to be compliant with the WAC 246-262-010(21) & WAC 246-262-060(5)(c)(vi) regarding diving envelopes for features where users enter the water from above the water surface.

On behalf of the City of Cheney; NAC & WTI respectfully requests your consideration of the current pool depth design at the **rope swing** for the future Aquatic Center. To support this request we provide the attached information, engineering exhibits, and following commentary:

- The review letter states that the "diving envelope" from WAC 246-262-010(21) applies to **all attractions** where users enter above pool water level and therefore requires the CNCA (enter less than 20" above the water surface) or FINA (enter 20" or greater above the water surface) water depths. We submit that the attached engineering calculations for the **AquaZip'N Rope Swing** product will demonstrate that the manufacturer's required water depths and the designed water depths provided at the Cheney Aquatic Center are more than sufficient to protect the safety of the users allowed to participate in this attraction. Calculations were completed for a 72" tall, 250lbs person, any body size smaller than the max would perform better, not worse. The manufacturer's minimum depth requirement is 4 feet. The current Cheney receiving pool water depths exceed the manufacturer's recommendations as it is located in an area that ranges from 6'-8" to 10'-6" deep. Please review the attached data in support of using the manufacturer's depth requirements in lieu of the CNCA diving envelope dimensions.

- WAC 246-262-060(5)(c)(vi) appears to apply specifically to "diving envelopes in pools or areas of pools designated for diving activities". The applicant submits that diving activities are generally defined as plunging into the water headfirst. Diving headfirst into water results in the need for deeper water to avoid a head & neck collision with the pool floor which is different than a feet-first or tucked entry plunge where the body is significantly slowed in the first two feet of water. The **rope swing** safety guidelines (provided in the exhibits) will note that users are required to enter the water in a feet-first manner. Diving from the unit is prohibited. The engineering calculations completed also assume a feet-first plummet into the water.

- The Model Aquatic Health Code also addresses the complexity of "other aquatic features" like this and would suggest that the manufacturer recommendations for design and operation would be adequate to install the feature.

  4.12.10[A] Other Aquatic Features Other AQUATIC FEATURES not otherwise addressed in the CODE, including but not limited to climbing walls, inflatables, and play structures, shall not be installed unless designed and operated in accordance with all manufacturer's installation and operations recommendations.

- 'A-frame' signs with all written safety guidelines will be publicly displayed near the rope swing (see page 12 for example) to meet the criteria of WAC 246-262-070(10). Participants will be screened by lifeguards to ensure they are within the minimum and maximum size requirements.

- See attached rope swing diagrams to understand how the hand holds are provided on the rope at even intervals between 57" and 87" above the deck. The relatively low height of the hand holds does not allow the users to gain much elevation above the water as they slide out over the surface.

- Safety padding rated for falls from 6ft or less are provided around the base of the rope swing structure and down the face of the pool wall to prevent injuries at the corner of the gutter. The rope swing itself has a safety catch, so when the user swings out over the water, they are prevented from sliding back toward the wall. Once the user drops into the pool, the rope self-retracts so the next user does not need to reach out over the water to grab the rope.

- This pool will be lifeguarded at all times while in operation and the lifeguard staff will be the first line of defense to screen bathers to make sure they are experienced swimmers, instruct swimmers on proper use of the attraction, and direct proper swimmer circulation to and from the activity within the pool to avoid congestion or collisions. The **rope swing** will have a dedicated lifeguard to closely supervise the safety of swimmers when the attraction is open for use. Cheney is dedicated to making this facility fun while also as safe as possible for their community members and patrons.

- The **AquaZip'n** has been designed and engineered to meet the following standards:
  - ASTM F2291-18 Amusement Rides and Devices
  - ASTM F2461-18 Aquatic Play Equipment
  - AISC Manual of Steel Construction
  - Other industry standards listed in the product data attached

- NAC submits that the design as described above and substantiated in the attached documentation meets the intent of providing a safe receiving pool for the **AquaZip'N Rope Swing** feature. NAC, WTI, and the City of Cheney respectfully requests a variance accordingly. If the State Board of Health has any follow-up conditions or actions required of the owner/operator, we are committed to reviewing them for implementation.

NAC Architecture (NAC) has teamed with Water Technology (WTI) on numerous aquatic projects and so we have a history of producing these projects successfully. WTI has been designing Aquatic venues for over 40 years. WTI is widely known in the industry as one of the leading aquatic design firms in North America. As one of the industry's leaders, WTI has represented the waterpark industry during CPSC meetings on review of VGB rules and has also been involved in reviewing/editing sections of the MAHC. They are also represented in the Washington DOH committee to update the existing administrative code to adopt a more comprehensive aquatic code like the MAHC. The NAC and WTI commitment to safe aquatic facilities is proven. The design of the receiving pool at the **AquaZip'n Rope Swing** for the Cheney Aquatic Center will not put the health and safety of the public at risk. The City of Cheney, having operated a public pool for many years is experienced and committed to the safety and the welfare of their patrons. On behalf of the City of Cheney, NAC Architecture would like to thank you for your

consideration of this Variance Request. Please feel free to contact me with any questions you may have regarding this request.

Thank you,

Brooke Hanley

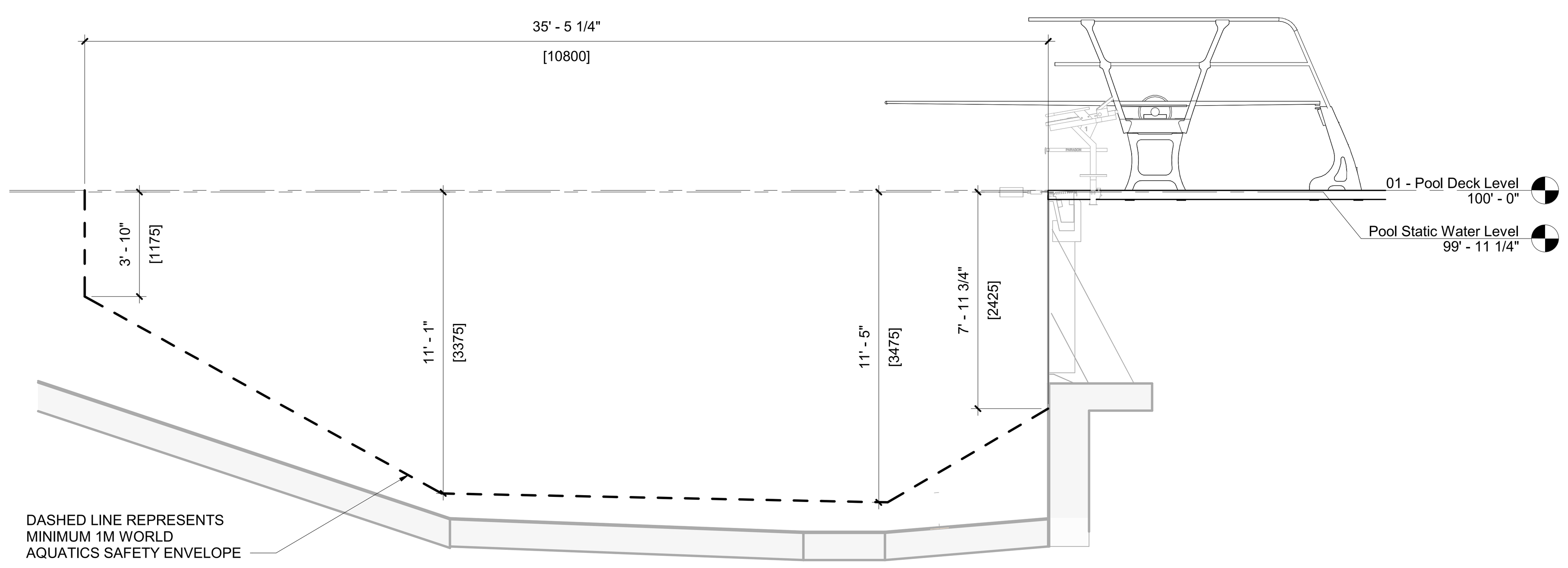
Brooke Hanley, AIA, Principal Architect, NAC Architecture

Attachments:

- AquaZip'n Safety Information and Fall Zone Engineering, including a floor plan and section of the receiving pool for the Cheney Aquatic Center.

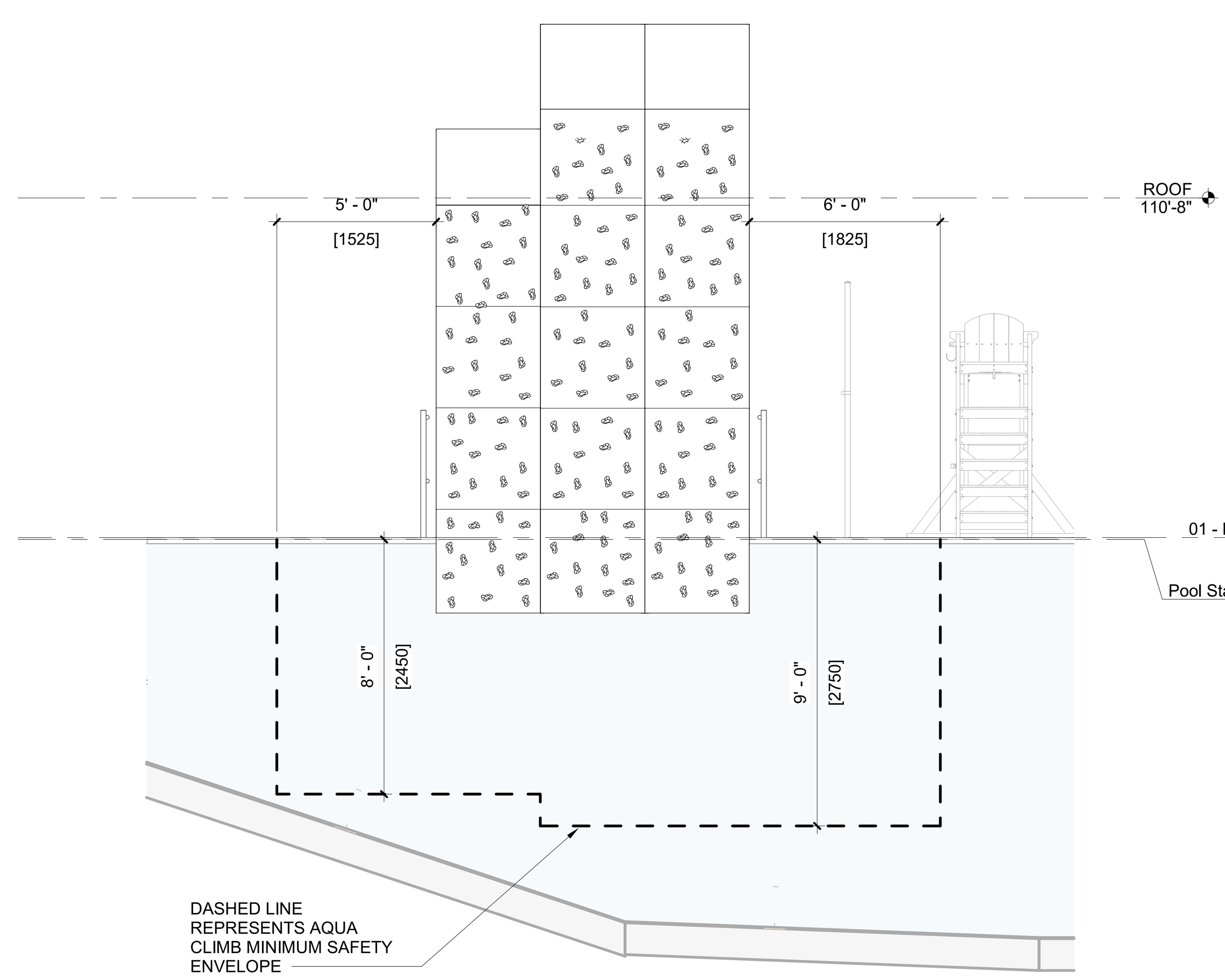
REVISIONS

NOTE:
ALL LANE DIVIDERS AND BACKSTROKE FLAGS MUST
BE REMOVED WHILE DIVING BOARD IS IN USE.



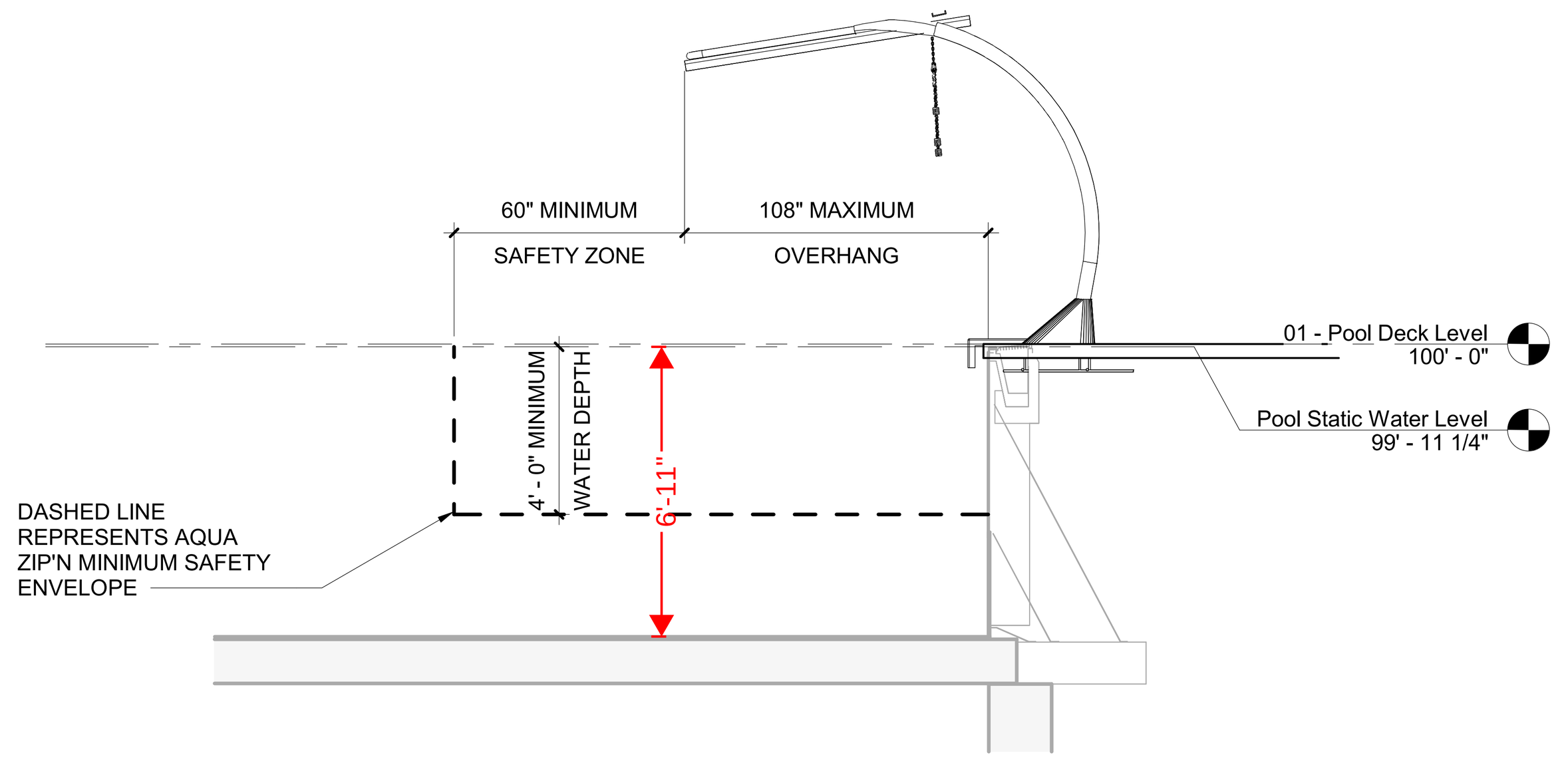
2 | POOL A - LAP POOL DIVING SAFETY ENVELOPE
PL115 | SECTION VIEW
1/4" = 1'-0"

NOTE:
ALL LANE DIVIDERS AND BACKSTROKE FLAGS MUST
BE REMOVED WHILE CLIMBING WALLS ARE IN USE.



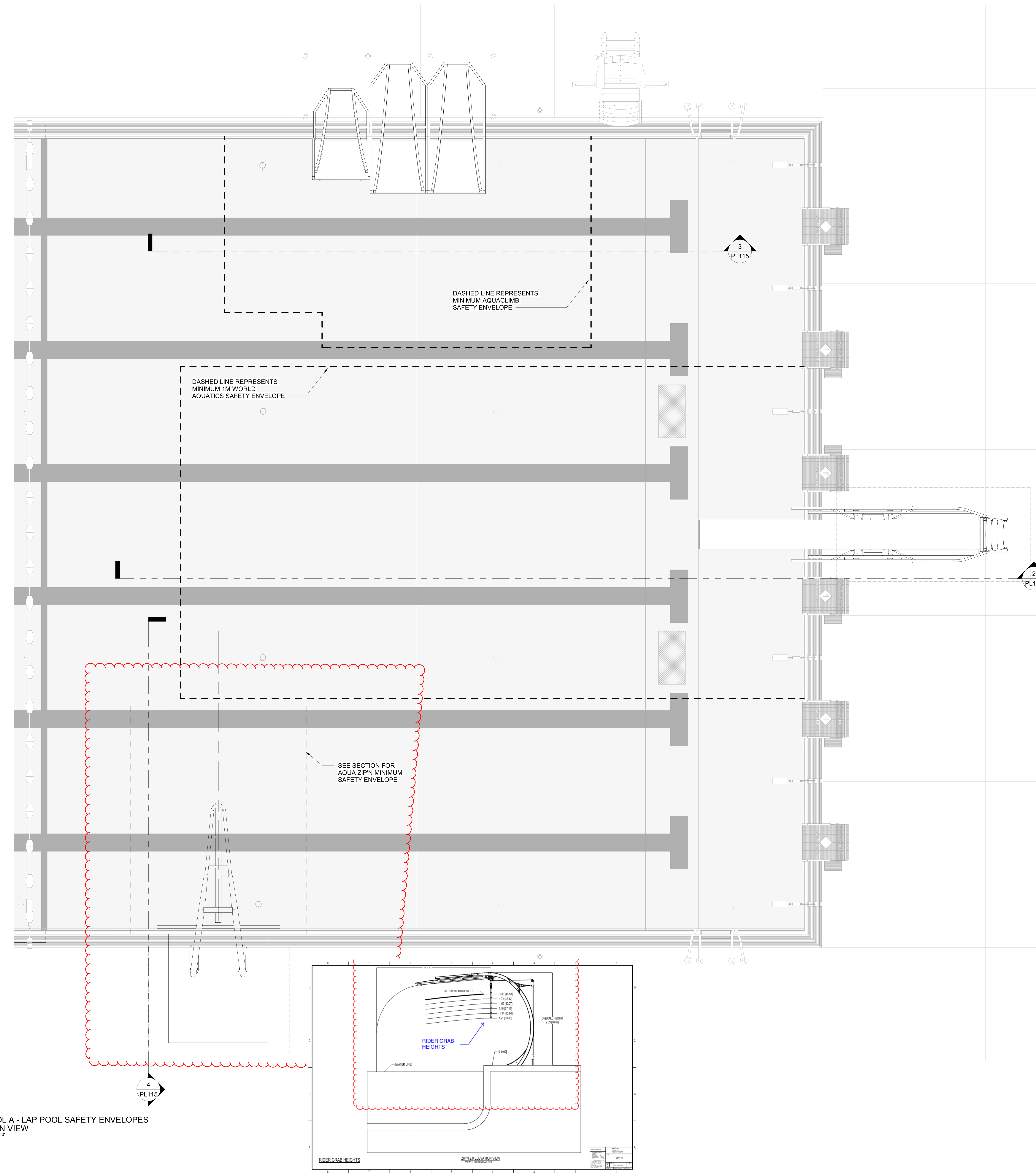
3 | POOL A - LAP POOL CLIMBING WALL SAFETY ENVELOPE
PL115 | SECTION VIEW
1/4" = 1'-0"

NOTE:
ALL LANE DIVIDERS AND BACKSTROKE FLAGS
MUST BE REMOVED WHILE AQUA ZIP'N IS IN USE.



4 | POOL A - LAP POOL AQUA ZIP'N SAFETY ENVELOPE
PL115 | SECTION VIEW
1/4" = 1'-0"

NOTE:
ALL LANE DIVIDERS AND BACKSTROKE FLAGS MUST BE
REMOVED WHILE CLIMBING WALLS, DIVING BOARD AND
AQUA ZIP'N ARE IN USE.



1 | POOL A - LAP POOL SAFETY ENVELOPES
PL115 | PLAN VIEW
3/8" = 1'-0"

BID SET

WTI
WATER TECHNOLOGY INC
World Leaders in Aquatic Planning, Design and Engineering
100 Park Avenue | Beaver Dam, WI 53916
t 920.887.7375 | #23458

CITY OF CHENEY
CHENEY AQUATIC CENTER
711 CEDAR ST. CHENEY, WA 99004

REGISTERED ARCHITECT
MATTHEW W. FEEBY
STATE OF WASHINGTON
4/16/2024

NAC
ARCHITECTURE
nacarchitecture.com
1203 WEST RIVERSIDE AVE
SPOKANE WA 99201
P:509.838.8240

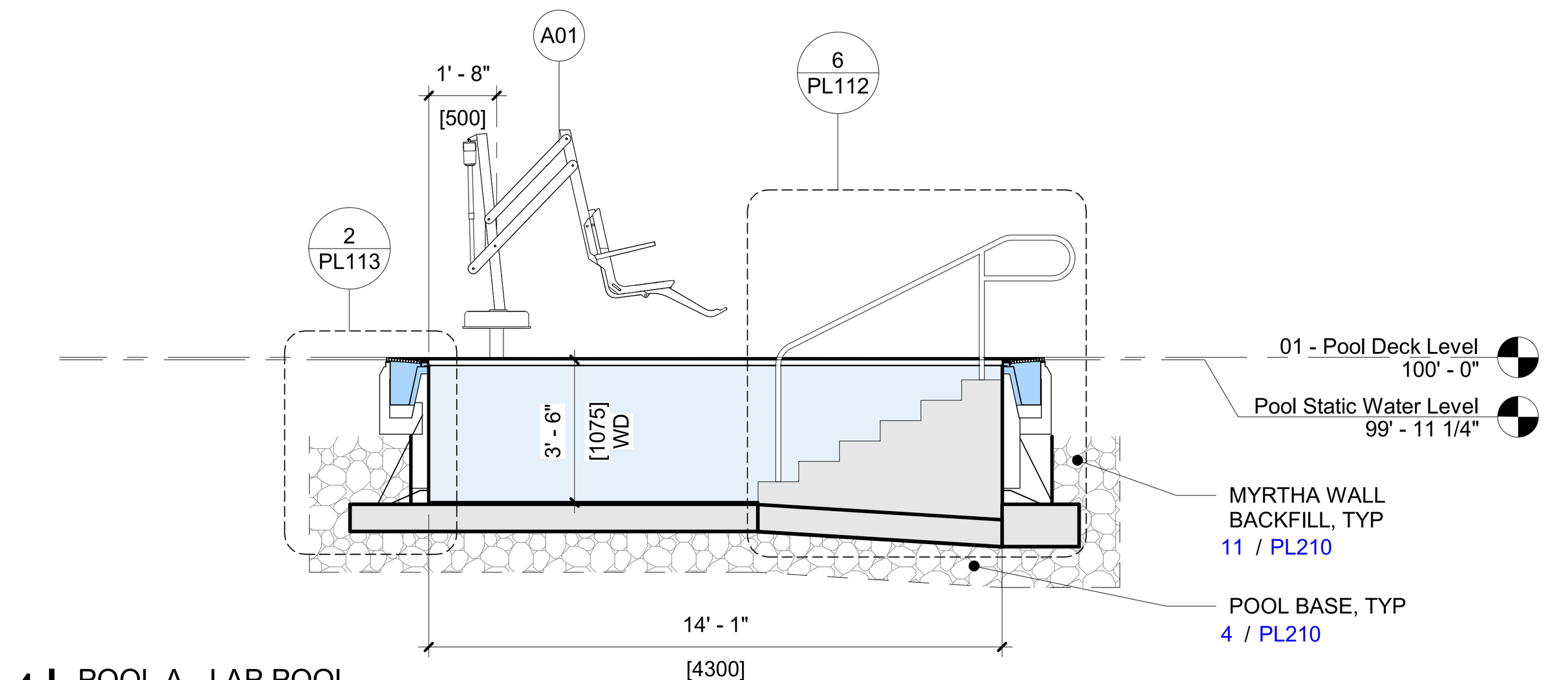
NAC NO	23458
DRAWN	MJC
CHECKED	GGA
DATE	04/16/2024

POOL A - LAP POOL
SAFETY
ENVELOPES

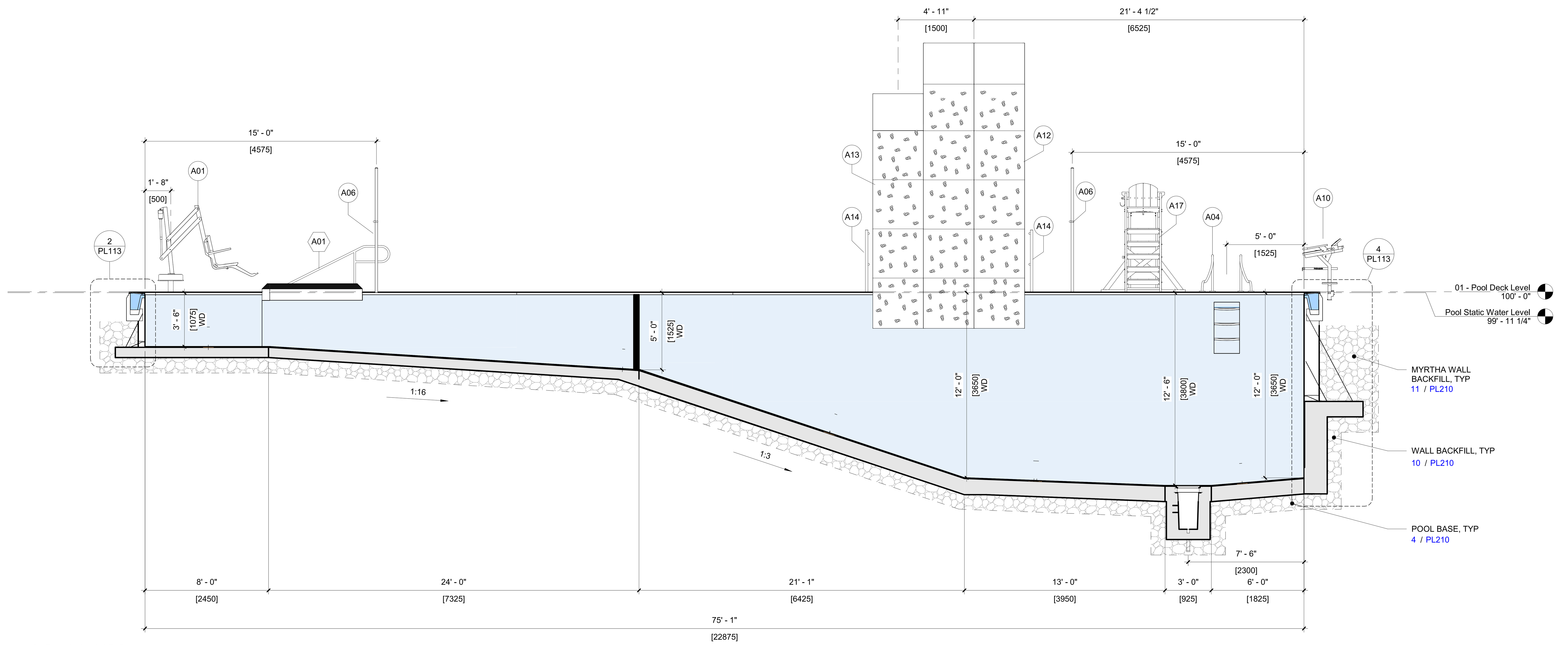
PL115

REVISIONS

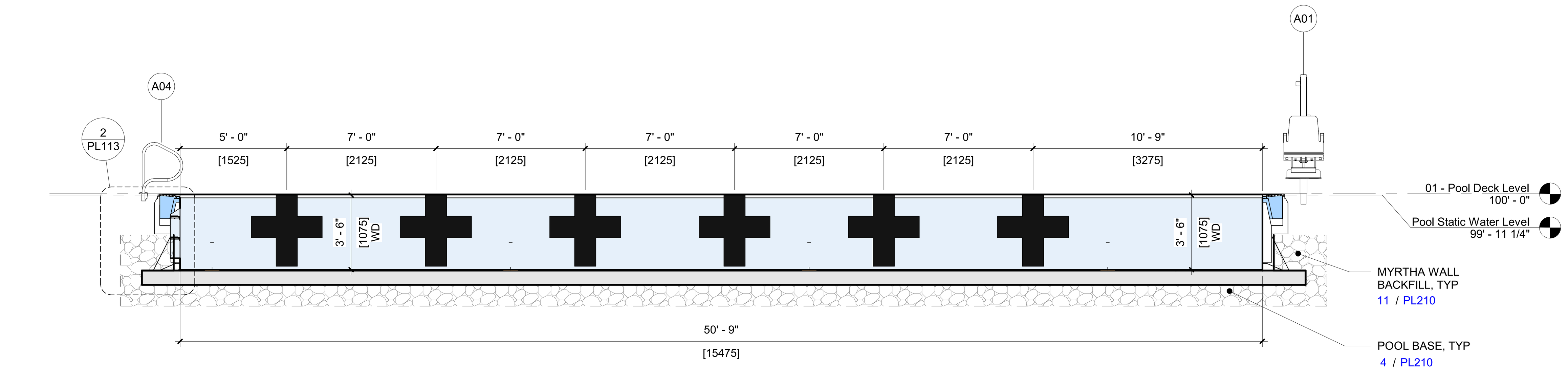
BID SET



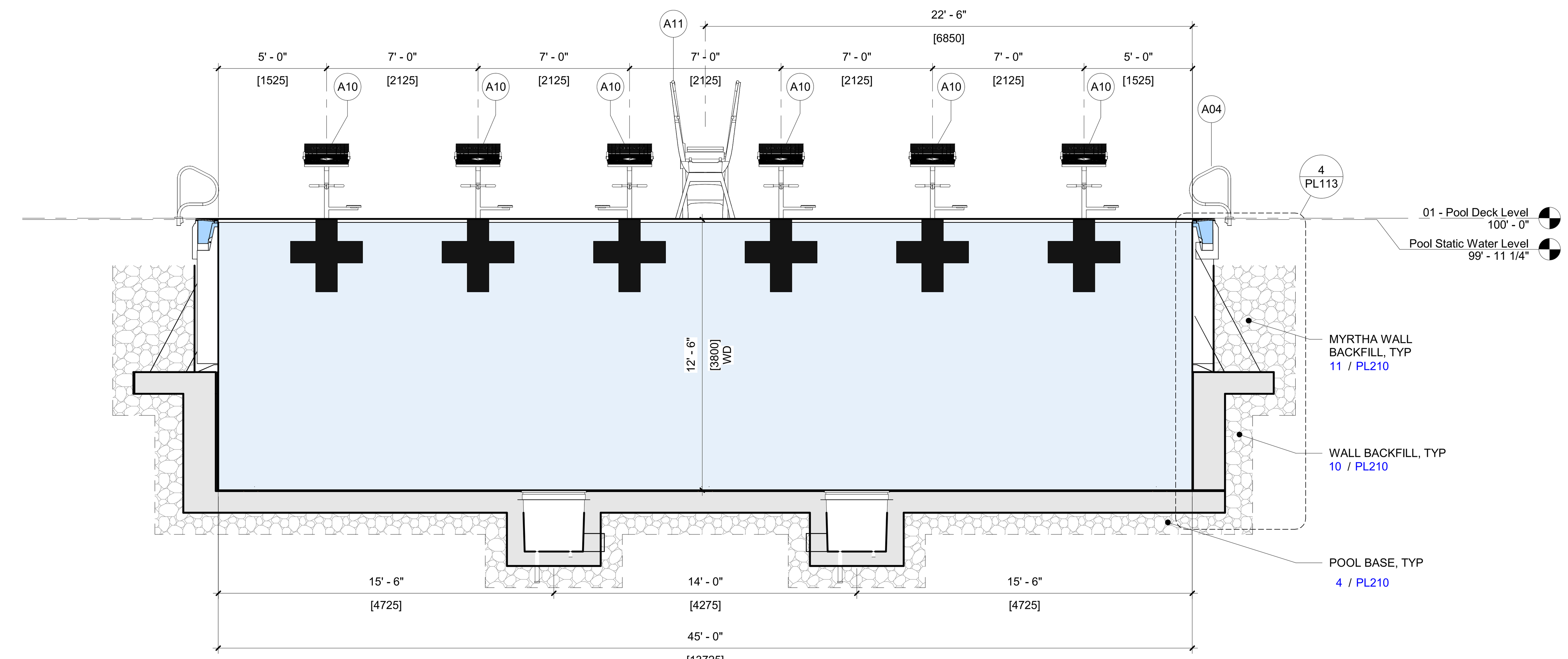
4 POOL A - LAP POOL SECTION VIEW
1/4" = 1'-0"



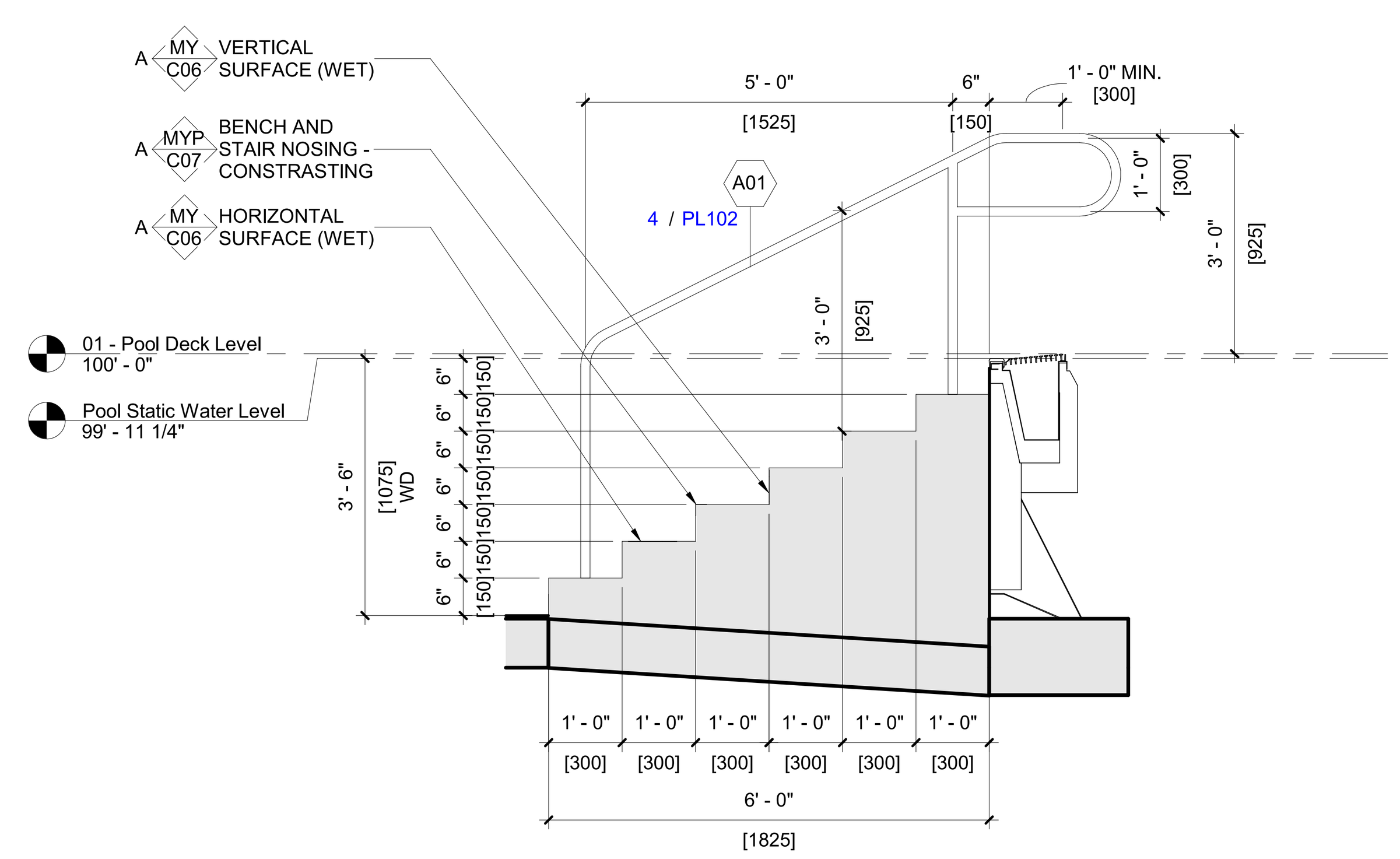
1 POOL A - LAP POOL SECTION VIEW
1/4" = 1'-0"



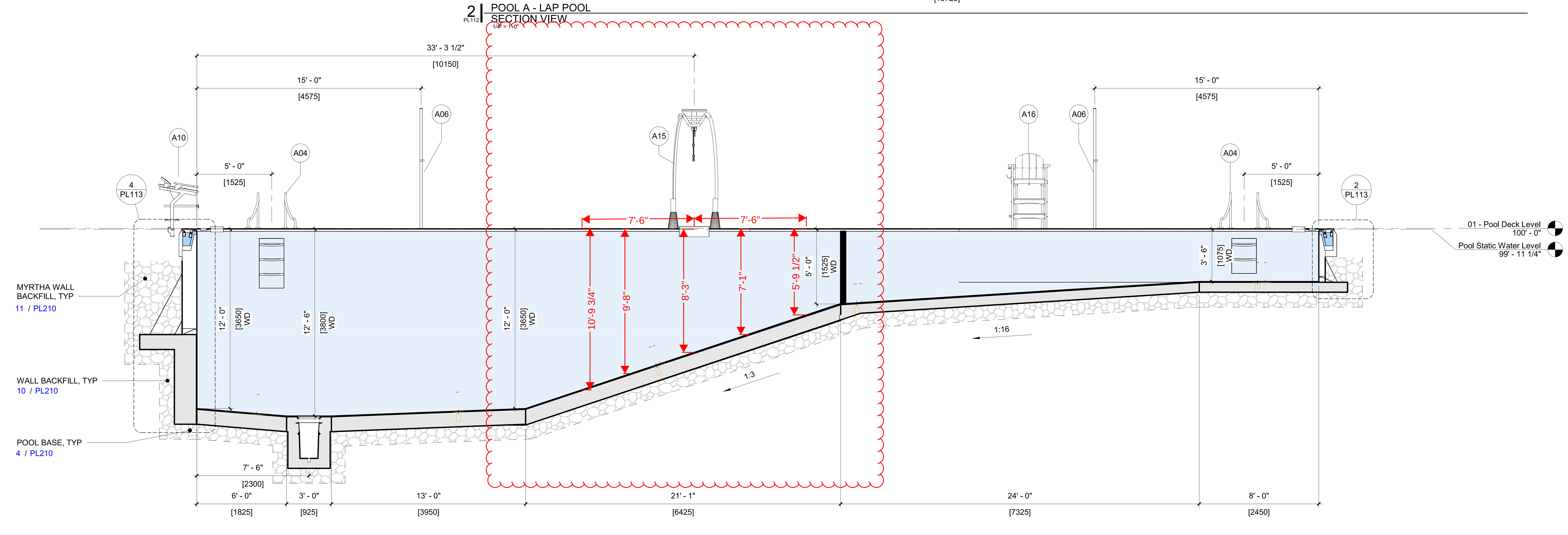
5 POOL A - LAP POOL SECTION VIEW
1/4" = 1'-0"



2 POOL A - LAP POOL SECTION VIEW



6 POOL A - LAP POOL STAIRS SECTION VIEW
1/2" = 1'-0"



3 POOL A - LAP POOL SECTION VIEW
1/4" = 1'-0"

WTI WATER TECHNOLOGY INC

World Leaders in Aquatic Planning, Design and Engineering
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CHENEY AQUATIC CENTER
711 CEDAR ST. CHENEY, WA 99004

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MATTHEW W. FRERY
STATE OF WASHINGTON

NAC ARCHITECTURE
nacarchitecture.com
1203 WEST RIVERSIDE AVE
SPOKANE WA 99201
P:509.838.8240

NAC NO	23458
DRAWN	MJC
CHECKED	GGA
DATE	04/16/2024

POOL A - LAP POOL SECTIONS

PL112

AQUAZIP'N®

Combining the thrill of a zip line with the fun of a rope swing

With only 4 feet of depth required, AquaZip'N® can easily be added as an exciting poolside adventure at:

- Camps
- Country Club
- Colleges/Universities
- Swim Clubs
- Recreation/Aquatic Facilities
- Health/Fitness Centers
- Military Wellness & Recreation
- Private Residences

POOLSIDE ADVENTURES™

PoolsideAdventures.com
800.956.6692
info@poolsideadventures.com

AquaZip'N®: A UNIQUE Poolside Adventure

With nothing like it on the market, AquaZip'N delivers poolside fun and excitement in a fresh new way. With this easy addition to your pool, you will drive demand from guests of all ages and increase your facility's programming capabilities on top of these benefits:

High Throughput

Launching riders into the water quickly, AquaZip'N keeps the line moving with a proprietary self-retracting trolley so kids can experience it again and again.

Position Anywhere

With a minimum water depth requirement of 4 feet, AquaZip'N can be added easily for thrilling poolside adventures in the shallow or deep end.

Minimal Footprint

AquaZip'N requires little deck space with its sleek frame that hangs out over the water and doesn't interfere with normal lap swimming. And with no water source required, it is an easy amenity to add.

Activates the Deep End

As a safer alternative or enhancement to diving boards, AquaZip'N attracts tweens and teens to those under-utilized, deep areas of a pool.

Easy to Install

The AquaZip'N 3-piece system comes pre-fabricated for quick assembly and installation at your facility on any pool gutter configuration.

100% Made in America

AquaZip'N is designed, engineered and manufactured in the USA to conform to all industry standards.

To learn how you can bring the adventure of AquaZip'N® to your facility, contact us today:

POOLSIDE ADVENTURES™

PoolsideAdventures.com | 800.956.6692 | info@PoolsideAdventures.com

Building Courageous Kids for Life's Great Adventure

AQUAZIP'N® SPECIFICATIONS

System Description

Deck mounted, overhead self-retracting pool rope swing. Components consist of Steel support structure, self retracting trolley system with handline. Manufactured off site. Designed to withstand chlorinated environments.

Components

Rope System

Rope system consists of a ⅝", 3-Strand Twisted, High Tenacity Polyester, Plied Yarn. High tenacity for durability, low stretch, superior UV resistance, excellent resistance to acids/chlorines. Attached to the Trolley using high density plastic connector and 3" stainless steel carabiner. See manufacturer's full specification for details.

Support Frame

The support frame shall be fabricated of 304 stainless steel sections powder coated in Glacier White, consisting of multiple bolt-together assemblies. The Frame height is 115" and maximum width of 39" with an overall length of 147" from back of structure to end of track.

Anchors

Anchors are to include either Hilti Chemical Anchors using Hilti HIT-HY 200 Adhesive—⅝" diameter or HAS-R stainless steel wedge anchor (or approved equivalent) with a 3-⅛" minimum embedment, (5qty anchors) per leg. Install anchors per manufacturer instruction.

Fasteners

All fixed connections: Bolts, Flat Washers, Nuts, are attached by grade 18-8 stainless steel or higher. Anchors will be 18-8 Stainless Steel or higher grade.

Trolley Cable Retraction Assembly

3/16" Dyneema 12-strand Cable

Warranty

AquaZip'N® is warrantied to the original purchaser to be free from defects in material and workmanship from the date of installation, during normal use and installation, with exclusions of cosmetic defects through wear and tear: Limited 2-Year Warranty

Design Recommendations

Deck & Gutter

The pool deck in the AquaZip'N® installation area should be as level as possible. If the pool has a coping greater than 1-½", or does not meet the standard base concrete requirements below, additional hardware components may be required. Please complete the Poolside Adventures™ Gutter Configuration Worksheet available on our website and contact a Poolside Adventures™ representative to determine the proper installation hardware and anchoring required.

Concrete Requirements

Standard length anchoring system requires a minimum concrete depth of 4" (with 6x6 W2.0 welded wire mesh ASTM A185) with 3000 psi rating or greater, embedded to a minimum depth of 3-⅛". See Hilti anchor requirements for further details. Further concrete requirements for proper installation includes a 4" thick, 6' wide (away from pool edge) of uninterrupted, un-cracked concrete slab section. Length (parallel with pool edge) of concrete slab can vary based on desired maximum rider weight:

- 8' long for 250 lbs rider load rating
- 7' long for 200 lbs rider load rating
- 6' long for 150 lbs rider load rating

Clearances & Safety Recommendations

Please contact a Poolside Adventures™ representative for current product information regarding pool depth and clearance zone recommendations based on the deck and configuration to be installed.

State certified engineered drawings and/or drawings specific to actual site installation details may be required for approval of AquaZip'N® installation. Standard structural engineering drawings are available at no charge. State or site-specific engineered drawings may be an additional cost. Please contact the appropriate local governing department for more information.

Poolside Adventures™ product guides, installation instructions, owner's maintenance guide and other resources are available at www.poolsideadventures.com or can be requested by calling 800-956-6692.

Operations Manual AquaZip'N

The new AquaZip'N design allows for minimal maintenance and high throughput. The following is the inspection checklist.

Daily Checklist:

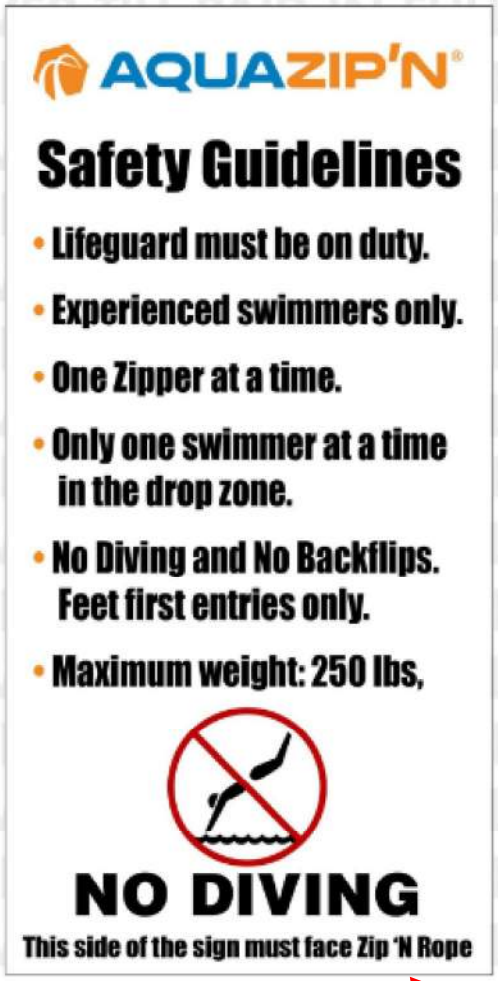
- Ensure proper trolley retraction by rolling trolley out over water, letting go and watching to see that trolley returns to original starting location.
- Check trolley wheels and bearings visually to ensure trolley is secure within its track.
- Visibly check retraction cable for wear & tear.
- Cable stretch is normal. However, if you notice the weight is contacting the bottom of the baseplate it is time to replace your retraction cable. Call Poolside Adventures at 800-956-6692 to order a replacement.
- Visibly check the rubber bumpers on the front and back of the track to ensure they are firmly in place and there is no visible cracking or imperfections.
- Spray silicone-based lubricant onto all wheel bearings to increase the smoothness and longevity of your trolley system.

Monthly Checklist:

- Inspect trolley to ensure secure attachments of retraction cable to trolley.
- Inspect hand rope for wear & tear.
- Inspect rubber bumpers on the front and back of the track for any cracks or imperfections. If any are found, please call Poolside Adventures at 800-959-6692 to order replacements.
- Check retraction cable for wear & tear.
    - Cable stretch and wear is normal. If you notice any significant wear on your retraction cable or if the weight is contacting the bottom of the baseplate when in operation it is time to replace your retraction cable. Call Poolside Adventures at 800-956-6692 to order a replacement.
- Check all bolts on the AquaZip'N structure to ensure they are firm & tight.
- Be sure acorn nuts are firmly secure on all threads able to be reached from the ground.
- Anchor bolts shall be taught to specifications.
- Inspect safety pad for visible signs of wear including cracks and gouges.

Seasonal/Annual Checklist:

- Remove trolley from track to complete thorough trolley inspection, ensuring all bolts are firm and all wheels and bearings are in good shape.
- Over time the wheels and bearings will need to be replaced. Call Poolside Adventures at 800-956-6692 to order replacement wheels.
- Store trolley indoors, in a cool dry location, during the off-season.
- Inspect concrete surface for cracking and weathering to which the PSI of concrete could become compromised.



"A" FRAME SIGN TO BE DISPLAYED AT ALL TIME THE AQUAZIP'N IS IN USE

Calculation Report

Hand Calculation on Projectile Analysis & Forces on the user

FEAmax LLC. Engineering Design, Analysis & Manufacturing Services.
Email: info@FEAmax.com; *Web*: www.FEAmax.com

5/3/2024

PROJECT INFO.

Change History:

Version Number	Date	Prepared by	Reviewed by	Contact
V 1.0	5/3/2024	Bill Bin	Frank Wang	Frank.Wang@feamax.com

CFD Requestor Info.:

Contact name:	Alex Salzman
Email:	Alex@PoolsideAdventures.com
Company name:	PYRAMIDE USA INC.
Address:	PO Box 530. Frederick, MD 21705

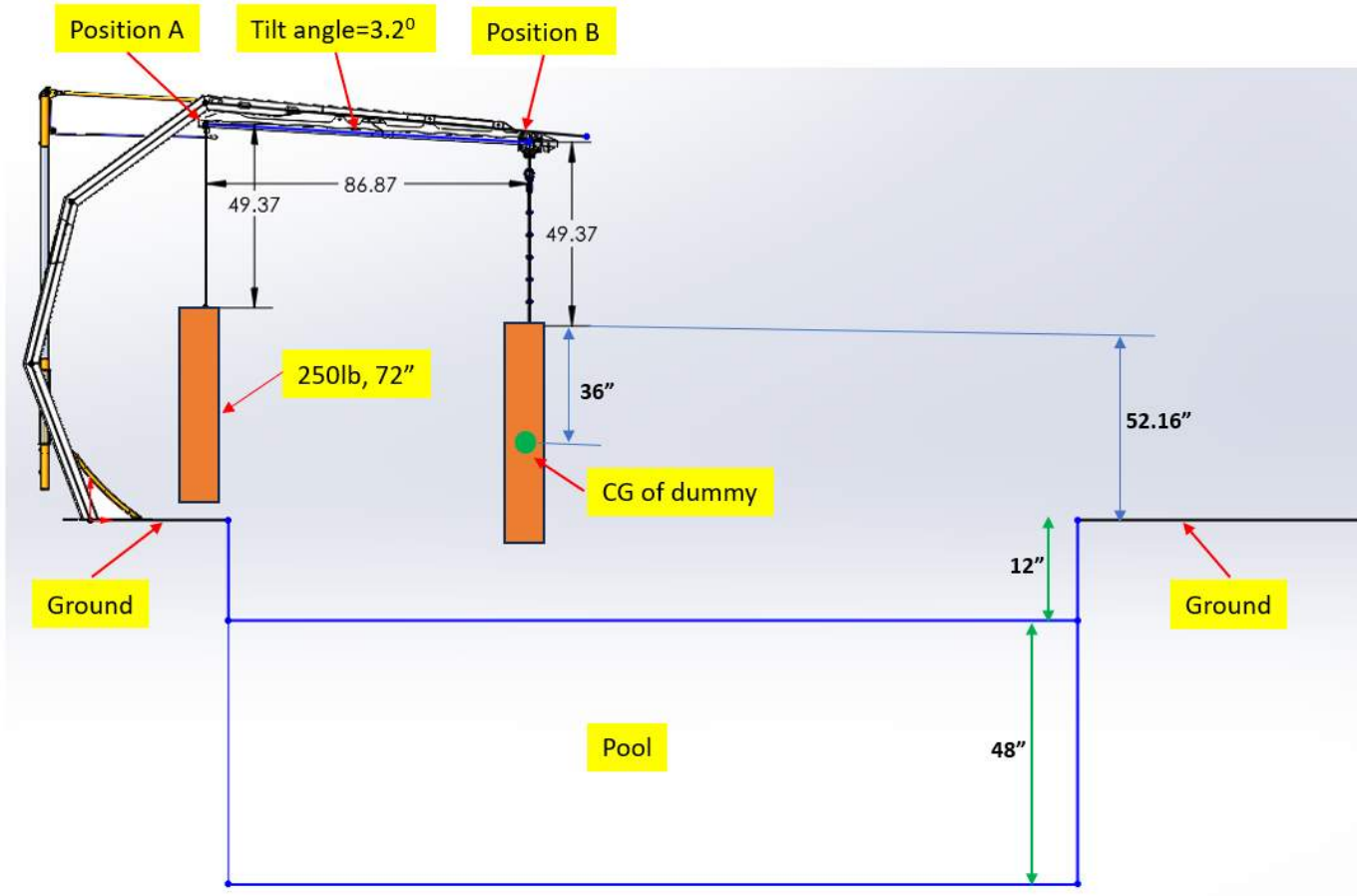
PROJECT DESCRIPTION

Project Description:

1. Perform hand calculations on the trolley system with the two cases.
2. The case #1 - Projectile Analysis: determine how far and how deep could a user go when launching from starting heights.
3. The case #2 - Forces on the user: determine the force on the user at beginning of ride and the end of ride.
4. The CAD model file for the calculation:
   - *Z0037C_V3.2 Master Assembly.SLDASM*
5. All related documents were received by 4/1/2024

CAD Model

1. The CAD model and the dimension information for calculation:



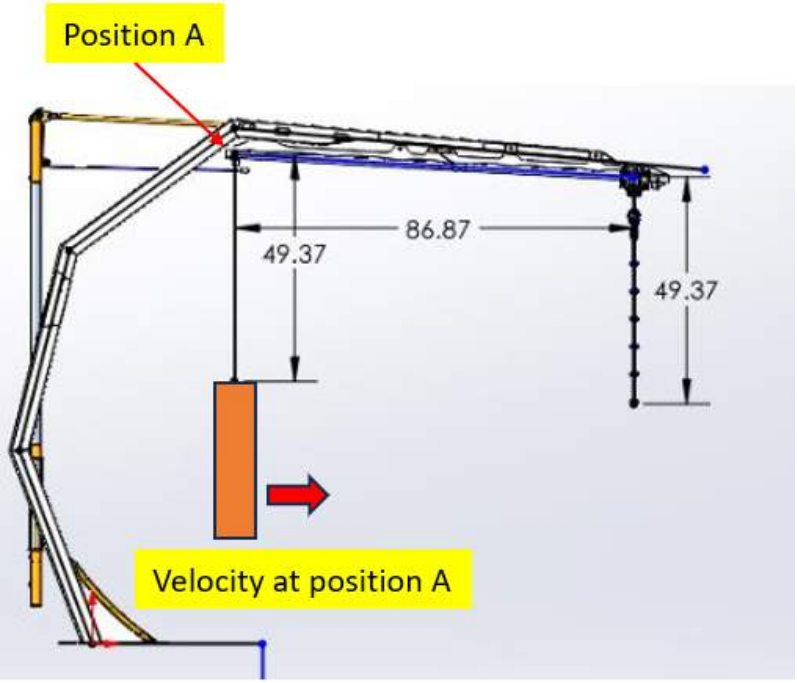
ASSUMPTIONS

Assumptions:

1. Assume a block/dummy on the rope with 250lbs mass and 6 feet height.
2. Assume the max jump forward distance is about 9.8 feet for a 250lbs adult from a standstill (worst case).
3. Considering the ideal condition, the person jumps at 45 degrees.
4. Assume it is frictionless contact at the top track rail.
5. Assume the 6 feet height dummy as a mass point at the CG (center of gravity).

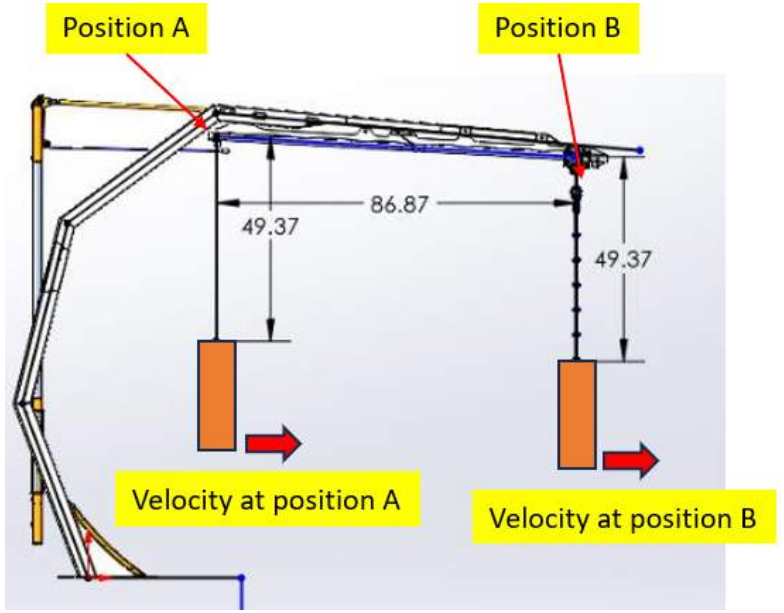
Calculation of initial velocity

1. Equations:
   - V x T = L
   - V = g x t / 2
   - In which: V is velocity, T is time, L is the length and g is the acceleration.
2. We have V = sqrt(L x g / 2), in which: L= 9.8 ft, g = 32 ft/s2
3. The calculated results:
   - The initial velocity at position A = sqrt(L x g / 2) = 12.56 ft/s



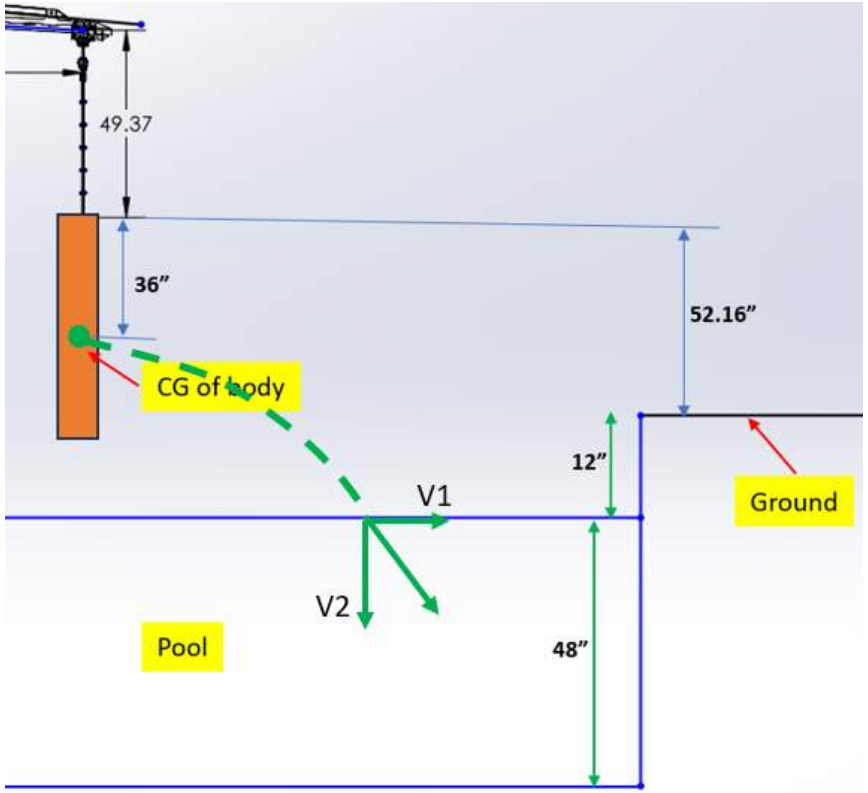
Item#1 – Projectile Analysis

1. Calculation#1 –velocity at position B:
   - Because of the frictionless contact and the tilt angle is only about 3 degrees between position A and B, we could assume the velocity at position B is the same as or very close to position A.
   - The velocity at position B = 12.56 ft/s
2. Calculation#2 – the moving distance before touch the water:
   - The initial horizontal speed V = 12.56 ft/s
   - The height above water (from CG of body to water) = 52.16+12-36 = 28.16 inch
   - The time before touch water t = sqrt(2L /g) = sqrt(2x28.16/32.15) = 0.38 s
   - The vertical velocity V2 = g x t = 12.33 ft/s
   - The horizontal velocity V1 = 12.57 ft/s
   - The moving distance before touch the water L = V1 x t = 4.75 ft



Item#1 – Projectile Analysis

3. Calculation#3 – the moving depth and distance in the water:
   - Equation: $F_d = 1/2 \cdot Cd \cdot \rho \cdot A \cdot v^2$
   - where:
   - F_d is the drag force, C_d is the drag coefficient, ρ is the density of the fluid (water is approximately 1000 kg/m3), A is the cross-sectional area of the object perpendicular to the flow of fluid, v is the velocity of the object relative to the fluid.
   - The drag coefficient (C_d) and the cross-sectional area (A) depend on the shape and orientation of the human body in the water. We'll need to make assumptions to proceed.



Item#1 – Projectile Analysis

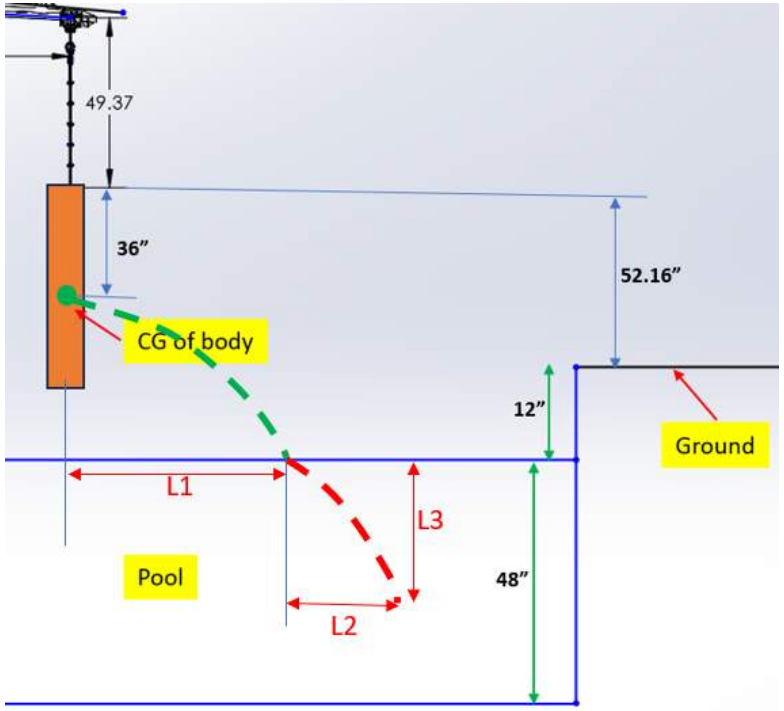
4. Calculation#4 – the moving depth and distance in the water:

- The depth and horizontal distance can be determined by integrating the motion equations under the influence of gravity and drag. However, the actual calculations can be very complex due to the non-linear drag force that depends on the velocity squared.
- Assume a constant average drag coefficient and ignoring buoyancy for the depth calculation, we can estimate the maximum depth and horizontal distance.
- Assume Cd=1.0 for a body position that is neither perfectly streamlined nor fully perpendicular to the flow. Assume cross-section area A=0.1 m^2, which is a rough estimate for a human body.
- Calculate the maximum depth and horizontal distance by considering the initial kinetic energy and the work done against the drag force. Distance = $\int_{V_i}^{0} \frac{1}{0.5Cd\rho Av} dv$ where v_i is the initial speed in the respective direction.
- The calculated maximum depth and horizontal distance the human can reach in water are approximately 0.84 meters.
- Note: these results are highly simplified. The actual values could differ significantly due to various factors such as the complex nature of drag in fluids, body orientation, and body shape effects.

Item#1 – Projectile Analysis

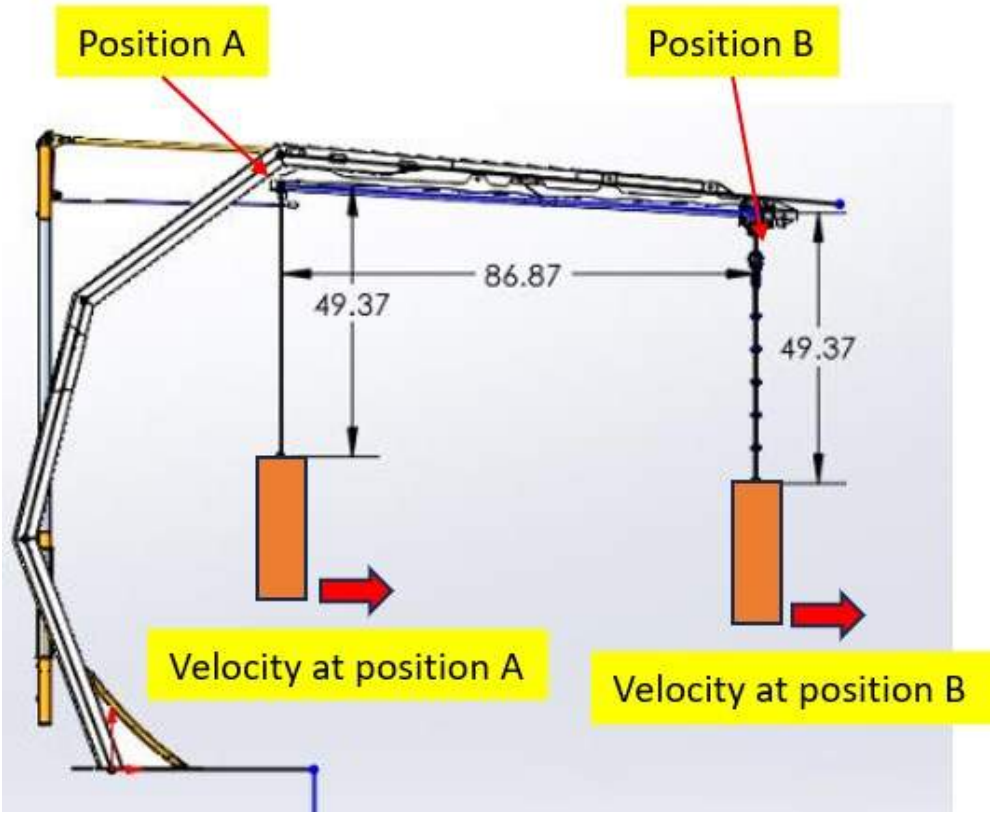
5. Calculation Results:

- Before touching the water, the body can move in horizontal direction L1 = 4.75 ft
- The max moving distance in horizontal direction in the water is about L2= 2.76 ft.
- The max depth in the water is about L3 = 2.76 ft.
- Note: if counting the body heigh 6ft, the max depth in the water would be 5.76 ft.



Item#2 – Forces on the user:

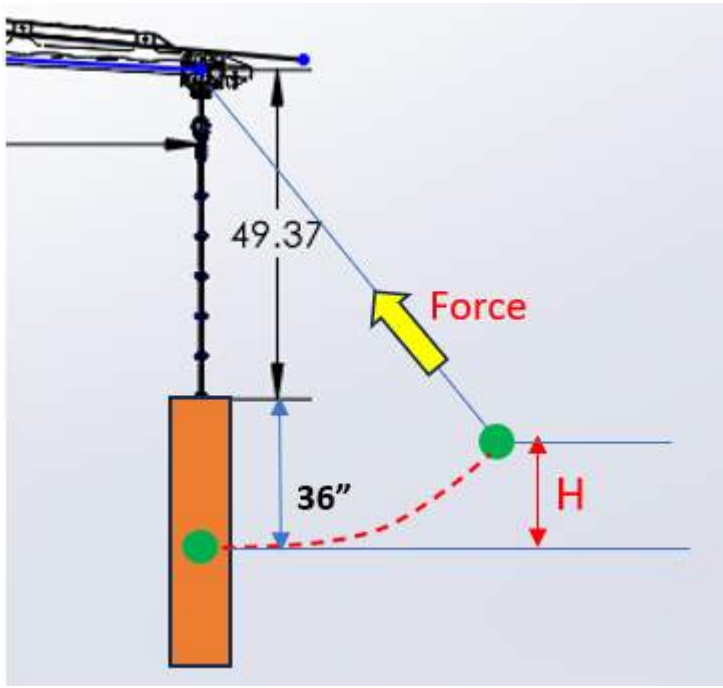
1. Calculation#1 – the max holding force on the user at position A:
   - Assume the body moves in horizontal direction, the initial holding force in vertical direction would be the same as the weight of user.
   - So, the max force on the user from rope at the beginning of ride (position A) is about 250 lbf.



Item#2 – Forces on the user:

2. Calculation#2 – the max holding force on the user at position B:

- Assume the user would hold the rope without release.
- The body would swing and cause higher force on the rope.
- Max force T_{max} = m x g + m x v^2 / r = 422 Lbf.
- The user swing height is about H = V^2 / 2g = 2.43 ft



3. Results:

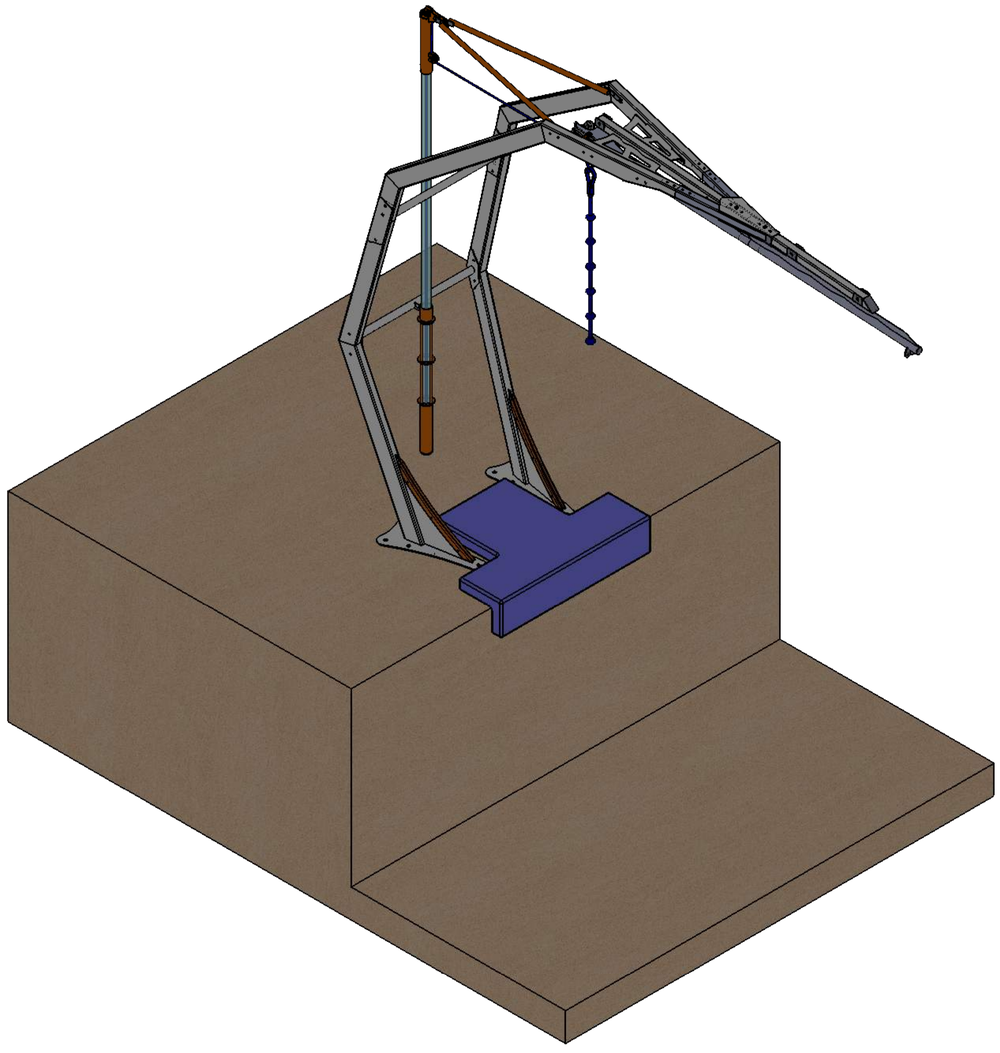
- The max force on the user (holding force on hands) from rope at the beginning of ride (position A) is about 250 Lbf.
- The max force on the user (holding force on hands) from rope at the end of ride (position B) is about 422 Lbf.
- The user can swing upward max height is about 2.43 ft.

REVISIONS			
REV.	DESCRIPTION	DATE	REV'D BY
A	Initial Release	7/27/2023	A. Salzman

Designed and engineered to the following standards:

- ASTM F2291-18 Amusement Rides and Devices
- ASTM F2461-18 Aquatic Play Equipment
- International Building Code (IBC) 2015 and ASCE 7, Minimum Design Loads for Building and Other Structures
- AISC Manual of Steel Contruction, 13th Edition
- ASD and Steel Design Guide 27 - Structural Stainless Steel

***Full structural analysis and stamped fabrication drawings available upon request



AquaZip'n V3.1 Architectural Guide

DESIGN

POOLSIDE ADVENTURES™

P.O. BOX 530
Frederick, MD 21705

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FAX: +1 240.575.6020
EMAIL: info@poolsideadventures.com

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ANGULAR: MACH ± 1 BEND ± 1
TWO PLACE DECIMAL ± .03 [0.76]
THREE PLACE DECIMAL ± .005 [0.127]
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	NAME	DATE
DESIGNED		
DRAWN		
CHECKED		
ENGINEERED		

COMMENTS:
-NONE-

PROJECT:

TITLE:

SIZE	DWG. NO.	REV
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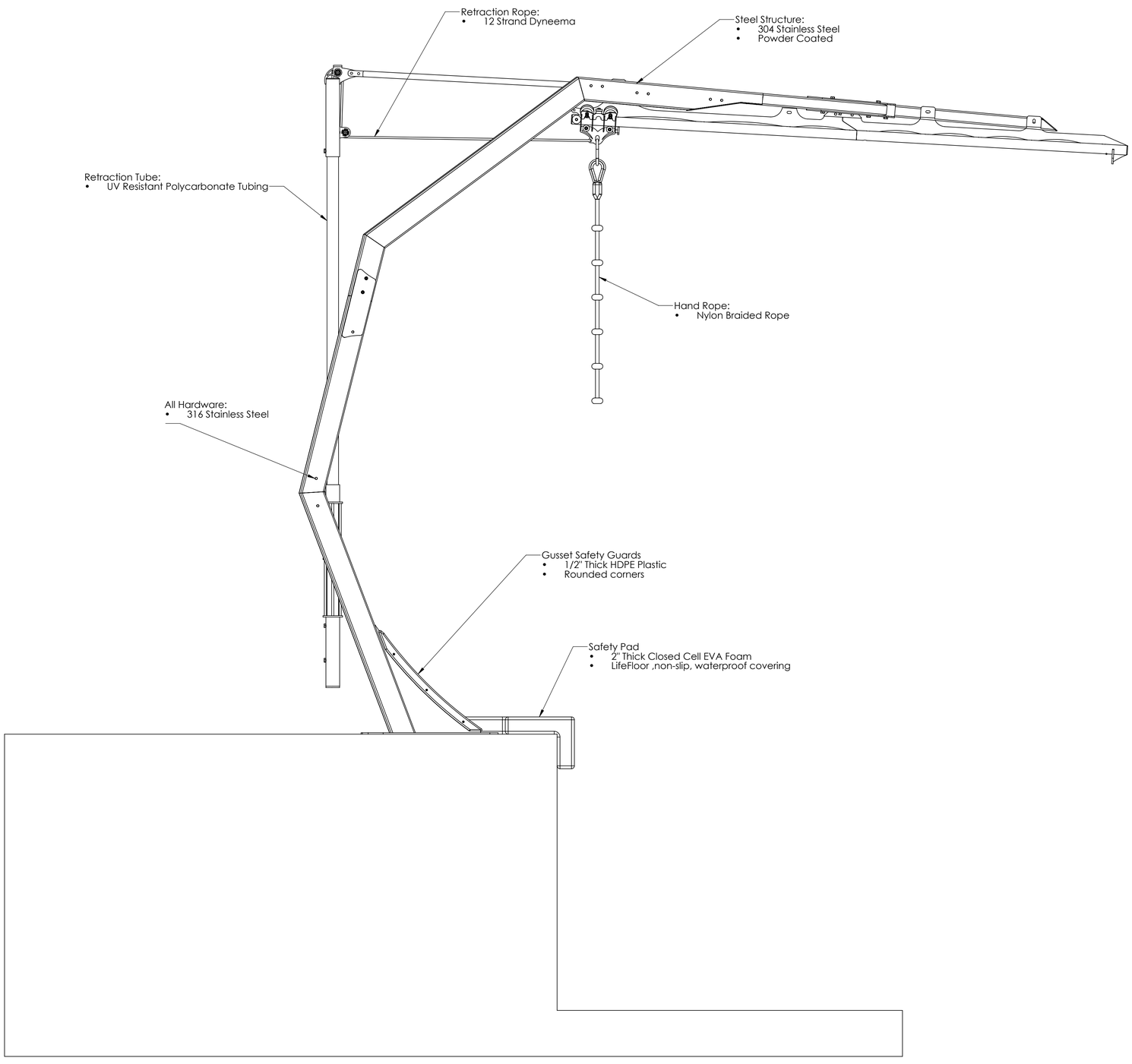

Material Specs

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WELDS: 1/4

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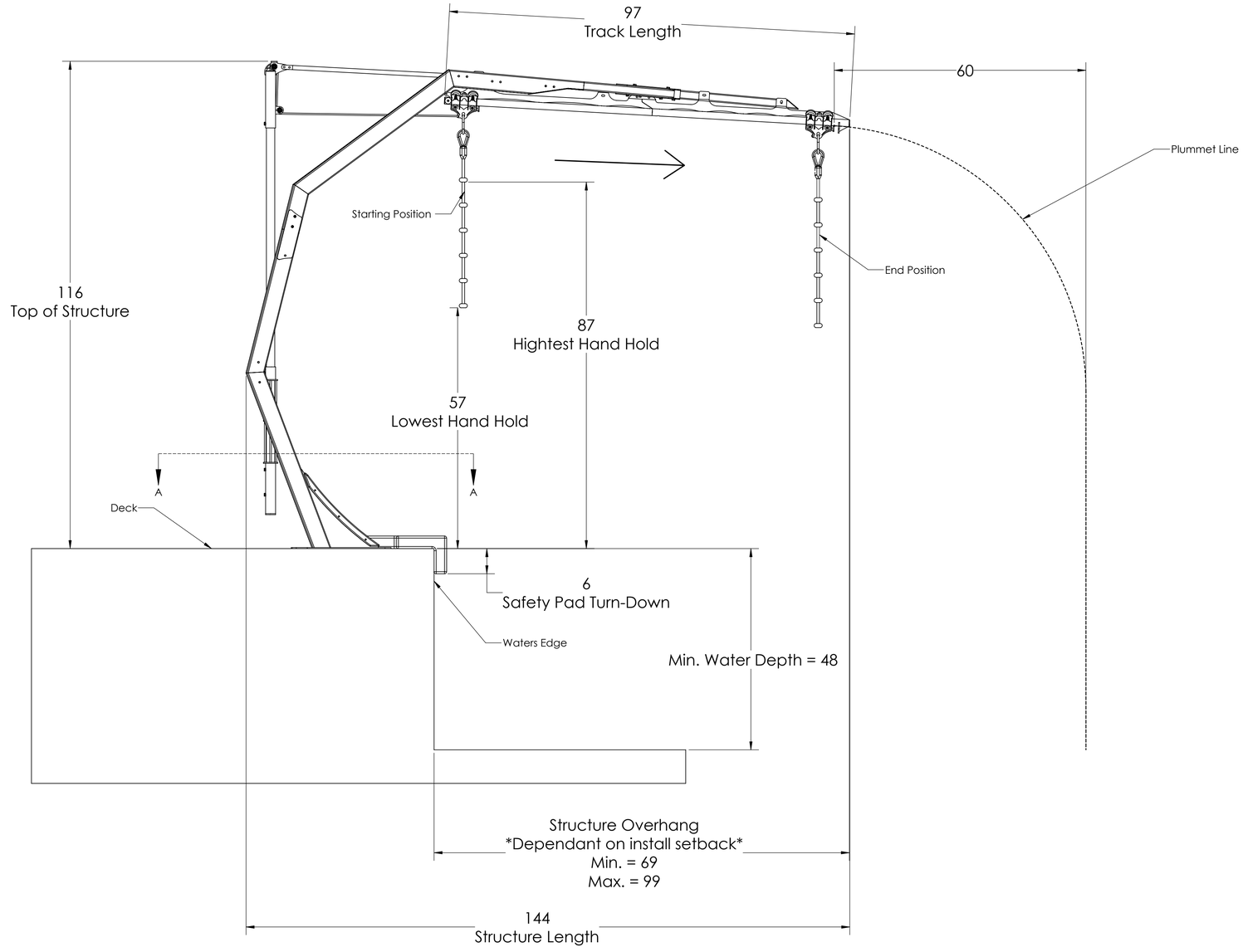
SCALE: 1:1 WEIGHT: 25799.23 SHEET 2 OF 7



Elevation View/Water Depth Req.

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WELDS +1/16 -0

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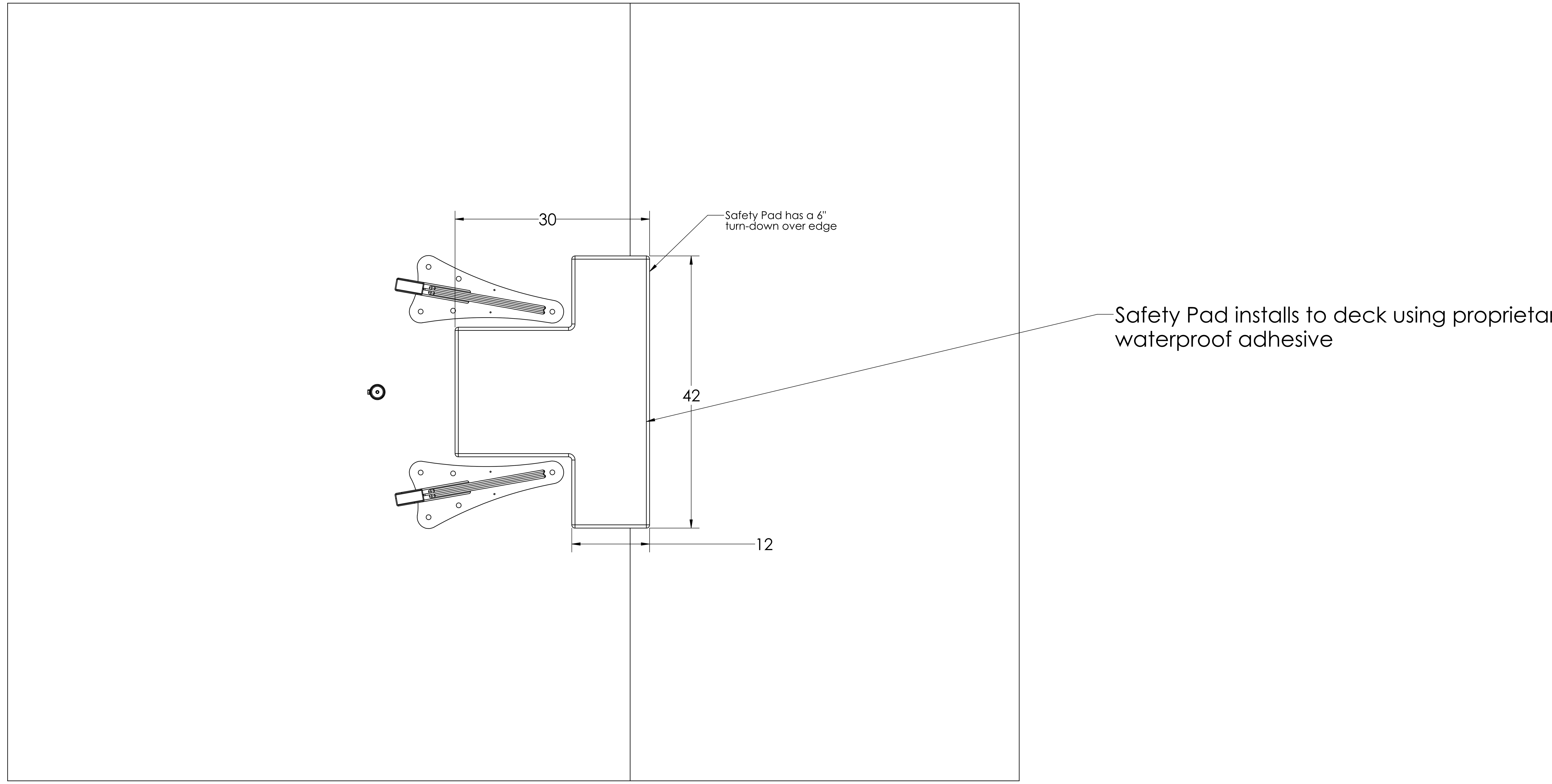
SCALE: 1:1 WEIGHT: 25799.23 SHEET 3 OF 7



SECTION A-A
SCALE 1 : 10

Safety Pad Dimensions

***Custome safety pads available upon request
to work with any gutter system***

Safety Pad Details

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TWO PLACE DECIMAL ± .02 [0.51]
THREE PLACE DECIMAL ± .005 [0.127]
WELDS: +1/16 -0

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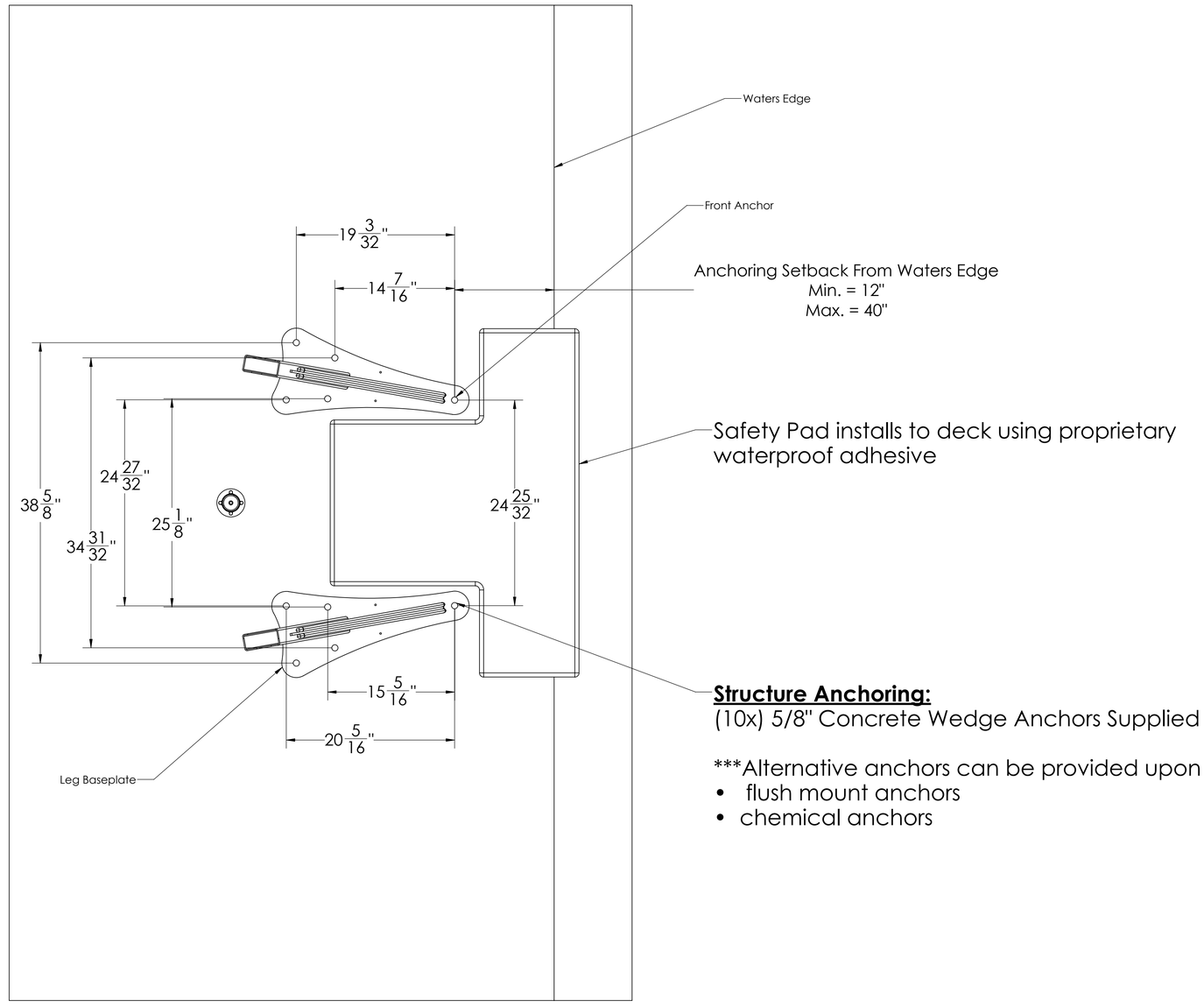
SCALE: 1:1 | WEIGHT: 25799.23 | SHEET 4 OF 7



SECTION A-A
SCALE 1 : 8

Structure Anchoring:
(10x) 5/8" Concrete Wedge Anchors Supplied

***Alternative anchors can be provided upon request:
- flush mount anchors
- chemical anchors

*****Anchor dimensions are for reference only, not to be used for installation. Anchor installation is done by using the Leg Baseplates themselves as drilling templates.*****

Anchoring Details

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TWO PLACE DECIMAL ± .02 [0.51]
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WELDS: ±1/16 in

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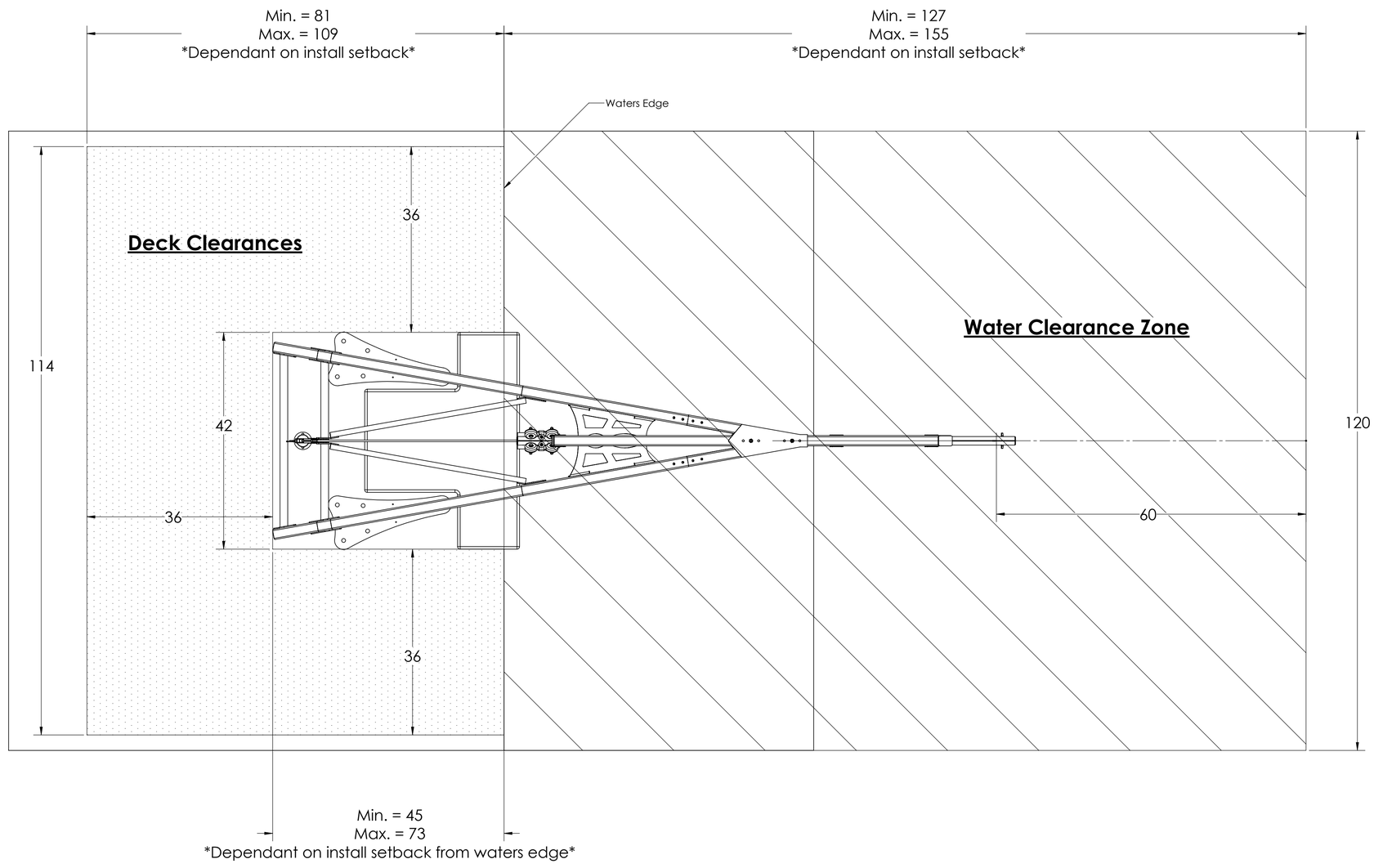
SCALE: 1:1 WEIGHT: 25799.23 SHEET 5 OF 7



Water and Deck Clearances

THIS DRAWING HAS BEEN GENERATED AND IS MAINTAINED BY A CAD SYSTEM. CHANGES SHALL BE INCORPORATED AS DIRECTED BY THE DESIGN ACTIVITY

UNLESS OTHERWISE SPECIFIED:

DIMENSIONS ARE IN INCHES [mm]
TOLERANCES:
FRACTIONAL ± 1/16
ANGULAR: MACH ± 1 BEND ± 1
TWO PLACE DECIMAL ± .02 [0.51]
THREE PLACE DECIMAL ± .005 [0.127]
WELDS: ~1/4

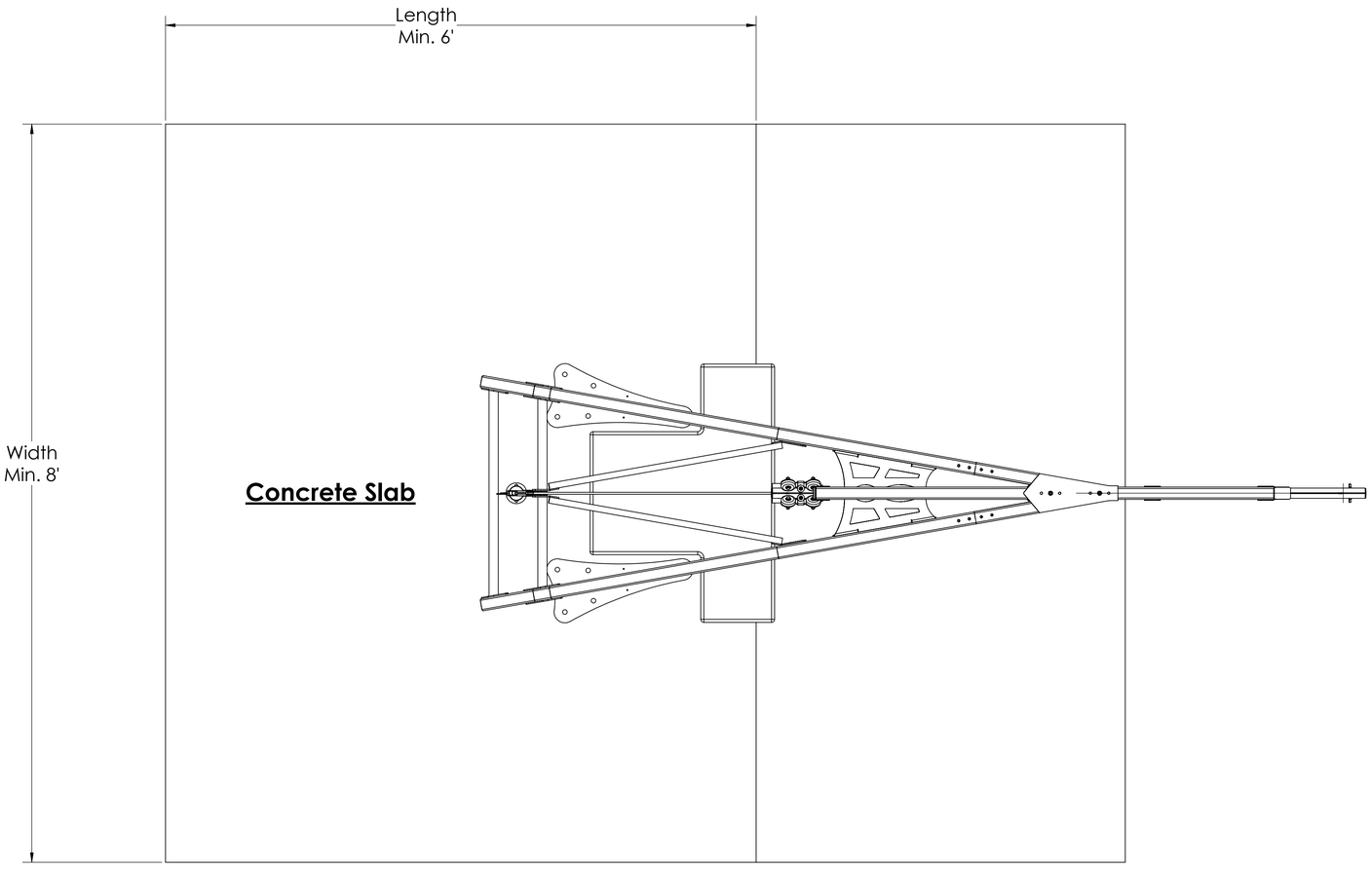
DO NOT SCALE DRAWING

Poolside Adventures
P.O. BOX 530
FREDERICK, MD 21705

TITLE:

SIZE	DWG. NO.	REV
D	Z0037C_V3.1 Architectural	

SCALE: 1:1 WEIGHT: 25799.23 SHEET 6 OF 7



Min. concrete thickness = 4"

Notes:
1. Location of front anchors no closer then 1' to front edge of pad.
2. Concrete dimensions shown are to acheive a min. required square footage. Alternative Lengths and widths can be accepted upon review.
3. Concrete width to be centered on AquaZip'n Frame.
4. Min. concrete thickness of 4" required, with 6x6 W2.0 welded wire mesh ASTM A185.
5. If concrete is new, minimum strength of 3000psi at 28 days is required.

Concrete Slab Requirements

THIS DRAWING HAS BEEN GENERATED AND IS MAINTAINED BY A CAD SYSTEM. CHANGES SHALL BE INCORPORATED AS DIRECTED BY THE DESIGN ACTIVITY

UNLESS OTHERWISE SPECIFIED:

DIMENSIONS ARE IN INCHES [mm]
TOLERANCES:
FRACTIONAL ± 1/16
ANGULAR: MACH ± 1 BEND ± 1
TWO PLACE DECIMAL ± .02 [0.51]
THREE PLACE DECIMAL ± .005 [0.127]
WELDS +1/16 -0

DO NOT SCALE DRAWING

Poolside Adventures
P.O. BOX 530
FREDERICK, MD 21705

TITLE:

SIZE D | DWG. NO. Z0037C_V3.1 Architectural | REV

SCALE: 1:1 WEIGHT: 25799.23 SHEET 7 OF 7

ASTM F2291-18 & ASTM F2461-18 STAMPED REVIEW

ZIP'N 3.0 PROTOTYPE

FRAMING AND COMPONENT DESIGN

BASE ANCHORS TYPE REVISED

Prepared For:
Pyramide USA, inc.
8 East 2ND Street
Frederick, MD 21701

WBCM PROJECT NO. 23.0171.00
Date: 06/29/2023
REV: 08/03/2023

WBCM
WHITNEY, BAILEY, COX & MAGNANI, LLC
100 Sterling Parkway – Suite 108 Mechanicsburg, PA 17050
MAIN (717) 691-4708 FAX (717) 691-4749

Zip'N 3.0 PROTOTYPE

Design Criteria:

Loading:

- **Live Load:**
  250 lbs MAX Point Load (based on 1 user/rider)
  Deflection Limit L/360

Material:

- Pipes and plates – A304 Stainless Steel, F_Y=30 ksi, Fu = 75 ksi
  Pipe – ASTM A312 Standard Spec. for Seamless, welded & Heavy Cold worked Austenitic Stainless Steel Pipe
  Tubing – ASTM A554 Standard Spec for Welded Stainless Steel Mechanical Tubing
- Bolts - ASTM F593 Type 304 Stainless steel bolts

References:

- ASTM F2291-18 Amusement Rides and Devices
- ASTM F2461-18 Aquatic Play Equipment
- Applicable provisions of International Building Code (IBC) 2015 and ASCE 7, Minimum Design Loads for Buildings and Other Structures
- AISC Manual of Steel Construction, 13th Edition, ASD and Steel Design Guide 27 – Structural Stainless Steel

NAC
ARCHITECTURE

Patty Hayes, Board Chair
Washington State Board of Health
PO Box 47990
Olympia, WA 98504-7990

CHENEY AQUATIC CENTER — Variance Letter Date: 2024.07.17

PROJECT IDENTIFICATION: Lap Pool #: SR009200 Leisure Pool #: SR009201

On Behalf of:
Cheney Aquatic Center, City of Cheney
Owner Contact: Dan Curley Phone: 509-498-9293
Owner Address: 609 2nd Street Cheney, WA 99004
Facility Address: 115 North 8th Street (formerly 711 Cedar Street), Cheney, WA 99004

Owner Representative: Brooke Hanley (NAC Architecture) 509-838-8240

Variance Request Contact:
NAC Architecture: Brooke Hanley Phone: 509-838-8240 Email: bhanley@nacarchitecture.com

Facility Information:
Cheney Aquatic Center - Project includes an outdoor 6-lane 25-yard lap pool & separate leisure pool with zero-entry, spray features, & lazy river. The pool building with locker rooms, lifeguard offices, party room, and mechanical spaces is about 5000sf. The entire facility is lifeguarded and enclosed securely.

Plan Submittal: Drawing Plans have been submitted for review.

Variance Request Citation:
WAC 246-262-160 states *the board may grant a variance from requirements of chapter **246-262** WAC if, in the sole discretion of the board, data and/or research provides sufficient evidence that the RWCF (attraction, device, equipment, procedure, etc.), will adequately protect public health and safety, as well as water quality.*

Variance Request: Code language related to Diving Envelope (WAC 246-262-010(21) & WAC 246-262-060(5)(vi)) for the **NinjaCross Obstacle Course** attraction.

Items noted in review comments include:

- **NinjaCross Obstacle Course** attraction receiving pool shall conform to the CNCA or FINA standards (depth application and setbacks)

In the Spokane Regional Health District review response issued by Steve Main dated May 24, 2024, Steve requests NAC Architecture (NAC) and WaterTechnology, Inc. (WTI) address important concerns regarding public safety related to the receiving pool for the proposed **NinjaCross Obstacle Course** attraction in

Pool B. The concern is to address the minimum depth of the pool to be compliant with the WAC 246-262-010(21) & WAC 246-262-060(5)(c)(vi) regarding diving envelopes for features where users enter the water from above the water surface.

On behalf of the City of Cheney, WA; NAC & WTI respectfully requests your consideration of the current pool depth design at the NinjaCross for the future Cheney Aquatic Center. To support this request we provide the attached information, engineering exhibits, and following commentary:

- The review letter states that the "diving envelope" from WAC 246-262-010(21) applies to **all attractions** where users enter above pool water level and therefore requires the CNCA (enter less than 20" above the water surface) or FINA (enter 20" or greater above the water surface) water depths. We submit that the attached independent engineering calculations for the **NinjaCross Obstacle Course** will demonstrate that the manufacturer's required water depths and the designed water depths provided at the Cheney Aquatic Center are sufficient to protect the safety of the users allowed to participate in this attraction. Calculations were completed for users ranging in height from 51" tall up to 72" tall, and weight ranging from 58lbs to 275lbs. The minimum user height is 48" and the maximum weight is 275lbs. The manufacturer's minimum depth requirement is 3'-6" feet depending on the obstacles purchased for the system. The current Cheney receiving pool water depth ranges from 3'-9" to 4'-0", which exceeds the minimums recommended. Please review the attached engineering calculations in support of using the manufacturer's depth requirements in lieu of the CNCA or FINA diving envelope dimensions. See page 14 for a graphic section depicting an average user height compared and their position in or above the water using each obstacle, in most cases a participant's feet will be submerged or right at the surface of the water. In these calculations, if a person were to drop into 3'-6" deep water from a height of 20" above the surface, the heaviest user would contact the pool floor feet-first with a force equivalent to contacting the ground after a 3.4" high jump on pavement. Quote from review letter, "The participant is expected to contact the pool bottom in a manner that is consistent with any shallow pool activities." The current design of the receiving pool exceeds these calculation assumptions by providing deeper water than the minimum required and will be lifeguarded to prevent people from incorrectly using the obstacles.

- WAC 246-262-060(5)(c)(vi) appears to apply specifically to "diving envelopes in pools or areas of pools designated for diving activities". The applicant submits that diving activities are generally defined as plunging into the water headfirst. Diving headfirst into water results in the need for deeper water to avoid a head & neck collision with the bottom of the pool which is different than a feet-first or tucked entry plunge where the body is significantly slowed in the first 2 feet of water (as noted by the calculations). The **NinjaCross Obstacle Course** safety guidelines (provided in the exhibits) will note that users are required to enter the water in a feet-first manner. Diving from the unit is prohibited. The engineering calculations completed also assumes a feet-first plummet into the water. As users traverse the obstacles, they will generally have their feet dragging in the water and would not drop

from a height above the water that is any different from stepping into the pool from the deck edge, see page 14.

- The Model Aquatic Health Code also addresses the complexity of “other aquatic features” like this and would suggest that the manufacturer recommendations for design and operation would be adequate to install the feature.

  4.12.10[A] Other Aquatic Features Other AQUATIC FEATURES not otherwise addressed in the CODE, including but not limited to climbing walls, inflatables, and play structures, shall not be installed unless designed and operated in accordance with all manufacturer’s installation and operations recommendations.

- ‘A-frame’ signs with all written safety guidelines will be publicly displayed near the NinjaCross (see page 100 for example) to meet the criteria of WAC 246-262-070(10).

- Safety padding rated for falls from 6ft or less are provided around the base of the truss structure and down the face of the pool wall to prevent injuries at the corner of the gutter. The entire leisure pool floor is also covered with a ¾” “SoftWalk” material that is available for Myrtha pools which provides a small amount of cushion between the concrete floor and the Myrtha floor membrane and is rated for falls from a height of 1ft per ASTM F1292-04 testing.

- This pool will be lifeguarded at all times while in operation and the lifeguard staff will be the first line of defense to screen bathers to make sure they are experienced swimmers, instruct swimmers on proper use of the attraction, and direct proper swimmer circulation to and from the activity within the pool to avoid congestion or collisions. The **NinjaCross** will have a dedicated lifeguard to closely supervise the safety of swimmers when the attraction is open for use.

- The **NinjaCross** has also been designed and engineered to meet the following standards: Where applicable, NinjaCross follows guidelines from the MAHC (model aquatic health code). As for ASTM, NinjaCross has registered their products as fitness/sporting goods equipment which fall under ASTM F2461-18 Section 1.3.8 Exclusions "1.3.8 Sports equipment, fitness equipment, and diving equipment." This system’s patents and trademarks are registered under Sporting Goods & Fitness equipment and is not classified as an Amusement Ride.

- The City of Cheney is dedicated to making this facility fun while also as safe as possible for their community members and patrons. During community outreach activities, the citizens of Cheney specifically requested a pool design that would have a variety of intriguing activities in varying water depths for users of all comfort levels in the water. Deep water pools come with their own safety risks and lifeguarding challenges. Rescues are much more likely to be needed in deep water where a bather in trouble cannot push off the bottom of the pool to bob back above the surface quickly until the lifeguard can assist them. Shallow water is easier to supervise and guard, so offering additional ways to activate the shallow water areas in a documented safe manner is important for this facility. Many aquatic centers across the country are replacing their lily pad crossing activities (a similar obstacle

course feature that only requires 3’-6” to 4’-0” deep water) with the NinjaCross obstacle course because it has been deemed safer than having the lily pad floatables anchored to the floor and permanently obscuring the view of the water below the pads from lifeguard supervision. The NinjaCross obstacles do not have those same supervision issues.

- NinjaCross Systems also provided a list of installed projects across the U.S. and included their receiving pool design depths for reference. Photos of people using the obstacles are also provided for reference. They depict the intention of use where some or most of the swimmer’s body will be submerged in the water depending on the height of the obstacle. **See pages 7-8.**

- NAC submits that the design as described above and substantiated in the attached documentation meets the intent of providing a safe receiving pool for the **NinjaCross Obstacle Course** feature. NAC, WTI, and the City of Cheney respectfully requests a variance accordingly. If the State Board of Health has any follow-up conditions or actions required of the owner/operator, we are committed to reviewing them for implementation.

NAC Architecture (NAC) has teamed with Water Technology (WTI) on numerous aquatic projects and so we have a history of producing these projects successfully. WTI has been designing Aquatic venues for over 40 years. WTI is widely known in the industry as one of the leading aquatic design firms in North America. As one of the industry’s leaders, WTI has represented the waterpark industry during CPSC meetings on review of VGB rules and has also been involved in reviewing/editing sections of the MAHC. They are also represented in the Washington DOH committee to update the existing administrative code to adopt a more comprehensive aquatic code like the MAHC. The NAC and WTI commitment to safe aquatic facilities is proven. The design of the receiving pool at the **NinjaCross Obstacle Course** for the Cheney Aquatic Center will not put the health and safety of the public at risk. The City of Cheney, having operated a public pool for many years is experienced and committed to the safety and the welfare of their patrons.

On behalf of the City of Cheney, NAC Architecture would like to thank you for your consideration of this Variance Request. Please feel free to contact me with any questions you may have regarding this request.

Thank you,

Brooke Hanley

Brooke Hanley, AIA, Principal Architect, NAC Architecture

Attachments:

- NinjaCross Safety Information and Fall Zone Engineering, including a floor plan and section of the receiving pool for the Cheney Aquatic Center.

REVISIONS

SCHEDULE - CUSTOM RAILGOODS - POOL B

POOL ID	EQUIPMENT ID	EQUIPMENT	QTY	MANUFACTURER	DESCRIPTION
B	01	HAND RAIL	5	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CUSTOM FABRICATED, 316L SS, 1.50" OD x .120 WALL THICKNESS, 500 GRIT FINISH MIN.
B	02	BARRIER RAILING	1	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CUSTOM FABRICATED, 316L SS, 1.50 OD X .120 WALL THICKNESS, 500 GRIT FINISH MIN BARRIER RAILING WITH HTTP KNOTLESS NETTING. PROVIDE 1 3/4" INCH SQUARE MESH. COLOR BY OWNER/ARCHITECT
B	03	BARRIER RAILING	1	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CUSTOM FABRICATED, 316L SS, 1.50 OD X .120 WALL THICKNESS, 500 GRIT FINISH MIN BARRIER RAILING WITH HTTP KNOTLESS NETTING. PROVIDE 1 3/4" INCH SQUARE MESH. COLOR BY OWNER/ARCHITECT

REFER TO MYRTHA DRAWINGS FOR EQUIPMENT INCLUDING BUT NOT LIMITED TO:

HANDRAIL ANCHORS
INWALL STEPS

SCHEDULE - WATER FEATURE - POOL B

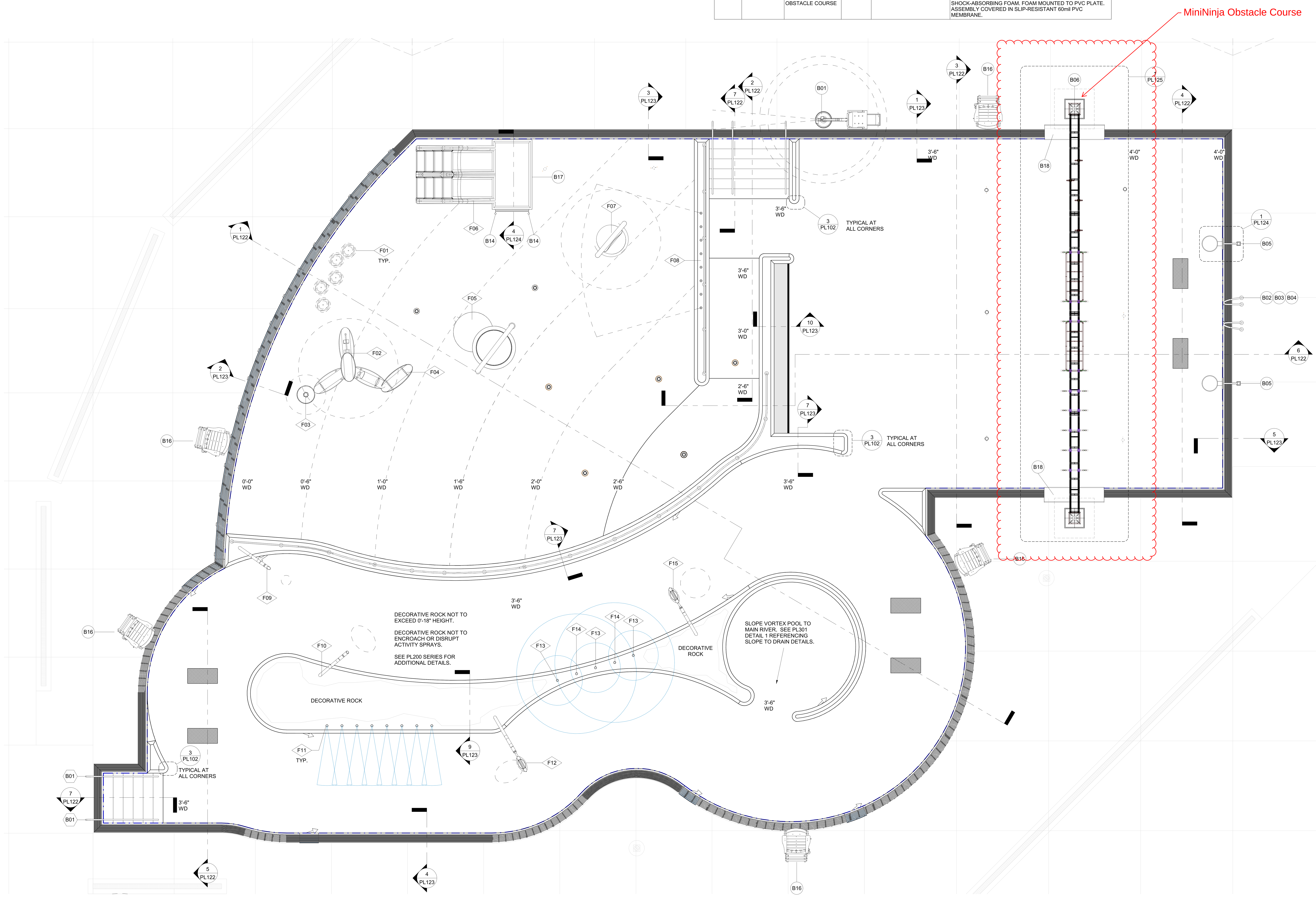
POOL ID	FEATURE ID	FEATURE	QTY	MANUFACTURER	DESCRIPTION	GPM (ea)	GPM (Total)
B	F01	WATER GEYSER	5	CUSTOM	FIELD FABRICATED WATER GEYSER	30	150
B	F02	PLAY FEATURE	1	WATERPLAY	WATERWAYS WATERFALL 3 INTERACTIVE PLAY FEATURE	15	15
B	F03	PLAY FEATURE	1	WATERPLAY	WATERWAYS BASIN INTERACTIVE PLAY FEATURE	5	5
B	F04	AQUATIC PLAY ACTIVITY	1	WATERPLAY	WATERPLAY GULLY PLAY ACTIVITY	7	7
B	F05	SOAKER	1	WATERPLAY	WATERPLAY MEGA SOAKER	40	40
B	F06	SLIDE	1	WATERPLAY	TOT SLIDE	10	10
B	F07	SPRAY FEATURE	1	WATERPLAY	AERIAL SPINSTER	25	25
B	F08	GROUND SPRAY	1	WATERPLAY	SPRAY TUNNEL 8 GROUND SPRAY FEATURE	24	24
B	F09	SPRAY FEATURE	1	WATERPLAY	RIVER SPOUT 1	40	40
B	F10	SPRAY FEATURE	1	WATERPLAY	RIVER NOOK 1	40	40
B	F11	GROUND SPRAY	8	WATERPLAY	TIDAL WAVE GROUND SPRAY	13	104
B	F12	SPRAY FEATURE	1	WATERPLAY	RIVER SPLASH 2	15	15
B	F13	GROUND SPRAY	3	WATERPLAY	CHARLOTTE'S WEB GROUND SPRAY	3	9
B	F14	GROUND SPRAY	2	WATERPLAY	TOWER SPRAY GROUND SPRAY	9	18
B	F15	SPRAY FEATURE	1	WATERPLAY	RIVER SPLASH 1	15	15

SCHEDULE - BASIS OF DESIGN - POOL B

POOL ID	EQUIPMENT ID	EQUIPMENT	QTY	MANUFACTURER	DESCRIPTION
B	01	POOL LIFT	1	SR SMITH, AQUA CREEK, OR EQUAL	STANDARD ANCHORED, ROTATIONAL POOL LIFT, WITH 400 LB MINIMUM LIFTING CAPACITY. MUST MEET ALL APPLICABLE ADA REQUIREMENTS, WHILE MAINTAINING REQUIRED DECK CLEARANCE. PACKAGE TO INCLUDE ARMRESTS, ANCHOR, LIFT COVER, BATTERY CHARGER, AND CADDY
B	02	WEDGE ANCHOR	25	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	CAST BRONZE, 4-1/4" LONG, ACCEPTS 1.500" OD TUBING
B	03	ESCUTCHEON PLATE	25	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	STAINLESS STEEL ROUND ESCUTCHEON FOR 1.50" O.D. RAILS
B	04	GRAB RAILS (PAIRS)	1	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	PRETZEL BEND STYLE, 1.50" OD x .120 WALL THICKNESS, 500 GRIT FINISH MIN.
B	05	BASKETBALL HOOP	2	SR SMITH	STAINLESS STEEL BASKETBALL HOOP WITH ROCKSOLID ANCHOR
B	06	OBSTACLE COURSE	1	NINJACROSS	40' LONG AQUATIC OBSTACLE COURSE ALTERNATE NO. 2
B	14	BARRIER STANCHION	2	PARAGON AQUATICS, SPECTRUM AQUATICS, SR SMITH OR EQUAL	1.900" OD x .145 WALL x 8'-0", PROVIDE SLIDING COLLAR, WITH EYE BOLT
B	16	LIFEGUARD CHAIR	5	TAILWIND, KEIFER, SPECTRUM AQUATICS, SR SMITH OR APPROVED EQUAL	RECYCLED PLASTIC WITH 304 SS HARDWARE, COLOR BY OWNER/ARCHITECT 40" SEAT HEIGHT (OWNER'S SAFETY CONSULTANT TO SPECIFY LOCATION.)
B	17	SAFETY PAD	1	RENOSYS	SAFETY PAD CONTAINING MULTIPLE LAYERS OF DENSE, SHOCK-ABSORBING FOAM. FOAM MOUNTED TO PVC PLATE. ASSEMBLY COVERED IN SLIP-RESISTANT 60mil PVC MEMBRANE.
B	18	SAFETY PAD AT OBSTACLE COURSE	2	RENOSYS	SAFETY PAD CONTAINING MULTIPLE LAYERS OF DENSE, SHOCK-ABSORBING FOAM. FOAM MOUNTED TO PVC PLATE. ASSEMBLY COVERED IN SLIP-RESISTANT 60mil PVC MEMBRANE.

POOL B-LEISURE POOL DATA

DESCRIPTION	QTY	UNITS			
POOL PERIMETER	343	FEET			
WATER SURFACE AREA	5,313	SQUARE FEET			
POOL WATER TEMPERATURE	88	°F			
POOL VOLUME	110,994	GALLONS			
SURGE TANK OPERATING VOLUME	6,637	GALLONS			
TOTAL VOLUME OF WATER	117,631	GALLONS			
CIRCULATION RATE	1,165	GPM			
TURNOVER/VOLUME/FLOW	60 MIN.	22,175	GAL.	370	GPM
TURNOVER/VOLUME/FLOW	120 MIN.	95,457	GAL.	795	GPM
FILTRATION RATE	0.96	GPM/FT²			
FILTER DRAIN RATE	300	GPM			
SURGE FACTOR	1.07	GAL/SQFT			
AVAILABLE SURGE CAPACITY IN SURGE TANK	5689	GALLONS			



1 PL120 POOL B - LEISURE POOL PLAN VIEW
1/4" = 1'-0"

BID SET

CITY OF CHENEY
CHENEY AQUATIC CENTER
711 CEDAR ST, CHENEY, WA 99004

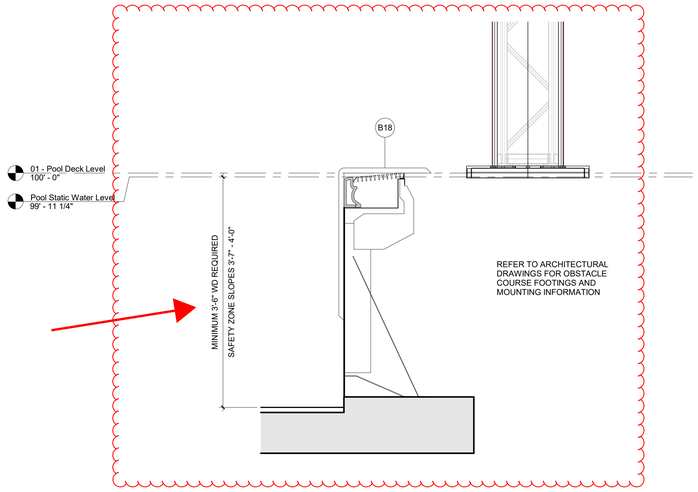
REGISTERED ARCHITECT
MATTHEW W. FREEBY
STATE OF WASHINGTON
7893
4/16/2024

NAC ARCHITECTURE
nacarchitecture.com
1203 WEST RIVERSIDE AVE
SPOKANE WA 99201
P:509.838.8240

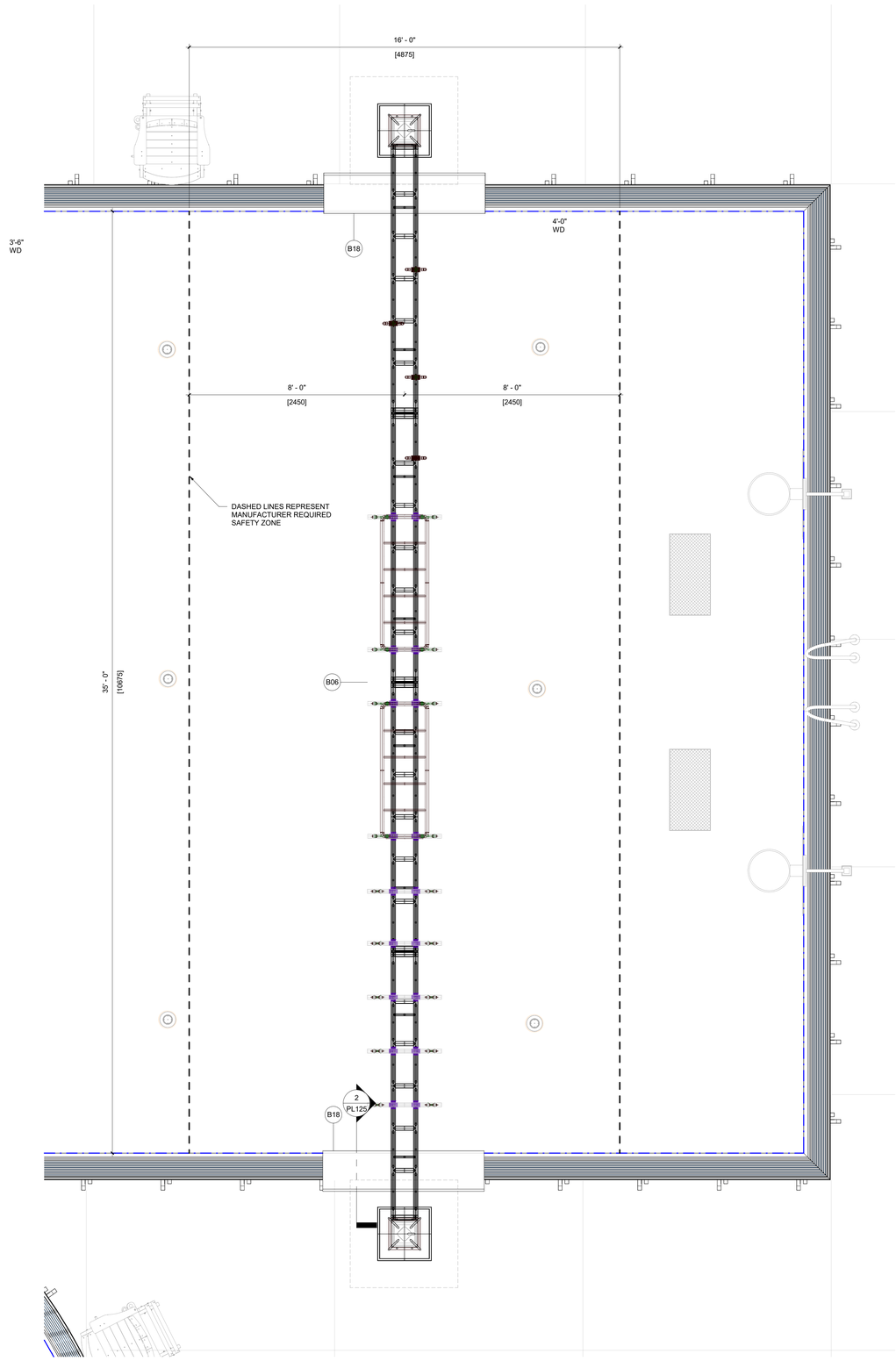
NAC NO	23458
DRAWN	MJC
CHECKED	GGA
DATE	04/16/2024

POOL B - LEISURE POOL PLAN

PL120



2 PL125 WALL AT OBSTACLE COURSE DETAIL VIEW
1" = 1'-0"



1 PL125 POOL B - LEISURE POOL ENLARGED OBSTACLE COURSE PLAN VIEW
1/2" = 1'-0"

REVISIONS

BID SET

WATER TECHNOLOGY INC.
World Leaders in Aquatic Planning, Design and Engineering
1007 3rd Avenue | Beaver Dam, WI 53916
t 920.887.2021 | f 920.887.2024

CITY OF CHENEY
CHENEY AQUATIC CENTER
711 CEDAR ST, CHENEY, WA 99004

7892 REGISTERED ARCHITECT
MATTHEW W. FISERY
STATE OF WASHINGTON
4/16/2024

NAC ARCHITECTURE
nacarchitecture.com
1203 WEST RIVERSIDE AVE
SPOKANE WA 99201
P:509.838.8240

NAC NO	23458
DRAWN	Author
CHECKED	Checker
DATE	04/16/2024

POOL B - LEISURE POOL OBSTACLE COURSE

PL125

List of similar product installations where the receiving pool depths are similar to Cheney's design

Hi Brooke,

MiniNinja projects are typically in 42" of water around 25'-35' in course length, while our retractable course often is over 25yd/25m pools or 50m pools that start at 42" then slope. In any case, we always design obstacle layouts per the intended water depth & user groups. A similar course has been installed in Europe for 15 years, while we have been building for 5 years around North America.

I'm also attaching some images of younger users that you will primarily find on the MiniNinja course.

Pirates Bay Waterpark, Baytown, TX (42")

Provo Rec Center, UT (42" slopes to 13')

New Ulm Rec Center, MN (48" slopes to 8')

Margaret Carpenter Rec Center, Thornton, CO (48" slopes to 12')

Canfor Leisure Pool, Prince George, BC (42" slopes to 8.5')

Idaho Outdoor Fieldhouse - Challenged Athletes Foundation Headquarters - Boise, ID (42" slopes to 8')

Blue Surf Bay Waterpark, Blue Springs, MO (42" slopes to 12.5')

The Landing Waterpark, Bettendorf, IA (42")

Watertown Family Aquatic Center, SD (42")

Northglenn Rec Center, CO (42" slopes to 54")

Wayman Palmer YMCA, Toledo, OH (42")

Fishers Parks & Rec Center, IN (42")

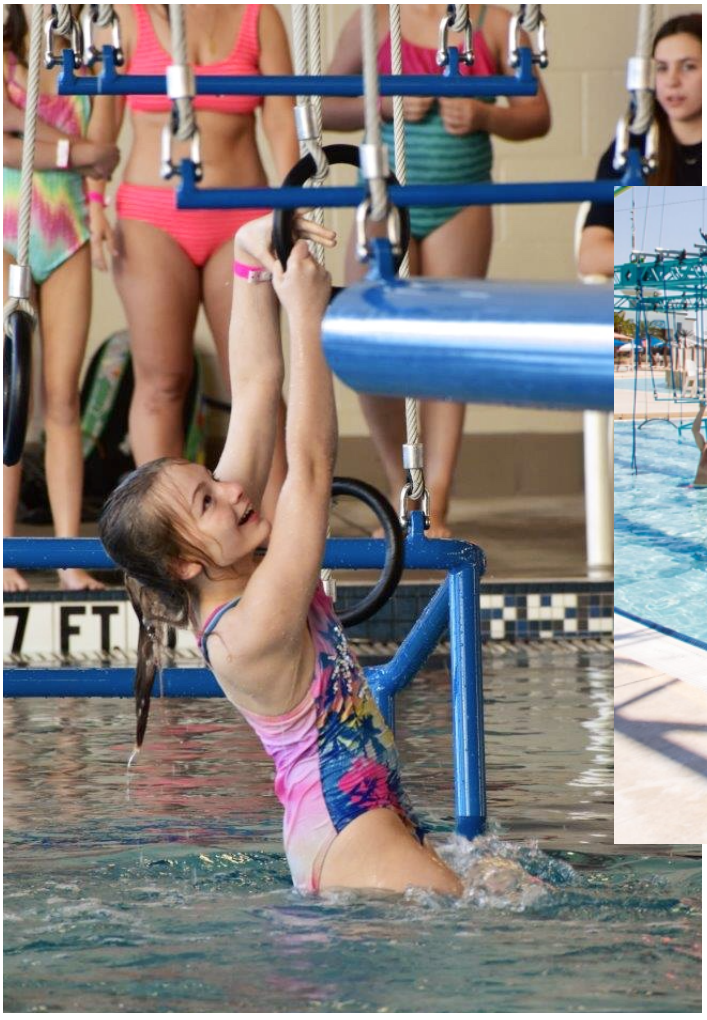
Margaritaville Hotel & Resort, KS (42")

Jasper Municipal Pool, IN (42")

-Kyle

Kyle Rieger, CPO | Managing Partner
NINJACROSS™ SYSTEMS - Transform Your Pool With the Push of a Button. Game On.
Patent No. US 9,889,387 B2

(O) 800.778.9702 | (M) 913.909.9761
Kyle@NinjaCrossSystems.com
www.NinjaCrossSystems.com


7 FT


5FT 0 IN

June 12, 2024

Stephen Wagner
Director of Design & Development
NinjaCross™ Systems
stevew@ninjacrosssystems.com

Re: NinjaCross™ Drop Zone Assessment
Spokane Regional Health District
Project #2024-03-129

Stephen,

As requested, Eclipse Engineering has completed the drop zone assessment while using the NinjaCross™ System for the above noted jurisdiction. We utilized data from the CDC to determine the 10th, 50th and 90th percentile for male and female children aged 10, 12, and 14 years old. Using these participants in addition to the maximum user weight for the system, we analyzed a variety of drop orientations into a pool depth of 3'6" from 20" above the surface of the pool, which is comparable to jumping into the water from the pool deck.

While considering the drop orientations from the available system obstacles, we concluded that a drop into the water while using the NinjaCross™ System per its intended use and safety standards would not present a life safety hazard from impacting the water's surface or contacting the pool floor. When a participant who is using the system per design drops from an obstacle, their acceleration stops when they contact the water's surface, and their velocity is significantly reduced within the first 24", thus allowing the participant to contact the pool floor without a sudden impact. The participant is expected to contact the pool bottom in a manner consistent with any shallow pool activities.

Please note that accidents and injuries can happen in any situation regardless of prevention measures put in place. It is the responsibility of the facility, staff, and local governing agencies to follow the operation and maintenance manuals of the NinjaCross™ system to ensure proper use. Eclipse Engineering does not guarantee the health and safety of any participant of a NinjaCross™ system or the facility itself.

Please contact us with any questions.

Sincerely,
Eclipse Engineering, PC

Wade Ambach, P.E.
Project Manager
wambach@eclipse-engineering.com

Digitally signed by Rolf Henry Armstrong
DN: E=rarmstrong@eeimt.com, CN=Rolf Henry Armstrong, O="Eclipse Engineering, P.C.", L=Bend, S=Oregon, C=US
Date: 2024.06.14 01:41:26-07'00'

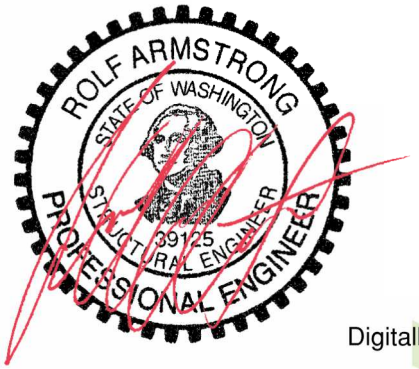
ROLF ARMSTRONG STATE OF WASHINGTON 19125 STRUCTURAL ENGINEER PROFESSIONAL ENGINEER

Rolf Armstrong, P.E., S.E.
CFO, Principal Engineer
rarmstrong@eclipse-engineering.com

Attachment: Safe Drop Zone Graphic

STRUCTURAL CALCULATIONS

NinjaCross – Drop Zone Assessment



Digitally signed by Rolf Henry Armstrong
DN: E=rarmstrong@eeimt.com, CN=Rolf Henry Armstrong, O="Eclipse Engineering, P.C.", L=Bend, S=Oregon, C=US
Date: 2024.06.14 01:40:52-07'00'

Prepared For:

NinjaCross Systems
Kyle W. Rieger, CPO
kyle@ninjacrosssystems.com

TABLE OF CONTENTS

Assumptions

A. DENSITY OF PERSON IS 980 KG/M^3.

B. COEFFICIENT OF DRAG OF PERSON DROPPING THROUGH WATER IS 1.0.

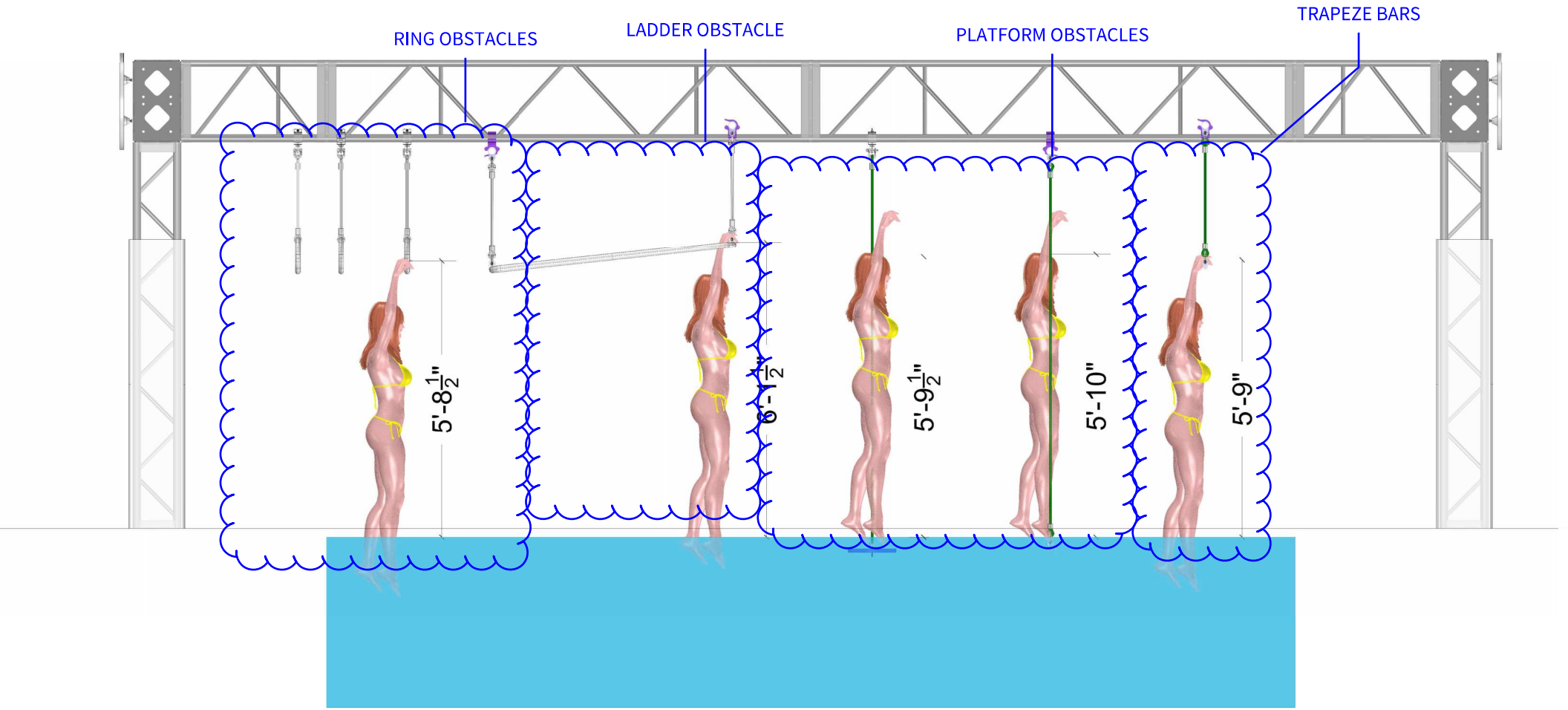
C. PERSON REMAINS STILL THROUGHOUT THE DROP UNTIL MAKING CONTACT WITH THE POOL FLOOR (IF APPLICABLE).

D. THE POOL DEPTH IS 3'-6".

E. PERSON DROPS WITH THEIR FEET 20 INCHES ABOVE THE TOP OF THE WATER.

F. PERSON DROPS FROM REST.

OBSTACLES AND USER CONDITIONS CONSIDERED IN EEPC FALL ZONE REVIEW
MINI NINJA SYSTEM



2 to 20 years: Girls
Stature-for-age and Weight-for-age percentiles

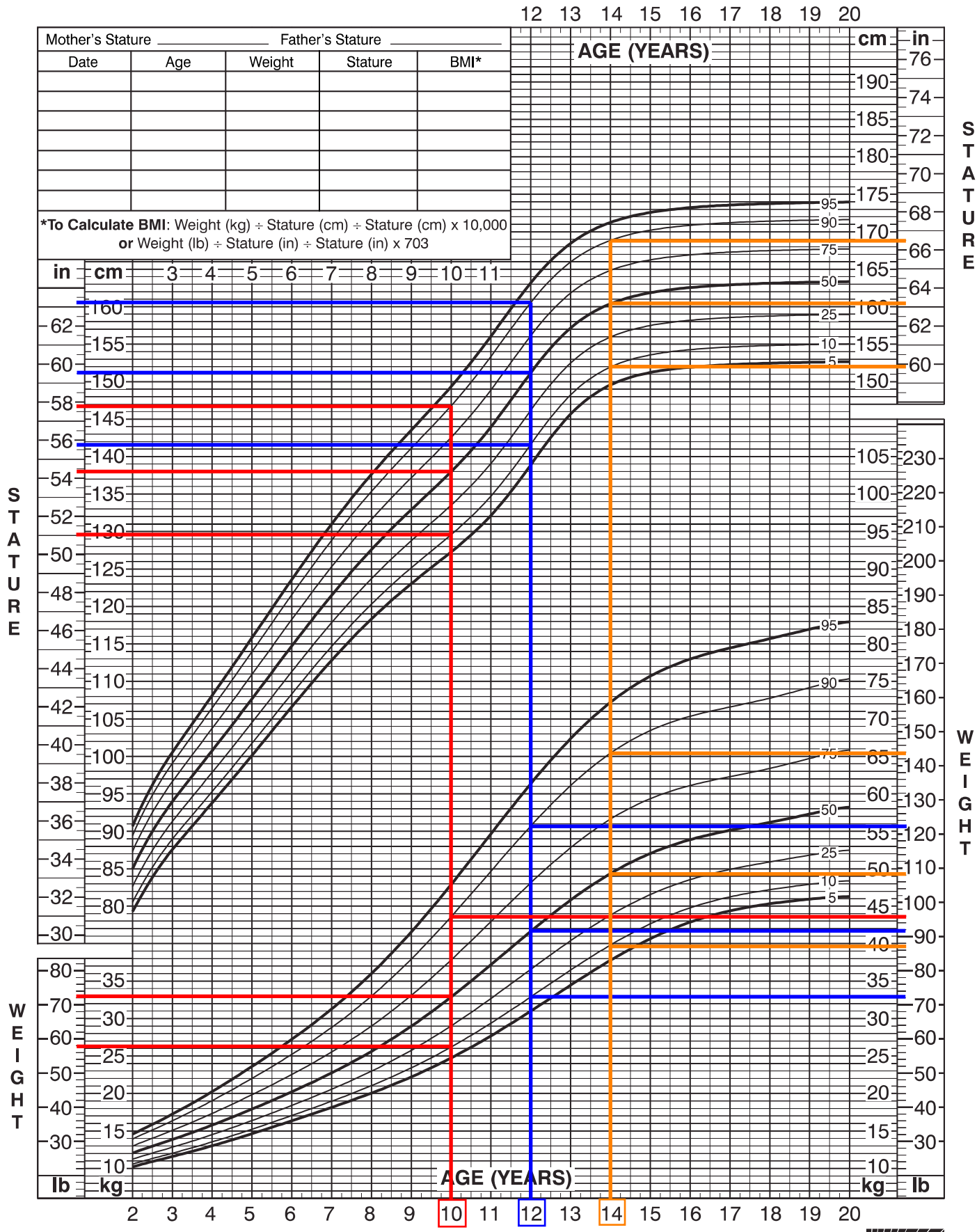
NAME _____

RECORD # _____

Mother's Stature _____ Father's Stature _____

Date	Age	Weight	Stature	BMI*

***To Calculate BMI**: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 11/21/00).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

2 to 20 years: Boys
Stature-for-age and Weight-for-age percentiles

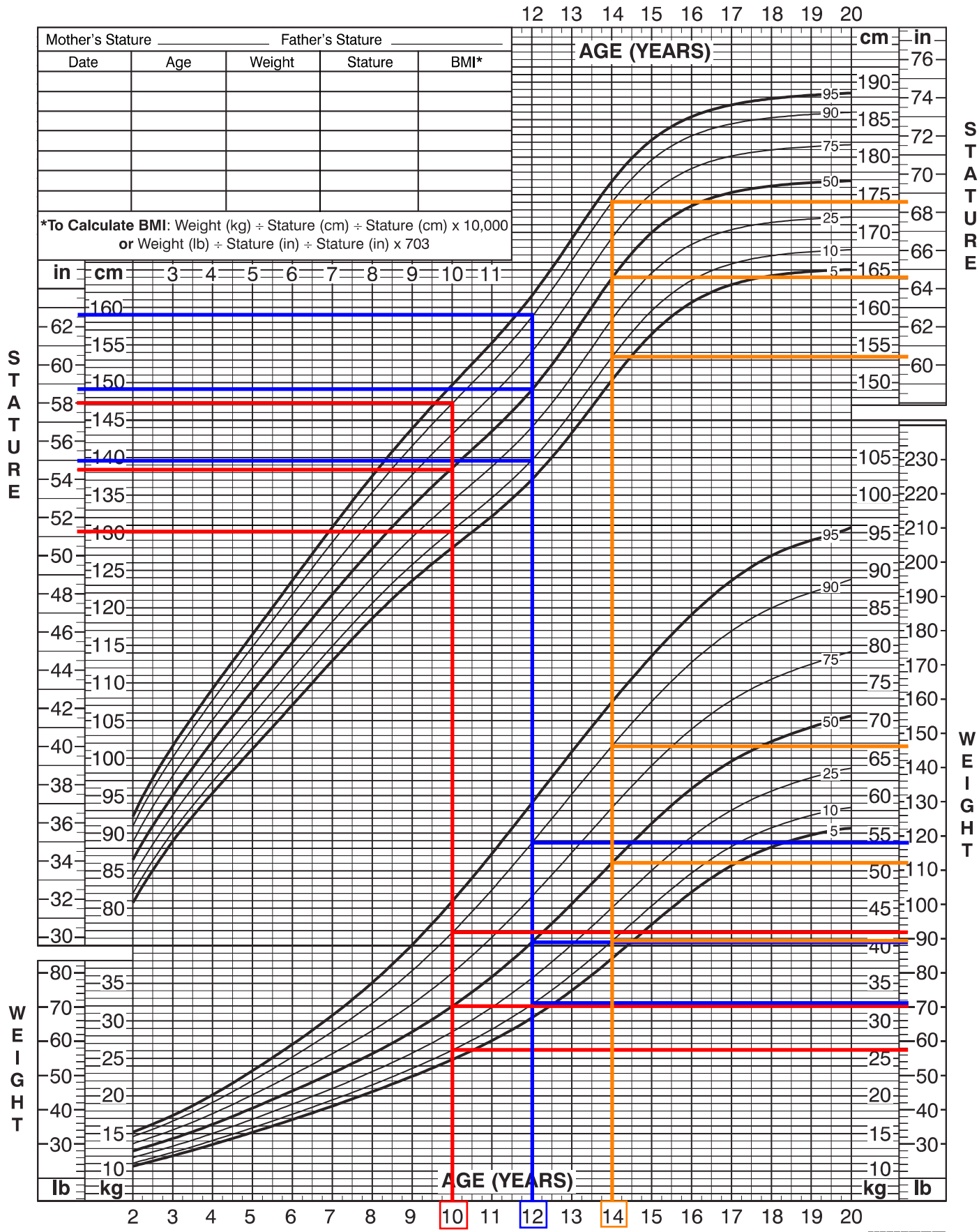
NAME _____

RECORD # _____

Mother's Stature _____ Father's Stature _____

Date	Age	Weight	Stature	BMI*

***To Calculate BMI**: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 11/21/00).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

Summary Data

Girls Stature & Weight for Age per CDC			
Age	Percentile	Weight (lb)	Height (in)
10	10	58	51
	50	72	54.5
	90	96	57.75
12	10	72	55.75
	50	92	59.5
	90	122	63.25
14	10	87	59.75
	50	108	63.25
	90	144	66.5

Boys Stature & Weight for Age per CDC			
Age	Percentile	Weight (lb)	Height (in)
10	10	58	51.25
	50	70	54.5
	90	92	58
12	10	71	55
	50	89	58.75
	90	118	62.75
14	10	89	60.5
	50	112	64.5
	90	146	68.5

NinjaCross System Design Participant	
Weight (lb)	Height (in)
275.0	72.0

NinjaCross System Design Participant Results				
	Vertical Drop	Diagonal Drop	Tucked Knee Drop	Horizontal Drop
Velocity at Pool Bottom	2.9 mph	2.9 mph	1.8 mph	0.0 mph
Effective Height of Drop	3.4 in	3.4 in	1.3 in	0.0 in

THE MAXIMUM VELOCITY AT WHICH THE PERSON HITS THE POOL FLOOR IS THAT WITH WHICH A PERSON HITS THE GROUND FROM A 3.4 INCH HEIGHT FALL.

$$mgh = \frac{1}{2}mv^2$$

Effective Height Above Ground $h = \frac{v^2}{2g}$

Please note that OSHA does not consider drops less than 4'-0" to require fall protection

Excerpt from https://www.osha.gov/fall-protection:
"OSHA requires that fall protection be provided at elevations of four feet in general industry workplaces."

Female Participant Results

Age	Percentile		Vertical Drop	Diagonal Drop	Tucked Knee Drop	Horizontal Drop
10	10	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	1.3 mph	0.9 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.7 in	0.3 in	0.0 in	0.0 in
12	10	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	1.3 mph	0.7 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.7 in	0.2 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	2.5 mph	2.2 mph	0.0 mph	0.0 mph
		Effective Height of Drop	2.4 in	2.0 in	0.0 in	0.0 in
14	10	Velocity at Pool Bottom	0.9 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.3 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	2.0 mph	1.8 mph	0.0 mph	0.0 mph
		Effective Height of Drop	1.6 in	1.3 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	2.9 mph	2.9 mph	0.0 mph	0.0 mph
		Effective Height of Drop	3.4 in	3.4 in	0.0 in	0.0 in

Male Participant Results

Age	Percentile		Vertical Drop	Diagonal Drop	Tucked Knee Drop	Horizontal Drop
10	10	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	1.1 mph	0.4 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.5 in	0.1 in	0.0 in	0.0 in
12	10	Velocity at Pool Bottom	0.0 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.0 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	1.1 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.5 in	0.0 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	2.2 mph	2.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	2.0 in	1.6 in	0.0 in	0.0 in
14	10	Velocity at Pool Bottom	1.1 mph	0.0 mph	0.0 mph	0.0 mph
		Effective Height of Drop	0.5 in	0.0 in	0.0 in	0.0 in
	50	Velocity at Pool Bottom	2.2 mph	1.8 mph	0.0 mph	0.0 mph
		Effective Height of Drop	2.0 in	1.3 in	0.0 in	0.0 in
	90	Velocity at Pool Bottom	3.1 mph	2.9 mph	0.0 mph	0.0 mph
		Effective Height of Drop	3.9 in	3.4 in	0.0 in	0.0 in

NinjaCross System
Design Participant Calculations

Drops Vertically into the Pool

Height of COM	h = 1.42	m	
Mass of Person	m = 124.72	kg =	275 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.19	m^2 =	2 ft^2
Length of Person	L = 1.83	m =	6 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	624.2	10.0	-634.2	0.29	0.9
0.200	4.7	432.1	18.2	-450.3	0.52	1.7
0.300	4.1	314.2	25.1	-339.3	0.72	2.4
0.400	3.5	236.3	31.0	-267.3	0.89	2.9
0.500	3.1	182.0	36.2	-218.1	1.04	3.4
0.600	2.7	142.4	40.8	-183.1	1.17	3.8
0.700	2.4	112.6	44.8	-157.4	1.28	4.2
0.800	2.2	89.6	48.4	-138.0	1.39	4.5
0.900	1.9	71.5	51.6	-123.1	1.48	4.9
1.000	1.7	56.9	54.5	-111.4	1.56	5.1
1.100	1.5	45.1	57.1	-102.2	1.63	5.4
1.200	1.4	35.4	59.4	-94.8	1.70	5.6
1.300	1.2	27.5	61.4	-88.9	1.76	5.8
1.400	1.0	20.9	63.2	-84.1	1.81	5.9
1.500	0.9	15.5	64.7	-80.2	1.85	6.1
1.600	0.8	11.1	66.0	-77.1	1.89	6.2
1.700	0.6	7.5	67.1	-74.6	1.92	6.3
1.800	0.5	4.7	68.0	-72.7	1.95	6.4
1.900	0.4	2.6	68.6	-71.2	1.96	6.4
1.980	0.3	1.4	69.0	-70.4	1.98	6.5
2.000	0.2	1.1	69.1	-70.3	1.98	6.5
2.100	0.1	0.3	69.4	-69.7	1.99	6.5

Drops Diagonally into the Pool

Height of COM	h = 1.15	m	
Mass of Person	m = 124.72	kg =	275 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.19	m^2 =	2 ft^2
Length of Person	L = 1.29	m =	4.24264069 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	623.6	14.1	-637.7	0.29	0.9
0.200	4.7	430.1	25.7	-455.8	0.52	1.7
0.300	4.0	310.8	35.4	-346.2	0.72	2.3
0.400	3.5	231.6	43.7	-275.3	0.88	2.9
0.500	3.0	176.1	51.0	-227.1	1.03	3.4
0.600	2.7	135.5	57.3	-192.8	1.16	3.8
0.700	2.3	104.9	62.9	-167.8	1.27	4.2
0.800	2.1	81.3	67.7	-149.0	1.37	4.5
0.900	1.8	62.7	72.1	-134.7	1.46	4.8
1.000	1.6	47.8	75.8	-123.6	1.53	5.0
1.100	1.4	35.9	79.1	-115.0	1.60	5.3
1.200	1.2	26.3	81.9	-108.2	1.66	5.4
1.300	1.0	18.6	84.3	-102.9	1.71	5.6
1.400	0.8	12.5	86.3	-98.8	1.75	5.7
1.500	0.6	7.7	87.9	-95.6	1.78	5.8
1.600	0.5	4.2	89.1	-93.3	1.80	5.9
1.700	0.3	1.8	90.0	-91.8	1.82	6.0
1.800	0.1	0.4	90.5	-90.9	1.83	6.0
1.900						
1.980						
2.000						
2.100						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.97	m	
Mass of Person	m = 124.72	kg =	275 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.28	m^2 =	3 ft^2
Length of Person	L = 0.91	m =	3 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.2	783.8	19.1	-802.9	0.27	0.9
0.200	4.0	475.9	33.5	-509.4	0.48	1.6
0.300	3.3	313.5	45.0	-358.5	0.64	2.1
0.400	2.7	216.6	54.5	-271.1	0.78	2.6
0.500	2.3	153.9	62.4	-216.3	0.89	2.9
0.600	2.0	110.7	69.1	-179.9	0.99	3.2
0.700	1.7	79.8	74.8	-154.7	1.07	3.5
0.800	1.4	57.0	79.7	-136.7	1.14	3.7
0.900	1.2	39.9	83.7	-123.6	1.20	3.9
1.000	1.0	26.9	87.1	-114.0	1.25	4.1
1.100	0.8	17.2	89.8	-107.0	1.29	4.2
1.200	0.6	9.9	91.9	-101.9	1.32	4.3
1.300	0.4	4.9	93.5	-98.4	1.34	4.4
1.400	0.2	1.7	94.5	-96.1	1.35	4.4
1.500	0.1	0.1	95.0	-95.1	1.36	4.5
1.600						
1.700						
1.800						
1.900						
1.980						
2.000						
2.100						

Drops Horizontally into the Pool

Height of COM	h = 0.81	m	
Mass of Person	m = 124.72	kg =	275 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.56	m^2 =	6 ft^2
Length of Person	L = 0.61	m =	2 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.0	985.0	25.4	-1010.4	0.24	0.8
0.200	2.8	463.1	41.3	-504.4	0.39	1.3
0.300	2.1	259.2	52.8	-312.0	0.50	1.7
0.400	1.6	157.4	61.6	-219.1	0.59	1.9
0.500	1.3	99.1	68.6	-167.7	0.65	2.1
0.600	1.0	62.6	74.1	-136.7	0.71	2.3
0.700	0.8	38.6	78.5	-117.0	0.75	2.5
0.800	0.6	22.4	81.8	-104.2	0.78	2.6
0.900	0.4	11.5	84.3	-95.8	0.80	2.6
1.000	0.3	4.6	86.0	-90.6	0.82	2.7
1.100	0.1	0.9	87.0	-87.9	0.83	2.7
1.200						
1.300						
1.400						
1.500						
1.600						
1.700						
1.800						
1.900						
1.980						
2.000						
2.100						

10-year-old Girl Calculations

Drops Vertically into the Pool

Height of COM	h = 1.16	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.30	m =	4.25 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.4	195.3	12.5	-207.8	0.25	0.8
0.200	3.1	96.7	20.8	-117.5	0.42	1.4
0.300	2.3	53.1	26.9	-79.9	0.54	1.8
0.370	1.9	35.5	30.2	-65.6	0.61	2.0
0.400	1.7	29.8	31.4	-61.2	0.64	2.1
0.500	1.3	16.1	34.7	-50.8	0.70	2.3
0.570	1.0	9.8	36.5	-46.3	0.74	2.4
0.600	0.9	7.7	37.1	-44.8	0.75	2.5
0.700	0.5	2.7	38.7	-41.4	0.78	2.6
0.730	0.4	1.8	39.0	-40.8	0.79	2.6
0.800	0.2	0.4	39.5	-39.8	0.80	2.6
0.850		0.0	39.6	-39.6	0.80	2.6

Drops Diagonally into the Pool

Height of COM	h = 0.97	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 0.92	m =	3.00520382 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.4	193.8	17.7	-211.5	0.25	0.8
0.200	3.1	93.4	29.3	-122.7	0.42	1.4
0.300	2.2	48.6	37.6	-86.2	0.54	1.8
0.370	1.8	30.5	42.0	-72.5	0.60	2.0
0.400	1.6	24.7	43.6	-68.3	0.62	2.0
0.500	1.0	11.1	47.7	-58.8	0.68	2.2
0.570	0.7	5.2	49.7	-54.9	0.71	2.3
0.600	0.6	3.5	50.3	-53.8	0.72	2.4
0.700	0.1	0.3	51.4	-51.7	0.74	2.4
0.730		0.0	51.5	-51.5	0.74	2.4
0.800						
0.850						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.83	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.20	m^2 =	2.125 ft^2
Length of Person	L = 0.65	m =	2.125 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = Vg(\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.0	202.3	20.7	-223.0	0.21	0.7
0.200	1.8	70.4	31.2	-101.6	0.32	1.0
0.300	1.1	27.8	37.8	-65.6	0.38	1.3
0.370	0.8	13.6	40.8	-54.3	0.41	1.4
0.400	0.6	9.5	41.7	-51.2	0.42	1.4
0.500	0.2	1.6	43.8	-45.4	0.44	1.5
0.570		0.0	44.2	-44.2	0.45	1.5
0.600						
0.700						
0.730						
0.800						
0.850						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.39	m^2 =	4.25 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	1.8	149.5	33.5	-182.9	0.16	0.5
0.200	0.8	32.9	45.7	-78.6	0.22	0.7
0.300	0.3	4.9	51.2	-56.1	0.24	0.8
0.370		0.0	52.3	-52.3	0.25	0.8
0.400						
0.500						
0.570						
0.600						
0.700						
0.730						
0.800						
0.850						

Drops Vertically into the Pool

Height of COM	h = 1.20	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.38	m =	4.54166667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.8	226.4	12.2	-238.6	0.26	0.9
0.200	3.5	123.2	20.8	-143.9	0.45	1.5
0.300	2.7	73.1	27.3	-100.4	0.59	1.9
0.400	2.1	44.9	32.4	-77.2	0.70	2.3
0.500	1.7	27.3	36.3	-63.6	0.79	2.6
0.600	1.3	15.9	39.4	-55.3	0.85	2.8
0.660	1.1	11.0	40.8	-51.8	0.89	2.9
0.700	0.9	8.3	41.7	-50.0	0.90	3.0
0.800	0.6	3.5	43.2	-46.8	0.94	3.1
0.850	0.4	1.9	43.8	-45.7	0.95	3.1
0.900	0.3	0.8	44.2	-45.0	0.96	3.1
0.990		0.0	44.4	-44.4	0.96	3.2

Drops Diagonally into the Pool

Height of COM	h = 1.00	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 0.98	m =	3.2114433 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = Vg(p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.8	225.2	17.2	-242.4	0.26	0.9
0.200	3.5	120.1	29.3	-149.4	0.45	1.5
0.300	2.6	68.7	38.3	-107.0	0.59	1.9
0.400	2.0	39.5	45.1	-84.7	0.69	2.3
0.500	1.5	21.6	50.3	-71.9	0.77	2.5
0.600	1.0	10.4	54.0	-64.4	0.83	2.7
0.660	0.8	5.9	55.6	-61.5	0.85	2.8
0.700	0.6	3.7	56.4	-60.1	0.86	2.8
0.800	0.2	0.5	57.6	-58.0	0.88	2.9
0.850		0.0	57.7	-57.7	0.88	2.9
0.900						
0.990						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.85	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.21	m^2 =	2.270833 ft^2
Length of Person	L = 0.69	m =	2.27083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.3	257.6	20.3	-277.8	0.22	0.7
0.200	2.1	98.2	31.3	-129.5	0.34	1.1
0.300	1.4	43.8	38.5	-82.3	0.42	1.4
0.400	0.9	19.0	43.3	-62.2	0.47	1.5
0.500	0.5	6.5	46.3	-52.8	0.50	1.6
0.600	0.2	0.9	47.8	-48.7	0.52	1.7
0.660		0.0	48.0	-48.0	0.52	1.7
0.700						
0.800						
0.850						
0.900						
0.990						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.42	m^2 =	4.541667 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.0	204.9	35.8	-240.7	0.17	0.6
0.200	1.0	52.0	50.1	-102.1	0.24	0.8
0.300	0.5	12.0	57.4	-69.4	0.27	0.9
0.400	0.1	0.4	60.0	-60.4	0.29	0.9
0.500						
0.600						
0.660						
0.700						
0.800						
0.850						
0.900						
0.990						

Drops Vertically into the Pool

Height of COM	h = 1.24	m	
Mass of Person	m = 43.54	kg =	96 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.47	m =	4.8125 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.2	266.3	12.0	-278.2	0.27	0.9
0.200	4.1	161.8	21.0	-182.9	0.48	1.6
0.300	3.3	105.0	28.3	-133.2	0.65	2.1
0.400	2.7	70.3	34.1	-104.4	0.78	2.6
0.490	2.3	49.3	38.5	-87.8	0.88	2.9
0.500	2.2	47.4	38.9	-86.3	0.89	2.9
0.600	1.8	31.6	42.9	-74.5	0.98	3.2
0.700	1.4	20.4	46.1	-66.5	1.06	3.5
0.790	1.2	13.1	48.4	-61.5	1.11	3.6
0.800	1.1	12.4	48.6	-61.0	1.12	3.7
0.900	0.8	6.7	50.5	-57.3	1.16	3.8
1.000	0.5	2.9	51.9	-54.8	1.19	3.9
1.100	0.3	0.7	52.7	-53.4	1.21	4.0
1.200		0.0	52.9	-52.9	1.22	4.0

Drops Diagonally into the Pool

Height of COM	h = 1.03	m	
Mass of Person	m = 43.54	kg =	96 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.04	m =	3.40295138 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.2	265.2	16.9	-282.1	0.27	0.9
0.200	4.0	159.1	29.7	-188.8	0.48	1.6
0.300	3.2	100.7	39.7	-140.5	0.65	2.1
0.400	2.6	64.8	47.8	-112.6	0.78	2.5
0.490	2.1	43.2	53.7	-96.8	0.87	2.9
0.500	2.1	41.2	54.2	-95.4	0.88	2.9
0.600	1.6	25.1	59.3	-84.4	0.96	3.2
0.700	1.2	14.0	63.2	-77.2	1.03	3.4
0.790	0.8	7.2	65.8	-73.0	1.07	3.5
0.800	0.8	6.6	66.0	-72.6	1.07	3.5
0.900	0.4	2.1	67.8	-69.9	1.10	3.6
1.000	0.1	0.1	68.5	-68.7	1.11	3.7
1.100						
1.200						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.87	m	
Mass of Person	m = 43.54	kg =	96 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.22	m^2 =	2.40625 ft^2
Length of Person	L = 0.73	m =	2.40625 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.7	345.5	20.4	-365.9	0.23	0.8
0.200	2.5	148.0	32.5	-180.5	0.37	1.2
0.300	1.8	74.2	40.8	-115.0	0.47	1.5
0.400	1.3	38.3	46.7	-85.0	0.54	1.8
0.490	0.9	20.1	50.6	-70.7	0.58	1.9
0.500	0.9	18.6	50.9	-69.6	0.58	1.9
0.600	0.5	7.4	53.8	-61.2	0.62	2.0
0.700	0.2	1.7	55.4	-57.0	0.64	2.1
0.790		0.0	55.8	-55.8	0.64	2.1
0.800						
0.900						
1.000						
1.100						
1.200						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 43.54	kg =	96 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.45	m^2 =	4.8125 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.4	306.2	39.4	-345.6	0.19	0.6
0.200	1.4	91.0	57.0	-148.0	0.27	0.9
0.300	0.8	29.3	67.0	-96.3	0.32	1.0
0.400	0.3	6.0	72.2	-78.2	0.34	1.1
0.490		0.0	73.6	-73.6	0.35	1.2
0.500						
0.600						
0.700						
0.790						
0.800						
0.900						
1.000						
1.100						
1.200						

12-year-old Girl Calculations

Drops Vertically into the Pool

Height of COM	h = 1.22	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.42	m =	4.64583333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.8	226.5	11.9	-238.4	0.26	0.9
0.200	3.5	123.3	20.3	-143.6	0.45	1.5
0.300	2.7	73.4	26.7	-100.1	0.59	1.9
0.400	2.2	45.2	31.7	-76.8	0.70	2.3
0.500	1.7	27.6	35.6	-63.2	0.79	2.6
0.600	1.3	16.2	38.6	-54.8	0.85	2.8
0.660	1.1	11.3	40.0	-51.3	0.89	2.9
0.700	0.9	8.6	40.8	-49.4	0.91	3.0
0.800	0.6	3.7	42.4	-46.2	0.94	3.1
0.860	0.4	1.9	43.0	-44.9	0.95	3.1
0.900	0.3	1.0	43.3	-44.3	0.96	3.2
1.000		0.0	43.7	-43.7	0.97	3.2

Drops Diagonally into the Pool

Height of COM	h = 1.01	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.00	m =	3.28510025 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.8	225.3	16.8	-242.1	0.26	0.9
0.200	3.5	120.3	28.6	-149.0	0.45	1.5
0.300	2.7	69.1	37.4	-106.5	0.59	1.9
0.400	2.0	39.9	44.2	-84.1	0.69	2.3
0.500	1.5	22.1	49.2	-71.3	0.77	2.5
0.600	1.0	10.8	52.9	-63.7	0.83	2.7
0.660	0.8	6.3	54.5	-60.7	0.85	2.8
0.700	0.6	4.0	55.3	-59.3	0.87	2.8
0.800	0.2	0.6	56.5	-57.2	0.89	2.9
0.860		0.0	56.7	-56.8	0.89	2.9
0.900						
1.000						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.86	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.22	m^2 =	2.322917 ft^2
Length of Person	L = 0.71	m =	2.32291667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.3	257.3	19.7	-277.1	0.22	0.7
0.200	2.0	97.6	30.3	-127.9	0.34	1.1
0.300	1.4	43.5	37.2	-80.8	0.41	1.4
0.400	0.9	19.0	41.9	-60.8	0.46	1.5
0.500	0.5	6.6	44.8	-51.4	0.50	1.6
0.600	0.2	1.0	46.3	-47.3	0.51	1.7
0.660		0.0	46.5	-46.5	0.52	1.7
0.700						
0.800						
0.860						
0.900						
1.000						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 32.65	kg =	72 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.43	m^2 =	4.645833 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.0	202.6	35.5	-238.1	0.17	0.6
0.200	1.0	51.0	49.5	-100.5	0.24	0.8
0.300	0.5	11.7	56.6	-68.3	0.27	0.9
0.400	0.0	0.3	59.2	-59.5	0.28	0.9
0.500						
0.600						
0.660						
0.700						
0.800						
0.860						
0.900						
1.000						

Drops Vertically into the Pool

Height of COM	h = 1.26	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.51	m =	4.95833333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.2	260.6	11.5	-272.2	0.27	0.9
0.200	4.0	156.2	20.2	-176.4	0.48	1.6
0.300	3.2	100.3	27.1	-127.4	0.64	2.1
0.400	2.6	66.6	32.7	-99.2	0.77	2.5
0.480	2.2	48.3	36.4	-84.7	0.86	2.8
0.500	2.1	44.6	37.2	-81.8	0.88	2.9
0.600	1.7	29.5	40.9	-70.4	0.97	3.2
0.700	1.4	18.9	43.9	-62.7	1.04	3.4
0.780	1.1	12.6	45.8	-58.4	1.08	3.6
0.800	1.1	11.3	46.2	-57.5	1.09	3.6
0.900	0.8	6.0	48.0	-54.0	1.14	3.7
1.000	0.5	2.5	49.2	-51.7	1.16	3.8
1.100	0.2	0.6	49.9	-50.5	1.18	3.9
1.190		0.0	50.1	-50.1	1.19	3.9

Drops Diagonally into the Pool

Height of COM	h = 1.04	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.07	m =	3.50607112 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	259.6	16.3	-275.9	0.27	0.9
0.200	4.0	153.5	28.5	-182.0	0.48	1.6
0.300	3.1	96.1	38.1	-134.3	0.64	2.1
0.400	2.5	61.3	45.7	-107.1	0.76	2.5
0.480	2.1	42.5	50.7	-93.2	0.85	2.8
0.500	2.0	38.6	51.8	-90.4	0.87	2.8
0.600	1.5	23.3	56.6	-79.8	0.95	3.1
0.700	1.1	12.8	60.2	-72.9	1.01	3.3
0.780	0.8	7.0	62.3	-69.3	1.04	3.4
0.800	0.8	5.8	62.7	-68.6	1.05	3.4
0.900	0.4	1.7	64.3	-66.1	1.08	3.5
1.000	0.0	0.1	65.0	-65.0	1.09	3.6
1.100						
1.190						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.89	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.23	m^2 =	2.479167 ft^2
Length of Person	L = 0.76	m =	2.47916667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.6	332.4	19.4	-351.8	0.23	0.8
0.200	2.4	138.5	30.7	-169.2	0.36	1.2
0.300	1.7	68.2	38.3	-106.5	0.45	1.5
0.400	1.2	34.7	43.7	-78.4	0.52	1.7
0.480	0.9	19.3	46.9	-66.2	0.55	1.8
0.500	0.8	16.5	47.6	-64.0	0.56	1.8
0.600	0.5	6.3	50.1	-56.4	0.59	1.9
0.700	0.2	1.2	51.4	-52.7	0.61	2.0
0.780		0.0	51.8	-51.8	0.61	2.0
0.800						
0.900						
1.000						
1.100						
1.190						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.46	m^2 =	4.958333 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.3	285.1	38.3	-323.4	0.18	0.6
0.200	1.3	81.9	54.9	-136.8	0.26	0.9
0.300	0.7	25.1	64.1	-89.2	0.31	1.0
0.400	0.3	4.3	68.7	-73.1	0.33	1.1
0.480		0.0	69.7	-69.7	0.33	1.1
0.500						
0.600						
0.700						
0.780						
0.800						
0.900						
1.000						
1.100						
1.190						

Drops Vertically into the Pool

Height of COM	h = 1.31	m	
Mass of Person	m = 55.33	kg =	122 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.61	m =	5.27083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.5	297.0	11.2	-308.2	0.28	0.9
0.200	4.5	196.1	20.2	-216.3	0.51	1.7
0.300	3.8	135.9	27.6	-163.5	0.69	2.3
0.400	3.2	96.8	33.8	-130.6	0.85	2.8
0.500	2.7	69.9	39.0	-108.9	0.98	3.2
0.550	2.5	59.5	41.4	-100.8	1.04	3.4
0.600	2.3	50.5	43.5	-94.0	1.09	3.6
0.700	1.9	36.1	47.3	-83.4	1.19	3.9
0.800	1.6	25.3	50.5	-75.8	1.27	4.2
0.900	1.3	17.0	53.1	-70.2	1.34	4.4
0.930	1.2	15.0	53.8	-68.8	1.35	4.4
1.000	1.1	10.8	55.3	-66.1	1.39	4.6
1.100	0.8	6.2	56.9	-63.1	1.43	4.7
1.200	0.5	3.0	58.1	-61.1	1.46	4.8
1.230	0.5	2.3	58.4	-60.6	1.47	4.8
1.300	0.3	1.0	58.9	-59.9	1.48	4.9
1.400	0.1	0.1	59.2	-59.3	1.49	4.9
1.430		0.0	59.2	-59.2	1.49	4.9

Drops Diagonally into the Pool

Height of COM	h = 1.08	m	
Mass of Person	m = 55.33	kg =	122 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.14	m =	3.72704199 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.5	296.1	15.9	-312.0	0.28	0.9
0.200	4.5	193.7	28.5	-222.2	0.51	1.7
0.300	3.7	132.1	38.8	-170.9	0.69	2.3
0.400	3.1	91.7	47.4	-139.2	0.84	2.8
0.500	2.6	63.8	54.6	-118.4	0.97	3.2
0.550	2.3	53.0	57.7	-110.7	1.03	3.4
0.600	2.1	43.7	60.5	-104.3	1.08	3.5
0.700	1.7	29.0	65.4	-94.4	1.16	3.8
0.800	1.4	18.2	69.4	-87.5	1.23	4.0
0.900	1.0	10.3	72.4	-82.7	1.29	4.2
0.930	0.9	8.5	73.2	-81.6	1.30	4.3
1.000	0.7	4.9	74.6	-79.5	1.33	4.4
1.100	0.4	1.6	76.0	-77.5	1.35	4.4
1.200	0.1	0.1	76.6	-76.7	1.36	4.5
1.230		0.0	76.6	-76.6	1.36	4.5
1.300						
1.400						
1.430						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.91	m	
Mass of Person	m = 55.33	kg =	122 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.24	m^2 =	2.635417 ft^2
Length of Person	L = 0.80	m =	2.63541667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.0	433.7	19.3	-453.0	0.24	0.8
0.200	2.8	200.5	31.3	-231.8	0.39	1.3
0.300	2.0	108.0	39.9	-147.9	0.50	1.6
0.400	1.5	61.4	46.3	-107.8	0.58	1.9
0.500	1.2	34.8	51.2	-86.0	0.64	2.1
0.550	1.0	25.7	53.1	-78.8	0.67	2.2
0.600	0.8	18.5	54.8	-73.3	0.69	2.3
0.700	0.6	8.5	57.3	-65.8	0.72	2.4
0.800	0.3	2.7	58.9	-61.6	0.74	2.4
0.900	0.1	0.2	59.6	-59.8	0.75	2.5
0.930		0.0	59.6	-59.6	0.75	2.5
1.000						
1.100						
1.200						
1.230						
1.300						
1.400						
1.430						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 55.33	kg =	122 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.49	m^2 =	5.270833 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.7	411.5	41.6	-453.1	0.20	0.7
0.200	1.6	134.8	61.6	-196.4	0.29	1.0
0.300	1.0	51.0	73.6	-124.6	0.35	1.2
0.400	0.5	16.2	80.8	-97.0	0.39	1.3
0.500	0.2	2.1	84.3	-86.4	0.40	1.3
0.550		0.1	84.8	-84.8	0.40	1.3
0.600						
0.700						
0.800						
0.900						
0.930						
1.000						
1.100						
1.200						
1.230						
1.300						
1.400						
1.430						

14-year-old Girl Calculations

Drops Vertically into the Pool

Height of COM	h = 1.27	m	
Mass of Person	m = 39.46	kg =	87 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.52	m =	4.97916667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	253.1	11.4	-264.5	0.27	0.9
0.200	3.9	148.6	19.9	-168.5	0.47	1.6
0.300	3.1	94.0	26.5	-120.5	0.63	2.1
0.400	2.5	61.5	31.9	-93.4	0.76	2.5
0.460	2.2	48.0	34.6	-82.5	0.82	2.7
0.500	2.0	40.6	36.2	-76.8	0.86	2.8
0.600	1.6	26.4	39.7	-66.1	0.94	3.1
0.700	1.3	16.4	42.5	-58.9	1.01	3.3
0.750	1.1	12.6	43.7	-56.3	1.04	3.4
0.800	1.0	9.5	44.7	-54.1	1.06	3.5
0.900	0.7	4.7	46.2	-50.9	1.10	3.6
0.990	0.4	1.9	47.2	-49.1	1.12	3.7
1.000	0.4	1.7	47.3	-49.0	1.12	3.7
1.100	0.1	0.2	47.8	-48.0	1.14	3.7
1.150		0.0	47.9	-47.9	1.14	3.7

Drops Diagonally into the Pool

Height of COM	h = 1.04	m	
Mass of Person	m = 39.46	kg =	87 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.07	m =	3.52080251 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	252.0	16.1	-268.2	0.27	0.9
0.200	3.9	145.9	28.1	-173.9	0.47	1.5
0.300	3.0	89.9	37.3	-127.1	0.63	2.1
0.400	2.4	56.3	44.6	-100.9	0.75	2.5
0.460	2.1	42.3	48.2	-90.6	0.81	2.7
0.500	1.9	34.8	50.4	-85.1	0.85	2.8
0.600	1.4	20.3	54.8	-75.2	0.92	3.0
0.700	1.0	10.7	58.2	-68.8	0.98	3.2
0.750	0.8	7.2	59.4	-66.6	1.00	3.3
0.800	0.7	4.4	60.5	-64.9	1.02	3.3
0.900	0.3	1.0	61.8	-62.8	1.04	3.4
0.990		0.0	62.1	-62.1	1.04	3.4
1.000						
1.100						
1.150						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.89	m	
Mass of Person	m = 39.46	kg =	87 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.23	m^2 =	2.489583 ft^2
Length of Person	L = 0.76	m =	2.48958333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.5	314.3	19.0	-333.4	0.23	0.7
0.200	2.3	127.6	29.8	-157.4	0.35	1.2
0.300	1.6	61.5	37.1	-98.6	0.44	1.4
0.400	1.1	30.5	42.2	-72.6	0.50	1.6
0.460	0.9	19.3	44.4	-63.8	0.53	1.7
0.500	0.7	13.8	45.7	-59.5	0.54	1.8
0.600	0.4	4.8	47.9	-52.7	0.57	1.9
0.700	0.1	0.6	49.0	-49.7	0.58	1.9
0.750		0.0	49.2	-49.2	0.58	1.9
0.800						
0.900						
0.990						
1.000						
1.100						
1.150						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 39.46	kg =	87 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.46	m^2 =	4.979167 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.2	262.2	37.4	-299.6	0.18	0.6
0.200	1.2	72.8	53.1	-125.9	0.25	0.8
0.300	0.6	21.0	61.7	-82.7	0.29	1.0
0.400	0.2	2.9	65.7	-68.6	0.31	1.0
0.460		0.0	66.4	-66.4	0.32	1.0
0.500						
0.600						
0.700						
0.750						
0.800						
0.900						
0.990						
1.000						
1.100						
1.150						

Drops Vertically into the Pool

Height of COM	h = 1.31	m	
Mass of Person	m = 48.98	kg =	108 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.61	m =	5.27083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.4	281.8	11.1	-292.9	0.28	0.9
0.200	4.3	178.8	19.7	-198.6	0.50	1.6
0.300	3.5	120.2	26.7	-147.0	0.67	2.2
0.400	2.9	83.4	32.5	-115.9	0.82	2.7
0.500	2.5	58.6	37.3	-95.9	0.94	3.1
0.600	2.1	41.1	41.4	-82.5	1.04	3.4
0.700	1.7	28.3	44.8	-73.1	1.13	3.7
0.800	1.4	18.9	47.6	-66.5	1.20	3.9
0.870	1.2	13.8	49.2	-63.0	1.24	4.1
0.900	1.1	11.9	49.8	-61.8	1.25	4.1
1.000	0.8	6.9	51.6	-58.4	1.30	4.3
1.100	0.6	3.3	52.8	-56.2	1.33	4.4
1.150	0.4	2.1	53.3	-55.4	1.34	4.4
1.200	0.3	1.1	53.7	-54.8	1.35	4.4
1.300	0.1	0.1	54.0	-54.1	1.36	4.5
1.330		0.0	54.0	-54.0	1.36	4.5

Drops Diagonally into the Pool

Height of COM	h = 1.08	m	
Mass of Person	m = 48.98	kg =	108 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.14	m =	3.72704199 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.4	280.9	15.7	-296.6	0.28	0.9
0.200	4.3	176.4	27.8	-204.2	0.49	1.6
0.300	3.5	116.3	37.6	-154.0	0.67	2.2
0.400	2.8	78.3	45.6	-123.9	0.81	2.7
0.500	2.3	52.6	52.2	-104.8	0.93	3.0
0.600	1.9	34.6	57.5	-92.1	1.02	3.4
0.700	1.5	21.7	61.8	-83.5	1.10	3.6
0.800	1.1	12.5	65.1	-77.6	1.16	3.8
0.870	0.9	7.8	66.9	-74.7	1.19	3.9
0.900	0.8	6.2	67.6	-73.7	1.20	3.9
1.000	0.5	2.2	69.1	-71.3	1.23	4.0
1.100	0.1	0.2	69.9	-70.2	1.24	4.1
1.150		0.0	70.0	-70.0	1.24	4.1
1.200						
1.300						
1.330						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.91	m	
Mass of Person	m = 48.98	kg =	108 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.24	m^2 =	2.635417 ft^2
Length of Person	L = 0.80	m =	2.63541667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.8	389.3	18.7	-408.0	0.24	0.8
0.200	2.6	170.8	30.0	-200.8	0.38	1.2
0.300	1.8	88.5	37.8	-126.3	0.48	1.6
0.400	1.4	48.3	43.6	-91.9	0.55	1.8
0.500	1.0	25.8	47.8	-73.6	0.60	2.0
0.600	0.7	12.5	50.8	-63.3	0.64	2.1
0.700	0.4	4.7	52.8	-57.5	0.66	2.2
0.800	0.2	0.8	53.9	-54.7	0.68	2.2
0.870		0.0	54.1	-54.1	0.68	2.2
0.900						
1.000						
1.100						
1.150						
1.200						
1.300						
1.330						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 48.98	kg =	108 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.49	m^2 =	5.270833 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.5	349.7	39.8	-389.6	0.19	0.6
0.200	1.4	107.6	57.9	-165.5	0.28	0.9
0.300	0.8	37.3	68.5	-105.8	0.33	1.1
0.400	0.4	9.6	74.4	-84.0	0.35	1.2
0.500	0.0	0.3	76.6	-76.9	0.37	1.2
0.600						
0.700						
0.800						
0.870						
0.900						
1.000						
1.100						
1.150						
1.200						
1.300						
1.330						

Drops Vertically into the Pool

Height of COM	h = 1.35	m	
Mass of Person	m = 65.31	kg =	144 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.69	m =	5.54166667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	316.4	10.9	-327.3	0.29	0.9
0.200	4.8	219.8	19.8	-239.5	0.52	1.7
0.300	4.1	158.7	27.3	-186.0	0.72	2.4
0.400	3.5	117.3	33.7	-151.1	0.89	2.9
0.500	3.0	87.9	39.3	-127.2	1.04	3.4
0.600	2.6	66.1	44.1	-110.2	1.17	3.8
0.700	2.3	49.6	48.3	-97.9	1.28	4.2
0.800	2.0	36.8	51.9	-88.7	1.37	4.5
0.900	1.7	26.8	55.0	-81.8	1.45	4.8
1.000	1.4	18.9	57.6	-76.5	1.52	5.0
1.100	1.1	12.7	59.8	-72.5	1.58	5.2
1.200	0.9	8.0	61.5	-69.5	1.63	5.3
1.300	0.7	4.5	62.9	-67.3	1.66	5.5
1.380	0.5	2.4	63.7	-66.1	1.68	5.5
1.400	0.4	2.0	63.8	-65.8	1.69	5.5
1.500	0.2	0.5	64.4	-65.0	1.70	5.6
1.600		0.0	64.6	-64.6	1.71	5.6

Drops Diagonally into the Pool

Height of COM	h = 1.11	m	
Mass of Person	m = 65.31	kg =	144 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.19	m =	3.91855008 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	315.7	15.3	-331.1	0.29	0.9
0.200	4.8	217.7	27.9	-245.6	0.52	1.7
0.300	4.0	155.1	38.5	-193.6	0.72	2.4
0.400	3.4	112.5	47.4	-159.9	0.89	2.9
0.500	2.9	81.9	55.1	-137.0	1.03	3.4
0.600	2.5	59.3	61.6	-120.9	1.15	3.8
0.700	2.1	42.2	67.1	-109.3	1.25	4.1
0.800	1.7	29.1	71.7	-100.9	1.34	4.4
0.900	1.4	19.2	75.5	-94.7	1.41	4.6
1.000	1.1	11.6	78.5	-90.2	1.47	4.8
1.100	0.8	6.2	80.8	-87.0	1.51	5.0
1.200	0.5	2.5	82.4	-84.9	1.54	5.1
1.300	0.2	0.5	83.2	-83.7	1.56	5.1
1.380		0.0	83.4	-83.4	1.56	5.1
1.400						
1.500						
1.600						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.93	m	
Mass of Person	m = 65.31	kg =	144 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.26	m^2 =	2.770833 ft^2
Length of Person	L = 0.84	m =	2.77083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.2	502.0	18.8	-520.8	0.25	0.8
0.200	3.0	244.5	31.0	-275.5	0.41	1.3
0.300	2.3	137.5	39.9	-177.4	0.53	1.7
0.400	1.7	82.2	46.7	-128.9	0.62	2.0
0.500	1.4	49.8	52.0	-101.8	0.69	2.3
0.600	1.0	29.5	56.1	-85.6	0.74	2.4
0.700	0.8	16.2	59.2	-75.4	0.78	2.6
0.800	0.5	7.7	61.4	-69.1	0.81	2.7
0.900	0.3	2.6	62.8	-65.4	0.83	2.7
1.000	0.1	0.2	63.5	-63.7	0.84	2.8
1.100						
1.200						
1.300						
1.380						
1.400						
1.500						
1.600						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 65.31	kg =	144 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.51	m^2 =	5.541667 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.9	501.1	43.3	-544.4	0.21	0.7
0.200	1.8	175.2	65.2	-240.4	0.31	1.0
0.300	1.1	72.2	78.8	-151.0	0.38	1.2
0.400	0.7	27.5	87.5	-115.0	0.42	1.4
0.500	0.3	6.9	92.4	-99.3	0.44	1.4
0.600	0.0	0.1	94.1	-94.2	0.45	1.5
0.700						
0.800						
0.900						
1.000						
1.100						
1.200						
1.300						
1.380						
1.400						
1.500						
1.600						

10-year-old Boy Calculations

Drops Vertically into the Pool

Height of COM	h = 1.16	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.30	m =	4.27083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.4	195.3	12.4	-207.7	0.25	0.8
0.200	3.1	96.7	20.7	-117.4	0.42	1.4
0.300	2.3	53.1	26.7	-79.9	0.54	1.8
0.370	1.9	35.5	30.0	-65.6	0.61	2.0
0.400	1.7	29.9	31.2	-61.1	0.64	2.1
0.500	1.3	16.1	34.6	-50.7	0.70	2.3
0.570	1.0	9.8	36.3	-46.2	0.74	2.4
0.600	0.9	7.7	37.0	-44.7	0.75	2.5
0.700	0.5	2.8	38.5	-41.3	0.79	2.6
0.730	0.4	1.8	38.8	-40.6	0.79	2.6
0.800	0.2	0.4	39.3	-39.7	0.80	2.6
0.860		0.0	39.4	-39.4	0.80	2.6

Drops Diagonally into the Pool

Height of COM	h = 0.97	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 0.92	m =	3.01993521 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = Vg(p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.4	193.8	17.6	-211.4	0.25	0.8
0.200	3.1	93.4	29.2	-122.6	0.42	1.4
0.300	2.2	48.7	37.4	-86.1	0.54	1.8
0.370	1.8	30.6	41.8	-72.4	0.60	2.0
0.400	1.6	24.8	43.4	-68.2	0.62	2.1
0.500	1.0	11.1	47.5	-58.6	0.68	2.2
0.570	0.7	5.3	49.5	-54.8	0.71	2.3
0.600	0.6	3.5	50.1	-53.6	0.72	2.4
0.700	0.1	0.3	51.2	-51.5	0.74	2.4
0.730		0.0	51.3	-51.3	0.74	2.4
0.800						
0.860						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.83	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.20	m^2 =	2.135417 ft^2
Length of Person	L = 0.65	m =	2.13541667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.0	202.2	20.6	-222.7	0.21	0.7
0.200	1.8	70.2	31.0	-101.3	0.32	1.0
0.300	1.1	27.8	37.5	-65.3	0.38	1.3
0.370	0.8	13.6	40.5	-54.0	0.41	1.4
0.400	0.6	9.5	41.5	-50.9	0.42	1.4
0.500	0.2	1.6	43.5	-45.1	0.44	1.5
0.570		0.0	43.9	-43.9	0.45	1.5
0.600						
0.700						
0.730						
0.800						
0.860						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 26.30	kg =	58 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.40	m^2 =	4.270833 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	1.8	149.1	33.4	-182.4	0.16	0.5
0.200	0.8	32.8	45.5	-78.3	0.22	0.7
0.300	0.3	4.9	51.0	-55.9	0.24	0.8
0.370		0.0	52.1	-52.1	0.25	0.8
0.400						
0.500						
0.570						
0.600						
0.700						
0.730						
0.800						
0.860						

Drops Vertically into the Pool

Height of COM	h = 1.20	m	
Mass of Person	m = 31.75	kg =	70 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.38	m =	4.54166667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.7	222.4	12.1	-234.5	0.26	0.9
0.200	3.5	119.6	20.6	-140.2	0.45	1.5
0.300	2.7	70.4	27.0	-97.4	0.59	1.9
0.400	2.1	42.8	32.0	-74.8	0.69	2.3
0.500	1.6	25.8	35.8	-61.6	0.78	2.5
0.600	1.2	14.7	38.8	-53.5	0.84	2.8
0.650	1.0	10.7	40.0	-50.7	0.87	2.8
0.700	0.9	7.5	41.0	-48.5	0.89	2.9
0.800	0.5	3.0	42.5	-45.5	0.92	3.0
0.840	0.4	1.8	42.9	-44.7	0.93	3.0
0.900	0.2	0.6	43.3	-43.9	0.94	3.1
0.980		0.0	43.5	-43.5	0.94	3.1

Drops Diagonally into the Pool

Height of COM	h = 1.00	m	
Mass of Person	m = 31.75	kg =	70 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 0.98	m =	3.2114433 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = Vg(p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.7	221.2	17.1	-238.3	0.26	0.9
0.200	3.4	116.6	29.0	-145.6	0.44	1.5
0.300	2.6	66.0	37.9	-103.9	0.58	1.9
0.400	2.0	37.5	44.6	-82.1	0.68	2.2
0.500	1.4	20.2	49.6	-69.8	0.76	2.5
0.600	1.0	9.4	53.1	-62.6	0.81	2.7
0.650	0.7	5.8	54.4	-60.2	0.83	2.7
0.700	0.5	3.1	55.4	-58.5	0.85	2.8
0.800	0.1	0.3	56.4	-56.7	0.86	2.8
0.840		0.0	56.5	-56.5	0.87	2.8
0.900						
0.980						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.85	m	
Mass of Person	m = 31.75	kg =	70 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.21	m^2 =	2.270833 ft^2
Length of Person	L = 0.69	m =	2.27083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = Vg(\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.3	249.7	20.1	-269.8	0.22	0.7
0.200	2.0	94.0	30.9	-124.9	0.34	1.1
0.300	1.3	41.4	37.9	-79.3	0.41	1.3
0.400	0.9	17.5	42.6	-60.1	0.46	1.5
0.500	0.5	5.7	45.4	-51.1	0.49	1.6
0.600	0.1	0.6	46.8	-47.4	0.51	1.7
0.650		0.0	46.9	-46.9	0.51	1.7
0.700						
0.800						
0.840						
0.900						
0.980						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 31.75	kg =	70 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.42	m^2 =	4.541667 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.0	196.0	35.4	-231.4	0.17	0.6
0.200	1.0	48.8	49.3	-98.1	0.24	0.8
0.300	0.5	10.7	56.2	-67.0	0.27	0.9
0.400	0.0	0.2	58.7	-58.9	0.28	0.9
0.500						
0.600						
0.650						
0.700						
0.800						
0.840						
0.900						
0.980						

Drops Vertically into the Pool

Height of COM	h = 1.24	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.47	m =	4.83333333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.2	260.6	11.8	-272.4	0.27	0.9
0.200	4.0	156.0	20.8	-176.8	0.48	1.6
0.300	3.2	100.0	27.8	-127.8	0.64	2.1
0.400	2.6	66.3	33.5	-99.7	0.77	2.5
0.480	2.2	48.0	37.3	-85.2	0.86	2.8
0.500	2.1	44.2	38.1	-82.3	0.88	2.9
0.600	1.7	29.1	41.9	-71.0	0.97	3.2
0.700	1.4	18.4	44.9	-63.4	1.04	3.4
0.770	1.1	12.9	46.7	-59.6	1.08	3.5
0.800	1.1	10.9	47.3	-58.2	1.09	3.6
0.900	0.8	5.6	49.1	-54.7	1.13	3.7
1.000	0.5	2.2	50.3	-52.5	1.16	3.8
1.010	0.4	2.0	50.4	-52.4	1.16	3.8
1.100	0.2	0.4	51.0	-51.4	1.17	3.9
1.170		0.0	51.1	-51.1	1.18	3.9

Drops Diagonally into the Pool

Height of COM	h = 1.03	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.04	m =	3.41768278 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	259.5	16.7	-276.3	0.27	0.9
0.200	4.0	153.2	29.3	-182.5	0.48	1.6
0.300	3.1	95.8	39.1	-134.9	0.64	2.1
0.400	2.5	60.9	46.9	-107.7	0.76	2.5
0.480	2.1	42.0	51.9	-93.9	0.85	2.8
0.500	2.0	38.1	53.1	-91.2	0.87	2.8
0.600	1.5	22.7	57.9	-80.6	0.94	3.1
0.700	1.1	12.3	61.6	-73.9	1.00	3.3
0.770	0.8	7.2	63.5	-70.6	1.03	3.4
0.800	0.7	5.4	64.1	-69.6	1.05	3.4
0.900	0.4	1.5	65.7	-67.2	1.07	3.5
1.000	0.0	0.0	66.2	-66.2	1.08	3.5
1.010		0.0	66.2	-66.2	1.08	3.5
1.100						
1.170						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.88	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.22	m^2 =	2.416667 ft^2
Length of Person	L = 0.74	m =	2.41666667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.7	331.9	20.0	-351.9	0.23	0.8
0.200	2.4	139.3	31.8	-171.1	0.37	1.2
0.300	1.7	68.7	39.7	-108.4	0.46	1.5
0.400	1.2	34.8	45.4	-80.2	0.52	1.7
0.480	0.9	19.3	48.7	-68.0	0.56	1.8
0.500	0.8	16.4	49.4	-65.7	0.57	1.9
0.600	0.5	6.1	52.0	-58.1	0.60	2.0
0.700	0.2	1.1	53.3	-54.4	0.61	2.0
0.770		0.0	53.6	-53.6	0.62	2.0
0.800						
0.900						
1.000						
1.010						
1.100						
1.170						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 41.72	kg =	92 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.45	m^2 =	4.833333 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.4	287.9	38.7	-326.6	0.18	0.6
0.200	1.3	83.4	55.6	-139.0	0.27	0.9
0.300	0.7	25.8	65.0	-90.8	0.31	1.0
0.400	0.3	4.5	69.8	-74.4	0.33	1.1
0.480		0.0	70.9	-70.9	0.34	1.1
0.500						
0.600						
0.700						
0.770						
0.800						
0.900						
1.000						
1.010						
1.100						
1.170						

12-year-old Boy Calculations

Drops Vertically into the Pool

Height of COM	h = 1.21	m	
Mass of Person	m = 32.20	kg =	71 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.40	m =	4.58333333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.8	224.5	12.0	-236.5	0.26	0.9
0.200	3.5	121.5	20.5	-142.0	0.45	1.5
0.300	2.7	71.9	26.9	-98.8	0.59	1.9
0.400	2.1	43.9	31.9	-75.8	0.70	2.3
0.410	2.1	41.8	32.3	-74.2	0.71	2.3
0.500	1.7	26.7	35.8	-62.4	0.78	2.6
0.600	1.3	15.4	38.8	-54.2	0.85	2.8
0.650	1.1	11.3	40.0	-51.3	0.87	2.9
0.700	0.9	8.0	41.0	-49.0	0.90	2.9
0.800	0.6	3.3	42.5	-45.9	0.93	3.1
0.850	0.4	1.8	43.0	-44.9	0.94	3.1
0.900	0.3	0.8	43.4	-44.2	0.95	3.1
0.990		0.0	43.6	-43.7	0.95	3.1
1.000						

Drops Diagonally into the Pool

Height of COM	h = 1.00	m	
Mass of Person	m = 32.20	kg =	71 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 0.99	m =	3.24090608 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.7	223.2	17.0	-240.2	0.26	0.9
0.200	3.5	118.4	28.9	-147.3	0.45	1.5
0.300	2.6	67.5	37.8	-105.3	0.58	1.9
0.400	2.0	38.7	44.5	-83.2	0.69	2.3
0.410	1.9	36.5	45.0	-81.6	0.70	2.3
0.500	1.5	21.1	49.5	-70.6	0.77	2.5
0.600	1.0	10.1	53.1	-63.2	0.82	2.7
0.650	0.8	6.3	54.4	-60.8	0.84	2.8
0.700	0.6	3.5	55.4	-59.0	0.86	2.8
0.800	0.2	0.4	56.6	-57.0	0.87	2.9
0.850		0.0	56.7	-56.7	0.88	2.9
0.900						
0.990						
1.000						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.86	m	
Mass of Person	m = 32.20	kg =	71 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.21	m^2 =	2.291667 ft^2
Length of Person	L = 0.70	m =	2.29166667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.3	253.6	20.0	-273.5	0.22	0.7
0.200	2.0	95.8	30.7	-126.6	0.34	1.1
0.300	1.4	42.5	37.7	-80.2	0.41	1.4
0.400	0.9	18.2	42.3	-60.6	0.46	1.5
0.410	0.8	16.6	42.7	-59.3	0.47	1.5
0.500	0.5	6.2	45.2	-51.4	0.49	1.6
0.600	0.2	0.8	46.7	-47.5	0.51	1.7
0.650		0.0	46.9	-46.9	0.51	1.7
0.700						
0.800						
0.850						
0.900						
0.990						
1.000						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 32.20	kg =	71 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.43	m^2 =	4.583333 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.0	199.5	35.5	-235.0	0.17	0.6
0.200	1.0	50.0	49.5	-99.4	0.24	0.8
0.300	0.5	11.2	56.5	-67.7	0.27	0.9
0.400	0.0	0.3	59.0	-59.3	0.28	0.9
0.410		0.1	59.0	-59.1	0.28	0.9
0.500						
0.600						
0.650						
0.700						
0.800						
0.850						
0.900						
0.990						
1.000						

Drops Vertically into the Pool

Height of COM	h = 1.25	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.49	m =	4.89583333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	256.1	11.6	-267.8	0.27	0.9
0.200	3.9	151.6	20.3	-171.9	0.47	1.6
0.300	3.2	96.4	27.2	-123.5	0.63	2.1
0.400	2.6	63.4	32.7	-96.0	0.76	2.5
0.470	2.2	47.5	35.9	-83.4	0.84	2.8
0.500	2.1	42.0	37.1	-79.1	0.87	2.8
0.600	1.7	27.4	40.8	-68.1	0.95	3.1
0.700	1.3	17.1	43.7	-60.8	1.02	3.3
0.760	1.1	12.5	45.1	-57.6	1.05	3.5
0.800	1.0	9.9	45.9	-55.9	1.07	3.5
0.900	0.7	5.0	47.6	-52.6	1.11	3.6
0.990	0.4	2.1	48.6	-50.7	1.14	3.7
1.000	0.4	1.9	48.7	-50.6	1.14	3.7
1.100	0.1	0.3	49.3	-49.5	1.15	3.8
1.160		0.0	49.3	-49.3	1.15	3.8

Drops Diagonally into the Pool

Height of COM	h = 1.04	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.06	m =	3.46187695 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	255.1	16.5	-271.5	0.27	0.9
0.200	3.9	148.8	28.7	-177.5	0.47	1.6
0.300	3.1	92.2	38.2	-130.4	0.63	2.1
0.400	2.4	58.1	45.7	-103.8	0.76	2.5
0.470	2.1	41.7	50.0	-91.8	0.83	2.7
0.500	1.9	36.0	51.7	-87.7	0.85	2.8
0.600	1.5	21.2	56.3	-77.5	0.93	3.1
0.700	1.1	11.2	59.8	-71.0	0.99	3.2
0.760	0.8	7.0	61.3	-68.3	1.01	3.3
0.800	0.7	4.8	62.2	-66.9	1.03	3.4
0.900	0.3	1.2	63.6	-64.7	1.05	3.4
0.990		0.0	64.0	-64.0	1.06	3.5
1.000						
1.100						
1.160						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.88	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.23	m^2 =	2.447917 ft^2
Length of Person	L = 0.75	m =	2.44791667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.6	321.4	19.6	-341.0	0.23	0.7
0.200	2.3	132.5	30.8	-163.3	0.36	1.2
0.300	1.6	64.5	38.4	-102.9	0.45	1.5
0.400	1.1	32.2	43.8	-76.0	0.51	1.7
0.470	0.9	19.0	46.5	-65.5	0.54	1.8
0.500	0.8	14.8	47.5	-62.3	0.55	1.8
0.600	0.5	5.3	49.9	-55.2	0.58	1.9
0.700	0.2	0.8	51.1	-51.9	0.60	2.0
0.760		0.0	51.3	-51.3	0.60	2.0
0.800						
0.900						
0.990						
1.000						
1.100						
1.160						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.45	m^2 =	4.895833 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.3	273.1	38.0	-311.0	0.18	0.6
0.200	1.2	77.3	54.3	-131.5	0.26	0.8
0.300	0.7	23.0	63.2	-86.2	0.30	1.0
0.400	0.2	3.5	67.6	-71.1	0.32	1.1
0.470		0.0	68.4	-68.4	0.33	1.1
0.500						
0.600						
0.700						
0.760						
0.800						
0.900						
0.990						
1.000						
1.100						
1.160						

Drops Vertically into the Pool

Height of COM	h = 1.30	m	
Mass of Person	m = 53.51	kg =	118 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.59	m =	5.22916667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.5	292.9	11.3	-304.2	0.28	0.9
0.200	4.5	191.3	20.2	-211.5	0.50	1.7
0.300	3.7	131.5	27.6	-159.0	0.69	2.3
0.400	3.1	93.0	33.7	-126.7	0.84	2.8
0.500	2.6	66.6	38.9	-105.4	0.97	3.2
0.540	2.5	58.3	40.7	-99.0	1.02	3.3
0.600	2.2	47.7	43.3	-90.9	1.08	3.5
0.700	1.9	33.7	47.0	-80.7	1.17	3.8
0.800	1.6	23.3	50.0	-73.3	1.25	4.1
0.900	1.3	15.4	52.6	-68.0	1.31	4.3
0.910	1.2	14.7	52.8	-67.5	1.32	4.3
1.000	1.0	9.5	54.6	-64.1	1.36	4.5
1.100	0.7	5.2	56.2	-61.4	1.40	4.6
1.200	0.5	2.3	57.3	-59.6	1.43	4.7
1.300	0.2	0.6	57.9	-58.5	1.44	4.7
1.400		0.0	58.1	-58.1	1.45	4.8

Drops Diagonally into the Pool

Height of COM	h = 1.07	m	
Mass of Person	m = 53.51	kg =	118 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.13	m =	3.69757921 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.5	292.0	15.9	-308.0	0.28	0.9
0.200	4.4	188.9	28.5	-217.5	0.50	1.7
0.300	3.6	127.6	38.8	-166.4	0.68	2.2
0.400	3.0	87.8	47.3	-135.1	0.83	2.7
0.500	2.5	60.5	54.3	-114.8	0.96	3.1
0.540	2.3	51.9	56.8	-108.7	1.00	3.3
0.600	2.1	40.9	60.2	-101.1	1.06	3.5
0.700	1.7	26.7	64.9	-91.6	1.15	3.8
0.800	1.3	16.3	68.7	-85.0	1.21	4.0
0.900	0.9	8.9	71.6	-80.5	1.26	4.1
0.910	0.9	8.3	71.8	-80.1	1.27	4.2
1.000	0.6	3.9	73.6	-77.5	1.30	4.3
1.100	0.3	1.0	74.8	-75.8	1.32	4.3
1.200		0.0	75.2	-75.2	1.33	4.4
1.300						
1.400						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.91	m	
Mass of Person	m = 53.51	kg =	118 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.24	m^2 =	2.614583 ft^2
Length of Person	L = 0.80	m =	2.61458333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.0	420.9	19.3	-440.2	0.24	0.8
0.200	2.7	192.3	31.3	-223.6	0.39	1.3
0.300	2.0	102.6	39.8	-142.4	0.50	1.6
0.400	1.5	57.7	46.1	-103.8	0.58	1.9
0.500	1.1	32.2	50.8	-83.0	0.63	2.1
0.540	1.0	25.1	52.4	-77.4	0.65	2.1
0.600	0.8	16.7	54.3	-71.0	0.68	2.2
0.700	0.5	7.3	56.7	-64.0	0.71	2.3
0.800	0.3	2.0	58.2	-60.2	0.73	2.4
0.900	0.0	0.0	58.7	-58.7	0.73	2.4
0.910		0.0	58.7	-58.7	0.73	2.4
1.000						
1.100						
1.200						
1.300						
1.400						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 53.51	kg =	118 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.49	m^2 =	5.229167 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.7	394.8	41.3	-436.1	0.20	0.6
0.200	1.5	127.5	60.8	-188.4	0.29	1.0
0.300	0.9	47.3	72.5	-119.8	0.35	1.1
0.400	0.5	14.3	79.4	-93.7	0.38	1.2
0.500	0.1	1.5	82.5	-84.0	0.39	1.3
0.540		0.1	82.9	-82.9	0.40	1.3
0.600						
0.700						
0.800						
0.900						
0.910						
1.000						
1.100						
1.200						
1.300						
1.400						

14-year-old Boy Calculations

Drops Vertically into the Pool

Height of COM	h = 1.28	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.54	m =	5.04166667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	256.2	11.3	-267.5	0.27	0.9
0.200	3.9	151.8	19.7	-171.5	0.48	1.6
0.300	3.2	96.7	26.4	-123.1	0.63	2.1
0.400	2.6	63.7	31.7	-95.5	0.76	2.5
0.470	2.2	47.9	34.9	-82.8	0.84	2.8
0.500	2.1	42.4	36.1	-78.5	0.87	2.8
0.600	1.7	27.8	39.7	-67.5	0.95	3.1
0.700	1.3	17.6	42.5	-60.1	1.02	3.4
0.770	1.1	12.3	44.1	-56.4	1.06	3.5
0.800	1.0	10.4	44.7	-55.1	1.08	3.5
0.900	0.7	5.4	46.4	-51.8	1.12	3.7
1.000	0.5	2.1	47.5	-49.7	1.14	3.7
1.010	0.4	1.9	47.6	-49.5	1.14	3.8
1.100	0.2	0.4	48.1	-48.5	1.16	3.8
1.110	0.2	0.3	48.2	-48.5	1.16	3.8
1.170		0.0	48.3	-48.3	1.16	3.8

Drops Diagonally into the Pool

Height of COM	h = 1.05	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.09	m =	3.56499669 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.1	255.2	16.0	-271.2	0.27	0.9
0.200	3.9	149.1	27.9	-177.0	0.47	1.6
0.300	3.1	92.6	37.1	-129.7	0.63	2.1
0.400	2.5	58.6	44.4	-103.0	0.76	2.5
0.470	2.1	42.3	48.7	-90.9	0.83	2.7
0.500	1.9	36.6	50.3	-86.8	0.85	2.8
0.600	1.5	21.8	54.8	-76.6	0.93	3.1
0.700	1.1	11.7	58.2	-70.0	0.99	3.2
0.770	0.8	6.8	60.0	-66.9	1.02	3.3
0.800	0.7	5.2	60.6	-65.8	1.03	3.4
0.900	0.4	1.4	62.1	-63.5	1.06	3.5
1.000	0.0	0.0	62.6	-62.6	1.06	3.5
1.010		0.0	62.6	-62.6	1.06	3.5
1.100						
1.110						
1.170						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.89	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.23	m^2 =	2.520833 ft^2
Length of Person	L = 0.77	m =	2.52083333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.5	321.8	18.8	-340.6	0.23	0.7
0.200	2.3	131.5	29.6	-161.1	0.36	1.2
0.300	1.6	63.9	36.8	-100.7	0.44	1.5
0.400	1.1	32.0	41.9	-74.0	0.50	1.7
0.470	0.9	19.0	44.6	-63.6	0.54	1.8
0.500	0.8	14.9	45.5	-60.4	0.55	1.8
0.600	0.5	5.5	47.8	-53.3	0.57	1.9
0.700	0.2	0.9	49.0	-49.9	0.59	1.9
0.770		0.0	49.2	-49.2	0.59	1.9
0.800						
0.900						
1.000						
1.010						
1.100						
1.110						
1.170						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 40.36	kg =	89 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.47	m^2 =	5.041667 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.2	269.8	37.5	-307.3	0.18	0.6
0.200	1.2	75.6	53.4	-129.1	0.25	0.8
0.300	0.6	22.3	62.2	-84.4	0.30	1.0
0.400	0.2	3.3	66.4	-69.7	0.32	1.0
0.470		0.0	67.1	-67.1	0.32	1.1
0.500						
0.600						
0.700						
0.770						
0.800						
0.900						
1.000						
1.010						
1.100						
1.110						
1.170						

Drops Vertically into the Pool

Height of COM	h = 1.33	m	
Mass of Person	m = 50.79	kg =	112 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.64	m =	5.375 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.4	286.4	10.9	-297.4	0.28	0.9
0.200	4.4	184.1	19.5	-203.6	0.50	1.6
0.300	3.6	125.0	26.5	-151.5	0.68	2.2
0.400	3.0	87.5	32.3	-119.8	0.83	2.7
0.500	2.5	62.1	37.1	-99.3	0.95	3.1
0.530	2.4	56.1	38.4	-94.5	0.99	3.2
0.600	2.1	44.1	41.2	-85.3	1.06	3.5
0.700	1.8	30.9	44.7	-75.6	1.15	3.8
0.800	1.5	21.0	47.6	-68.6	1.22	4.0
0.890	1.2	14.3	49.7	-64.0	1.27	4.2
0.900	1.2	13.7	49.9	-63.6	1.28	4.2
1.000	0.9	8.2	51.8	-60.0	1.33	4.4
1.100	0.7	4.3	53.2	-57.5	1.36	4.5
1.180	0.5	2.2	54.0	-56.1	1.38	4.5
1.200	0.4	1.7	54.1	-55.8	1.39	4.6
1.300	0.2	0.3	54.6	-55.0	1.40	4.6
1.370		0.0	54.7	-54.7	1.40	4.6

Drops Diagonally into the Pool

Height of COM	h = 1.09	m	
Mass of Person	m = 50.79	kg =	112 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.16	m =	3.80069895 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.4	285.6	15.4	-301.0	0.28	0.9
0.200	4.3	181.7	27.5	-209.2	0.50	1.6
0.300	3.5	121.2	37.3	-158.5	0.68	2.2
0.400	2.9	82.5	45.3	-127.8	0.82	2.7
0.500	2.4	56.2	51.9	-108.1	0.94	3.1
0.530	2.3	50.0	53.7	-103.6	0.97	3.2
0.600	2.0	37.6	57.4	-95.0	1.04	3.4
0.700	1.6	24.2	61.8	-85.9	1.12	3.7
0.800	1.2	14.5	65.2	-79.7	1.18	3.9
0.890	0.9	8.2	67.6	-75.8	1.23	4.0
0.900	0.9	7.6	67.9	-75.5	1.23	4.0
1.000	0.5	3.1	69.6	-72.8	1.26	4.1
1.100	0.2	0.6	70.6	-71.3	1.28	4.2
1.180		0.0	70.9	-70.9	1.29	4.2
1.200						
1.300						
1.370						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.92	m	
Mass of Person	m = 50.79	kg =	112 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.25	m^2 =	2.6875 ft^2
Length of Person	L = 0.82	m =	2.6875 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	3.8	403.4	18.5	-421.8	0.24	0.8
0.200	2.6	178.7	29.6	-208.3	0.38	1.2
0.300	1.9	93.5	37.4	-130.9	0.48	1.6
0.400	1.4	51.7	43.2	-94.9	0.55	1.8
0.500	1.0	28.2	47.4	-75.7	0.61	2.0
0.530	0.9	23.3	48.5	-71.8	0.62	2.0
0.600	0.7	14.2	50.5	-64.7	0.65	2.1
0.700	0.5	5.8	52.7	-58.5	0.68	2.2
0.800	0.2	1.4	53.9	-55.2	0.69	2.3
0.890		0.0	54.2	-54.2	0.69	2.3
0.900						
1.000						
1.100						
1.180						
1.200						
1.300						
1.370						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 50.79	kg =	112 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.50	m^2 =	5.375 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.5	365.3	40.1	-405.4	0.19	0.6
0.200	1.4	113.8	58.5	-172.3	0.28	0.9
0.300	0.9	40.4	69.3	-109.6	0.33	1.1
0.400	0.4	11.0	75.4	-86.4	0.36	1.2
0.500	0.1	0.6	77.9	-78.5	0.37	1.2
0.530		0.0	78.0	-78.0	0.37	1.2
0.600						
0.700						
0.800						
0.890						
0.900						
1.000						
1.100						
1.180						
1.200						
1.300						
1.370						

Drops Vertically into the Pool

Height of COM	h = 1.38	m	
Mass of Person	m = 66.21	kg =	146 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.74	m =	5.70833333 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	318.0	10.5	-328.6	0.29	0.9
0.200	4.8	221.9	19.2	-241.1	0.52	1.7
0.300	4.1	160.8	26.6	-187.4	0.72	2.4
0.400	3.5	119.4	32.9	-152.3	0.90	2.9
0.500	3.1	89.8	38.3	-128.2	1.04	3.4
0.600	2.7	68.0	43.1	-111.0	1.17	3.8
0.700	2.3	51.3	47.2	-98.5	1.28	4.2
0.800	2.0	38.4	50.7	-89.1	1.38	4.5
0.900	1.7	28.2	53.8	-82.0	1.47	4.8
1.000	1.4	20.2	56.4	-76.6	1.54	5.0
1.100	1.2	13.9	58.6	-72.5	1.60	5.2
1.200	1.0	9.0	60.4	-69.4	1.65	5.4
1.300	0.7	5.2	61.8	-67.1	1.68	5.5
1.400	0.5	2.6	62.9	-65.4	1.71	5.6
1.500	0.3	0.9	63.5	-64.4	1.73	5.7
1.600	0.1	0.1	63.8	-63.9	1.74	5.7
1.640		0.0	63.9	-63.9	1.74	5.7

Drops Diagonally into the Pool

Height of COM	h = 1.12	m	
Mass of Person	m = 66.21	kg =	146 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.09	m^2 =	1 ft^2
Length of Person	L = 1.23	m =	4.03640121 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	5.7	317.3	14.9	-332.2	0.29	0.9
0.200	4.8	219.8	27.2	-247.0	0.52	1.7
0.300	4.1	157.4	37.5	-194.9	0.72	2.4
0.400	3.5	114.7	46.3	-160.9	0.89	2.9
0.500	3.0	84.0	53.8	-137.8	1.04	3.4
0.600	2.5	61.3	60.2	-121.5	1.16	3.8
0.700	2.1	44.0	65.6	-109.7	1.26	4.1
0.800	1.8	30.8	70.2	-101.0	1.35	4.4
0.900	1.5	20.6	74.0	-94.6	1.43	4.7
1.000	1.1	12.9	77.1	-90.0	1.48	4.9
1.100	0.9	7.2	79.4	-86.6	1.53	5.0
1.200	0.6	3.2	81.1	-84.3	1.56	5.1
1.300	0.3	0.9	82.1	-83.0	1.58	5.2
1.400	0.0	0.0	82.5	-82.5	1.59	5.2
1.500						
1.600						
1.640						

Drops with Tucked Knees into the Pool

Height of COM	h = 0.94	m	
Mass of Person	m = 66.21	kg =	146 lb
Density of Water	ρ_w = 1000.00	kg/m^3	
Density of Person	ρ_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.27	m^2 =	2.854167 ft^2
Length of Person	L = 0.87	m =	2.85416667 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A \rho_w v^2$

Bouyancy Equation $F_B = V g (\rho_w - \rho_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	4.2	511.0	18.2	-529.2	0.25	0.8
0.200	3.0	247.9	30.0	-277.8	0.41	1.3
0.300	2.2	139.4	38.5	-177.9	0.52	1.7
0.400	1.7	83.6	45.1	-128.7	0.61	2.0
0.500	1.4	51.0	50.2	-101.2	0.68	2.2
0.600	1.0	30.5	54.2	-84.6	0.74	2.4
0.700	0.8	17.1	57.2	-74.3	0.78	2.6
0.800	0.5	8.4	59.4	-67.8	0.81	2.7
0.900	0.3	3.1	60.8	-63.9	0.83	2.7
1.000	0.1	0.4	61.6	-62.0	0.84	2.8
1.100						
1.200						
1.300						
1.400						
1.500						
1.600						
1.640						

Drops Horizontally into the Pool

Height of COM	h = 0.66	m	
Mass of Person	m = 66.21	kg =	146 lb
Density of Water	p_w = 1000.00	kg/m^3	
Density of Person	p_B = 980.00	kg/m^3	
Cross-Sectional Area	A = 0.53	m^2 =	5.708333 ft^2
Length of Person	L = 0.30	m =	1 ft
Volume of Person	V = 0.33	m^3	
Coefficient of Drag	C_D = 1.00		
Gravitational Constant	g = 9.81	m/s^2	

Drag Force Equation $F_D = \frac{1}{2} C_D A p_w v^2$

Bouyancy Equation $F_B = V g (p_w - p_B)$

Initial Conditions		
t_o =	0	s
v_o =	3.157	mph

ELAPSED TIME (s)	**VELOCITY AT TIME (mph)**	DRAG FORCE (N)	BOUYANCY FORCE (N)	NET FORCE (N)	DISTANCE (m)	**TOTAL DEPTH (ft)**
0	**7.1**				0.0	**0.0**
0.100	2.9	506.8	43.1	-549.9	0.21	0.7
0.200	1.7	176.6	64.7	-241.3	0.31	1.0
0.300	1.1	72.8	78.2	-151.0	0.37	1.2
0.400	0.7	27.9	86.8	-114.7	0.41	1.4
0.500	0.3	7.1	91.7	-98.8	0.44	1.4
0.600	0.0	0.1	93.5	-93.6	0.45	1.5
0.700						
0.800						
0.900						
1.000						
1.100						
1.200						
1.300						
1.400						
1.500						
1.600						
1.640						

Product Solutions

biscuits • bones • treats • desserts

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NINJA CROSS™

NinjaCross MiniNinja Rules

1. Participants must be a minimum of 48-inches tall
2. Participants maximom weight of 275lbs
3. Wait your turn to start, follow direction by facility staff at all times
4. Diving, jumping, running, pushing, etc. is strictly prohibited
5. Participants to use systems solely at their own risk – this is a skill-based system and is meant to be challenging. Owner, operator, manufacturer and any additional parties will not be held responsible for any injury on the system
6. Climbing obstacles cables, structure column legs or any other components on the system is strictly prohibited
7. Touching obstacle frame or support truss, electronics, or any other components other than the obstacles is strictly prohibited
8. Only use if you are capable of safely swimming the length of the pool and able to hold your breath under-water for 10-seconds or more. Non-Swimmers are not permitted.
9. Only 1 participant per obstacle set at a time, no more than 3 participants on the system at one time
10. Use only under supervision of lifeguard or attendant
11. If you fall into water, move on to next obstacle or swim out of the lane
12. If you feel exhausted or weak, stop participation and swim out of lane to closest pool wall
13. Do not push, shove or harass other guests – bullying will not be tolerated and you may be asked to leave the facility
14. Do not use this equipment while under the influence of alcohol or drugs
15. No diving allowed anywhere around this system
16. Leave MiniNinja pool area promptly after completing the course or if you are unable to complete the course
17. Participants assume all risk of injury due to misuse of the NinjaCross MiniNinja or failure to follow rules

NINJA CROSS

NinjaCross Systems

MiniNinja

Standard Operating Procedures and Operations Manual V1.1

Contact Ninja*Cross* Systems at:

Phone- 800-778-9702

Email- Support@NinjaCrossSystems.com

Section 1

Introduction

The purpose of this operations manual is to provide the owner/operator with the basic rules and maintenance information necessary to operate the NinjaCross MiniNinja System in a manner designed to minimize problems and ensure the safety of the participant(s). This manual deals with the operation of the NinjaCross equipment only. It does not address pool operations, health codes, water quality, or local ordinances.

Facilities should follow the manufacturer's guidelines for installation, safe inspection, maintenance, operations and use of its various fitness systems and features. However, your employer should provide you with a specific set of guidelines and training if you are responsible for these inspections

Most local regulatory agencies have public swimming pool standards. It is recommended that local codes, regulations, and guidelines be followed. This will insure a harmonious relationship between the pool/slide operation and the local authorities.

To assist owners and operators in providing a safe, fun, and enjoyable experience for all facility patrons, NinjaCross Systems provides the following additional services;

- Annual NinjaCross Inspections
- Annual on-site safety training for lifeguards and operators
- Maintenance programs to prolong the life of your investment

Section 2

Terms

Box Truss - a type of truss that uses four major cords with connecting cords to form a strong structure that takes the shape of a rectangular box.

Corner Block - a 12” square aluminum block that mounts to the Aluminum Box truss section. All Static Lines attach at a Corner Block and all cross members of the Obstacle Frame attached at Corner Blocks.

Designated Safety Area - the area that includes all pool space under the obstacle frame and the adjacent 8-feet on either side of the Obstacle Frame stretching from end of pool to opposite end.

Eye Clamp - A clamp that allows attachment of a NetForm Rope or other item to the Obstacle Frame.

Mounting Plate - the square aluminum plate that secures the Obstacle Frame to the pool deck. The plate is anchored by wedge anchors.

NetForm Rope - the rope that connects an obstacle to the Obstacle Frame

Obstacle - a combination of aluminum parts, ropes, and hardware that create a means for the participant to traverse.

OAB (Obstacle Attachment Bar) - An aluminum bat that attached to the Obstacle Frame and allows Obstacles with dual ropes to be attached.

Obstacle Frame - the aluminum truss that Obstacles hang from, Static Cables and Lifting Cables attach to, and BackUp System attaches to.

Obstacle Frame Leg - the aluminum truss vertical sections that hold the Obstacle Frame at elevation. These legs are mounted to the pool deck via the Mounting Plates.

Participant - the guest that is using the NinjaCross MiniNinja system

Pinch Block - an aluminum block with indents that allows it to secure into the tube of the Obstacle Frame. Used for connecting Obstacles to the Obstacle Frame.

Safety Padding - a section of padding applied to deck and pool wall that protects participant from falls against the pool deck.

Swivel Clamp - A dual clamp system that allows attachment of the OAB to the Obstacle Frame.

Section 1

NinjaCross MiniNinja Standard Rules

1. Follow the directions of facility personnel at all times

2. Wait your turn prior to starting

3. Diving, jumping, running, pushing, etc. is strictly prohibited

4. Participants to use system solely at their own risk - this is a skill-based system and is meant to be challenging

5. Climbing obstacle cables, legs, or any other components on their system is strictly prohibited

6. Touching obstacle frame or support truss, electronics, or any other components other than the obstacles is strictly prohibited

7. Do climb the ropes or onto the Obstacle Frame. Do not try to hold onto the Obstacle Frame

8. Only use if you are capable of swimming and able to hold your breath under water for 10-seconds or more

9. Only one participant per obstacle set at a time. A maximum or 2 participants may be on a single lane at any one time. The minimum distance between participants shall be no less than 10’

10. Use only under the supervision of lifeguard or attendant

11. Swinging, leaping, jumping, or swimming in adjacent lane is strictly prohibited

12. No standing on Above Water Level obstacles

13. If you fall on an obstacle, move onto the next obstacle and attempt to complete

14. If you feel exhausted or weak, stop participation and swim out of lane to closet pool wall

15. Do not push, shove, or harass other guests - bullying will not be tolerated, and you will be asked to leave facility.

16. Recommended Minimum age 5 years old

17. Minimum Height 48” tall

18. Maximum Weight 270lbs

19. Participant must not wear lifejacket, shoes (including swim shoes), loose jewelry, or other item of clothing that may get caught in obstacles

20. Lifeguards are responsible for final determination of swim ability, age, and height according to the existing rules of the facility.

21. Intoxicated person are not allowed to use the system or operate the system

22. No spectators in the designated safety area of the pool

Section 2

End of Day Procedures

End of Day Washdown

•This procedure should be followed on a daily basis

• Rinse the Obstacle Frame including the Ropes, Plates, and other attachments with fresh water

Section 3

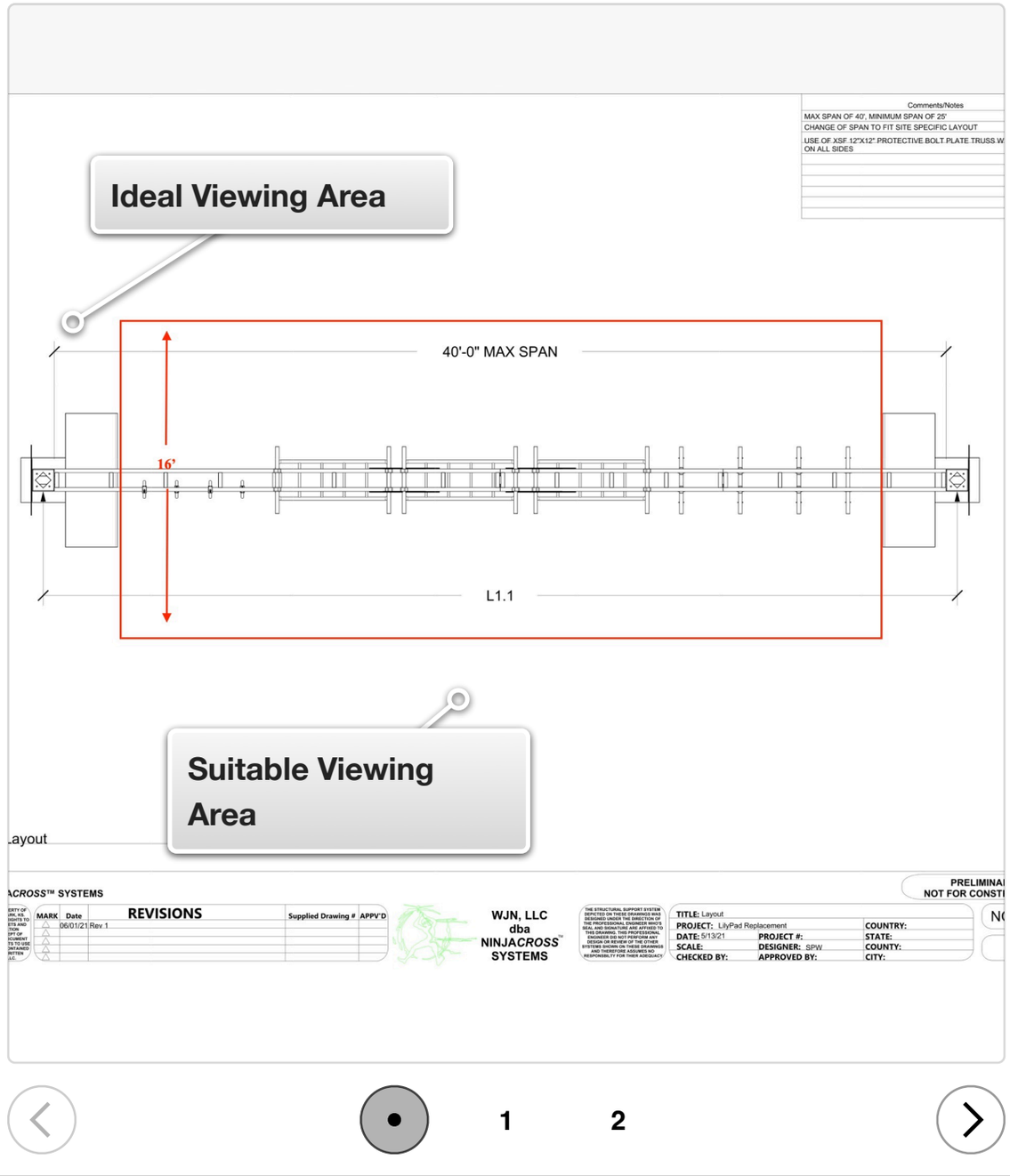
Designated Safety Area

he Designated Safety Area is the zone where only participants may be in the pool during the operating time of the NinjaCross MiniNinja System. The safety area is detailed as the area directly under the Obstacle Frame as well as an additional 8-feet on either side of the Obstacle Frame stretching from end of pool to end of pool.

During operations, spectators are prohibited from entering the Designated Safety Area.

Participants who quit the course without finishing shall be instructed to exit the course to the outside of the Designated Safety Area without crossing the path of other participants and exit the Designated Safety Area as quickly and safely as possible.

Interactive 3.1 Designated Safety Area



Section 4

Seasonal Shut Down Procedures

Long Term Shutdown

Procedures

When storing the Ninja*Cross* MiniNinja system for winter or long-term storage, the following steps should be taken.

1. Remove all obstacles and store in a secure safe location. Take care in storing obstacles as to not damage the materials or coating.

2. Store Ropes in a dry location free of any chemicals.

Section 1

Obstacle Types

There are two types of obstacles with the NinjaCross MiniNinja System a) OAB mounted obstacles, and b) Direct frame mounted obstacles.

OAB mounted obstacles are those obstacles that use 2 or more cables attached to the obstacle and require a spacing of more than 12” between the NetForm ropes. The OAB attaches to the Obstacle Frame by way of 2 Swivel Clamps. Obstacles attach to the OAB via the stud connection on the OAB and the shackles of the NetForm Rope.

Examples of OAB Mounted Obstacles are:

Trapeze Bars Low Bars Ladders

Direct frame mounted obstacles are those obstacles that attach to the Obstacle Frame directly by use of an Eye Clamp or other method.

Examples of Direct Mounted Obstacles are:

Sea of Discs Overhead Rings CannonBall Alley

Section 2

Obstacle Mounting Procedures

In order to mount any obstacle using a Swivel Clamp or Eye Clamp the following procedures need to be followed

1. Ensure that the Obstacle Frame is fully deployed in its operational position and the pool is clear of all swimmers.

2. Choose location for obstacle to be mounted.

3. Choose correct type of clamp for the obstacle to be installed

4. Unscrew the wing nut on the clamp to allow clamp to easily open

5. Place clamp in position, close the clamp over the Obstacle Frame tube, close bolt into clamp tab ensuring that the wing nut and washer clear the top of the clamp.

6. Tighten the wing nut until snug, do not over tighten as damage may occur to the Obstacle Frame truss

7. Attach obstacle to Eye Clamp or attach OAB to Swivel Clamps.

   a. If using an Eye Clamp, open the shackle at end of the NetForm Rope by turning the shackle pin counterclockwise using an Allen wrench. Place shackle over the open eye of the clamp and insert shackle pin into the shackle through the eye of the clamp. Tighten shackle pin (the use of blue Loctite will ensure shackle does not come loose.)

   b. If using an OAB, open the shackle at end of the NetForm Rope by turning the shackle pin counter-clockwise using an Allen wrench. Place shackle over the open stud of the OAB and insert shackle pin into the shackle through the stud of the OAB. Tighten shackle pin (the use of blue Loctite will ensure shackle does not come loose.)

When moving Obstacles from initial installed location, please refer to the Obstacle Water Depth Chart included in this manual to ensure obstacles are installed over the proper depth of pool.

Access to truss can be by use of a secured ladder in the pool leaned up against the Obstacle Frame or by use of the EZ Dock floating dock system. Care must be taken to not put excessive lateral force on the Obstacle Frame at any time, and at no time should staff sit, stand, or walk on the Obstacle Frame for access.

Obstacle Water Depth

Obstacle	Min Water Depth in Feet
Overhead Rings	4
Rising Rings	4
Cannonball Alley	5
Low Bar	4
Trapeze Bar	4
Ladder	4
Camelback	5

Section 3

Obstacle Frame

The Obstacle Frame is a 12”x12” aluminum box truss connected by way of Corner Blocks. The Obstacle Frame is the connection point for all Obstacles. The Obstacle Frame is designed to distribute the weight of the Obstacles and participants over a specified range according to the individual design of each system.

The Obstacle Frame is bolted together with 5/8”x2.5” Stainless Steel or Galvanized Bolts. The bolts utilize 5/8” washers and 5/8” nylon washers. The Nylon Washers prevent galvanic reactions from occurring on the different metal types of the bolts and Obstacle Frame.

12”x12” 6-way Corner Blocks are installed every at the vertical legs. All cross members of the Obstacle Frame are connected at Corner Blocks. Corner Blocks utilize the same 5/8” hardware as other parts of the Obstacle Frame.

Section 1

Obstacle Frame Maintenance

Cleaning

Powder coated aluminum should require little maintenance, other than to gently clean with water and a mild liquid detergent to remove any dirt or splashes. A microfiber cloth or sponge should be used to wipe over the surfaces of the furniture. Removing surface water with a drying cloth (like you would use on your car) will help avoid water spots. Avoid using any abrasive cleaning agents or materials, as these could mark the surface of the powder coat. Do not use steel wool or Scotch-Brite on powder coated surfaces.

Section 2

Obstacle Maintenance

Aluminum Obstacles

Cleaning

Powder coated aluminum should require little maintenance, other than to gently clean with water and a mild liquid detergent to remove any dirt or splashes. A microfiber cloth or sponge should be used to wipe over the surfaces of the furniture. Removing surface water with a drying cloth (like you would use on your car) will help avoid water spots. Avoid using any abrasive cleaning agents or materials, as these could mark the surface *of the powder coat. Do not use steel wool or Scotch-Brite on powder coated surfaces.*

Paint and Coatings Care

Over the course of use, the obstacles will receive chips and scraps on the coated surfaces. It is important that these chips and scraps be attended to as soon as they are discovered to prevent them from worsening. When a chip or scrap is discovered it is important to follow these procedures.

1. Remove obstacle from the water
2. Completely dry the obstacle and wipe clean any dirt or residues
3. Apply touch up paint to effected area
4. Allow paint to completely dry before allowing obstacle to get wet

Ropes

Cleaning

Rinse with clean fresh water, do not use chemicals or abrasives.

Section 3

Material Specific Maintenance

The following pages have information on the proper methods for cleaning specific types of metals found in the Ninja*Cross* MiniNinja System. If you have any questions, please contact Ninja*Cross* Systems for advise.

Care and Cleaning of Stainless Steel

Introduction

Cleanliness and stainless steel are closely related and, in many applications, each is dependent upon the other. In the handling of food, chemicals, pharmaceuticals and in the use of stainless steel as a construction material (roofs, wall panels, entry ways, signs, etc.), stainless steel provides the degree of corrosion resistance that is necessary to prevent product contamination or surface rusting. However, stainless steel performs best when clean — cleanliness is essential for maximum resistance to corrosion.

This handbook describes various practices for cleaning stainless steel during manufacture and in use. This includes methods for removing free-iron contamination on stainless steel surfaces that may have been picked up from metalworking tools; and for removing general accumulation of dirt, grime and surface stains that occur during normal handling and exposure to the elements.

The reader should keep in mind that there are few specific rules for a cleaning procedure. Accordingly, the methods discussed in this handbook are suggestions. Each manufacturer or user, after obtaining competent advice with respect to their individual requirements, should select methods appropriate to those requirements.

What is Stainless Steel?

Stainless steel is not a single alloy, but rather the name applies to a group of iron-based alloys containing a minimum 10.5% chromium. Other elements are added and the chromium content increased to improve the corrosion resistance and heat resisting properties, enhance mechanical properties, and/or improve fabricating characteristics. There are over 50 stainless steel grades that were originally recognized by the American Iron and Steel Institute (AISI). Three general classifications are used to identify stainless steel. They are:

1) Metallurgical structure.

2) The AISI numbering system (200, 300 and 400 series numbers).

3) The Unified Numbering System, which was developed by the American Society for Testing Materials (ASTM) and the Society of Automotive Engineers (SAE) to apply to all commercial metals and alloys.

The various types of stainless steel are detailed in a designer handbook, “Design Guidelines for the Selection and Use of Stainless Steel,” available from the Specialty Steel Industry of North America (SSINA). Several other publications are also available, including: “Stainless Steel Fabrication,” “Stainless Steel Fasteners,” “Stainless Steel Finishes,” “Stainless Steel Specifications,” and “Stainless Steel Architectural Facts,” to mention a few.

Alloy Types

304 is the basic chromium-nickel austenitic stainless steel and has been found suitable for a wide range of applications. It is the most readily available in a variety of product forms. This grade is easy to form and fabricate with excellent resistance to corrosion.

•304L is the low carbon version of 304. It is sometimes specified where extensive welding will be done.

•316 offers a more corrosion-resistance through the addition of molybdenum. This grade is desirable where the possibility of severe corrosion exists, such as heavy industrial atmospheres and marine environments.

•316L is the low carbon version of 316.

•430 is a straight chromium ferritic stainless steel with lower corrosion resistance than the 300 series. It is principally employed for interior use.

Cleaning of Stainless Steel

Stainless steels need to be cleaned for aesthetic considerations and to preserve corrosion resistance. Stainless steel is protected from corrosion by a thin layer of chromium oxide. Oxygen from the atmosphere combines with the chromium in the stainless steel to form this passive chromium oxide film that protects from further corrosion. Any contamination of the surface by dirt, or other material, hinders this passivation process and traps corrosive agents, reducing corrosion protection. Thus, some form of routine cleaning is necessary to preserve the appearance and integrity of the surface. Stainless steels are easily cleaned by many different methods. They actually thrive with frequent cleaning, and unlike some other materials, it is impossible to “wear out” stainless steel by excessive cleaning. The effect of surface/pattern roughness, grain/pattern orientation and designs that allow for maximum rain cleaning (exterior applications)should be considered.

Types of surface contaminants

• Dirt -Like any surface that is exposed to the environment, stainless steel can get dirty. Dirt and soil can consist of accumulated dust and a variety of contaminates that come from many sources, ranging from the wind to everyday use. These contaminates will vary greatly in their effect on appearance and corrosively and ease of removal. While some may be easily removed, others may require specific cleaners for effective removal. It may be necessary to identify the contaminate or experiment with various cleaners. Frequently, warm water with or without a gentle detergent is sufficient.

Next in order are mild non-scratching abrasive powders such as typical household cleaners. These can be used with warm water, bristle brushes, sponges, or clean cloths. Ordinary carbon steel brushes or steel wool should be avoided as they may leave particles embedded on the surface which can lead to RUSTING. For more aggressive cleaning, a small amount of vinegar can be added to the scouring powder. Cleaning should always be followed by rinsing in clean hot water. When water contains mineral solids, which leave water spots, it is advisable to wipe the surface completely with dry towels.

• Fingerprints and Stains -Fingerprints and mild stains resulting from normal use in consumer and architectural applications are the most common surface contaminates. Fortunately, these usually affect only appearance and seldom have an effect on corrosion resistance. They are easy to remove by a variety of simple cleaning methods. Fingerprints are probably the most troublesome marks to remove from the surface of smooth polished or bright finished stainless steel. Fortunately, they can be removed with a glass cleaner or by gentle rubbing with a paste of soda ash (sodium carbonate) and water applied with a soft rag. Once again, this should be followed by a thorough warm water rinse. There are several special surface finishes where fingerprints present special problems: polished No. 6, etched, some abrasive blasted finishes, and light electrochemical colors applied over satin or brushed finishes.

(NOTE: there are several special finishes designed to withstand fingerprints: embossed, swirl patterns, lined patterns, etc.).

• Shop oil and Grease -Shop oils, which may carry grease, grit and metal chips, commonly produce surface soiling after many shop operations. Greases and other contaminates may also soil surfaces in food preparation and many other household and commercial situations. These soils may be corrosive in themselves or may not allow the surface to maintain passivity, and so periodic removal is a necessity. Initially, soap or detergent and water may be tried or a combination of detergent and water plus a solvent. The removal of oil and grease from stainless steel parts by immersion in chemical solvents is frequently used with cold-formed or machined parts that are laden with lubricants. This process, in its simplest form, consists of bringing liquid solvent into contact with the surface to be cleaned and allowing dissolution to take place; for example, washing a surface with trichloroethylene or similar liquid or stirring a batch of small parts in a container of solvent. Non-halogenated solvents, such as acetone, methyl alcohol, ethyl alcohol, methyl ethyl ketone, benzene, isopropyl alcohol, toluene, mineral spirits, and turpentine work well.

Many of these solvents are widely used as individual cleaners, but there are thousands of blended or compound cleaners on the market. Users are advised to contact suppliers of solvents for information on their applications on stainless steel.

Types of Cleaners and Methods

General Precautions

In selecting cleaning practices, consider the possibility of scratching and the potential for post-cleaning corrosion caused by incompletely removed cleaners. Scratching can occur on a bright mirror finish by cleaners that contain hard abrasives, or even by "grit" in wash water. This is usually not a problem on dull finishes, or those surfaces finished with a coarse polishing grit. The best preventative measure is to avoid using abrasive cleaners unless absolutely necessary. When abrasives are needed, first experiment on an inconspicuous area. A "soft abrasive," such as pumice, should be used. Abrasives can permanently damage some colored and highly polished finishes. Advice should be obtained from the finish supplier when cleaning special finishes. Many cleaners contain corrosive ingredients which require thorough post-clean rinsing with clean water; however, thorough rinsing is recommended for all cleaning procedures.

• **Clean Water and Wipe** - The simplest, safest, and least costly method that will adequately do the job is always the best method. Stainless surfaces thrive with frequent cleaning because there is no surface coating to wear off stainless steels. A soft cloth and clean warm water should always be the first choice for mild stains and loose dirt and soils. A final rinse with clean water and a dry wipe will complete the process and eliminate the possibility of water stains.

• **Solvent Cleaning** -Organic solvents can be used to remove fresh fingerprints and oils and greases that have not had time to oxidize or decompose. The preferred solvent is one that does not contain chlorine, such as acetone, methyl alcohol, and mineral spirits. There are many compounded or blended organic cleaners that are commercially available and attempt to optimize both clean ability and safety attributes. Cleaning can be accomplished by immersing smaller articles directly into the solvent, wiping with solvent-impregnated cloths, or by sophisticated vapor or spray methods. The wiping technique sometimes leaves a streaked surface.

Effective Cleaning Methods

• **Household Cleaners -** Household cleaners fall into two categories: detergent (non-abrasive) and abrasive cleaners. Both are effective for many mild dirt, stain, and soil deposits, as well as light oils such as fingerprints. The abrasive cleaners are more effective but introduce the possibility of scratching the surface. However, the degree of abrasiveness will vary greatly with the particular product, and some brands will produce noticeable scratching on only the most highly polished and some colored surfaces. All of these cleaners vary widely with respect to their acidity and the amount of chloride they contain. A neutral cleaner low in chloride is preferred unless the user is assured that the surface can be thoroughly rinsed after cleaning. The fact that the label states “for stainless steel” is no guarantee that the product is not abrasive, not acidic, or low in chloride. The cleaning method generally employed with these cleaners is to apply them to the stainless surface and follow by cloth wiping, or to wipe directly with a cleaner-impregnated soft cloth. In all cases, the cleaned surface should be thoroughly rinsed with clean water and wiped dry with a soft cloth if water streaking is a consideration.

• **Commercial Cleaners -** Many commercial cleaners compounded from phosphates, synthetic detergents, and alkalis are available for the cleaning of severely soiled or stained stainless surfaces. When used with a variety of cleaning methods, these cleaners can safely provide effective cleaning. Manufacturers should be consulted and their recommendations followed whenever using cleaners of this kind. The general precautions stated above also pertain to these cleaners.

Care of Stainless Steel

The cleaner stainless steel can be kept while in storage, being processed or during use, the greater the assurance of optimum corrosion resistance. Some tips on the care of stainless steel are listed below:

1) Use paper or other protective wrapping on the surface of the stainless steel until processing is complete.*

2) Handle stainless steel with clean gloves or cloths to guard against stains or finger marks.

3) Avoid the use of oily rags or greasy cloths when wiping the surface.

4) Do routine cleaning of exposed surfaces. Buildings with window washing systems can utilize this method to clean exterior panels.

5) Where possible, after cleaning, rinse thoroughly with water.

6) Cleaning with chloride-containing detergents must be avoided.

7) Even the finest cleaning powders can scratch or burnish a mill-rolled finish. On polished finishes, rubbing or wiping should be done in the direction of the polish lines, NOT across them.

8) **DO NOT USE SOLVENTS** in closed spaces or while smoking.

*Many adhesive-backed papers and plastic sheets or tape applied to stainless steel for protection “age” in fairly short periods of time and become extremely difficult to remove.

Manufacturers should be contacted regarding information as to how long protective films or paper can be left in place.

Acknowledgments

The Specialty Steel Industry of North America (SSINA) acknowledges that this new handbook contains information originally published by the Committee of Stainless Steel Producers, American Iron and Steel Institute, which no longer exists. Current SSINA member companies were represented on that committee. The SSINA wishes to acknowledge the contributions of the Nickel Development Institute and its consultant, Technical Marketing Resources (Pittsburgh, PA) for help in preparing the contents of this handbook.

Powder Coating Care and Maintenance

Proper Care of Powdered Surfaces Is Essential

Powder coatings that are applied to metal products exposed to the weather will inevitably degrade over time. A number of conditions, including those found in nature, will contribute to shortening the life of this type of protective finish.

•Sun

•Rain

•Wind

•Pollution

•Cold weather

•Salt water

•Electrical current

•Dissimilar metals

How to Maintain Powder Coated Surfaces

1. Avoid harsh chemicals: Unlike spray paint, powder coating is much more resistant to things like rust, corrosion, peeling and fading. However, that resistance does not mean it's completely fine to use chemical cleaners and solvents to clean powder coated items. Harsh cleaners and solvents like acetone can actually damage powder coating.

2. Clean gently: You can still clean powder coated surfaces. Just wipe off dust with a soft clock. If more cleaning is necessary, use a highly diluted, mild soap in water and a soft towel or soft sponge to very gently clean. Rinse with a little water, then dry with another soft towel.

3. Wax: If your powder coated metal has lost its gloss and shine, after removing dirt with mild soap, you can apply a thin layer of wax just like you do after you wash your car. After the wax dries, wipe all of it off and powder coated metal will look like new.

4. Don't paint: If you're wondering if you can touch up imperfections and rust with paint, the answer is no. Because of how the powder coating process works, paint won't adhere to powder coated surfaces. If your powder coating is starting to show signs of wear and tear, it's time to have a professional either repair or redo the powder coating.

5. Maintenance schedules: We recommend you regularly inspect and clean your powder coated items. How often you wipe your metal surfaces clean depends on the amount of dirt and grime in the area, the time of year, and if there's been any intense weather like a hurricanes or tornados.

NetForm Ropes

System Inspection

NetForm structures and associated hardware including backing nets, cables and fasteners should be inspected by a competent person after installation and on a regular scheduled basis thereafter. It is good practice to keep a dated and signed maintenance log of each netting system to assure that all safety measures have been followed.

The system must be inspected following alterations, repairs and impact loading. If any welding or cutting operations occur near the structures, weld protection must be provided for that area, and more frequent inspections should be conducted in proportion to the dangers involved.

NetForm should be inspected on a daily and weekly basis.

• Daily Inspection should include a quick visual of the NetForm and any backing netting, to look for any obvious broken net mesh or frays. Report for replacement any missing NetForm cross joints or tees.

• Weekly Inspection should include any lashing cord that may be used in the NetForm system, including loose and broken lashes. Repair as necessary. Visually check and hand-test all rope handrails, hardware, cables, anchors, etc. All hardware should be in place with no substitutes. Document any faults with a photograph to help expedite repairs.

General Environmental Inspection

NetForm, backing nets or hardware that show deterioration from mildew, corrosion, wear, or stress, that may affect their strength, must be immediately removed from service for further inspection, repair or disposal.

• Inspect the NetForm and backing nets for cuts, pulls, fraying of material and discoloration indicating material aging.

• Inspect cross joints and tees for stress cracking.

• Inspect support cables for cuts, twists, kinks, fraying of strands and corrosive rust.

• Inspect support and anchor hardware to assure fasteners are properly secured and that no pieces are missing. Look for damaging rust that may affect hardware strength or abrade the NetForm or backing nets.

Repairs

Field repairs and modifications may be done with guidance and materials from the manufacturer. Photographs are always the best way to convey the extent of a fault area. If replacement of a net panel or system is required, the manufacturer will determine the best method of replacement.

ABS Wrap/Signage Care

• Clean debris from wraps and signage as they appear dirty. Failure to remove debris may make care more difficult over time.

• Test any cleaning solutions on a small section of wrap before using to clean wrap.

• Use a wet, non-abrasive detergent and a soft clean rag for cleaning.

• Rinse thoroughly with clean water. Dry with a microfiber cloth.

• If choosing to wax the wrap, use only waxes that do not contain petroleum distillates

• Do not use mechanical brushes or pressure washers to clean the wraps. Doing so may damage the graphics or wraps themselves.

Vertical Truss Leg Wraps are not included in base MiniNinja System. NinjaCross Systems suggests the use of wraps to prevent access to the Obstacle Frame.

Section 1

Daily Pre-use Inspections

Prior to use each day, the system must undergo a complete Pre-use Daily Inspection to ensure that the system components are in proper working order and ready for use. This is a comprehensive inspection that is done at start of each day.

The complete system SHALL undergo the following inspections as laid out and documented. Any problems, concerns, or points of interests SHALL be noted in the inspection logs for review by NinjaCross Systems.

1. Ensure that the Obstacle Frame Legs are secured to the mounting plates.

2. Ensure Obstacle Frame is secure and not damaged.

3. Ensure that all Obstacles are in proper placement and not entangled in the Obstacle Frame, OAB's, or Signage.

4. Check the pool and surrounding deck for parts, hardware, or materials that may have fallen.

5. Ensure all Obstacles are at their proper depth in the pool and are located as designed.

6. Inspect NetForm Ropes for damage, broken strands, or opening or fraying. Check for mildew or staining.

7. Have lifeguards run through both lanes to ensure system is operating correctly.

8. Ensure that all signage is undamaged, visible without obstructions, and can be viewed by participants on the deck.

9. Document inspection and note any concerns or problems.

Section 2

Quarterly Inspection

The complete system **SHALL** undergo the following quarterly inspections as laid out and documented. Any problems, concerns, or points of interests **SHALL** be noted in the inspection logs for review by NinjaCross Systems.

1. Obstacle Frame -

a. Check that Obstacle Frame joints, where two Truss Sections meet or a Truss Section and Corner Block meet, are secure and not loose.

b. Ensure that all hardware is present at every joint, each Truss Section is bolted to a Truss Section or Corner Block with 4 bolt assemblies.

c. Check for chipped paint

d. Checked for cracked paint, cracked paint may indicate a stress fracture in the truss cord.

e. Ensure that the Obstacle Frame is level both side to side and front to back

f. Rinse frame with fresh water

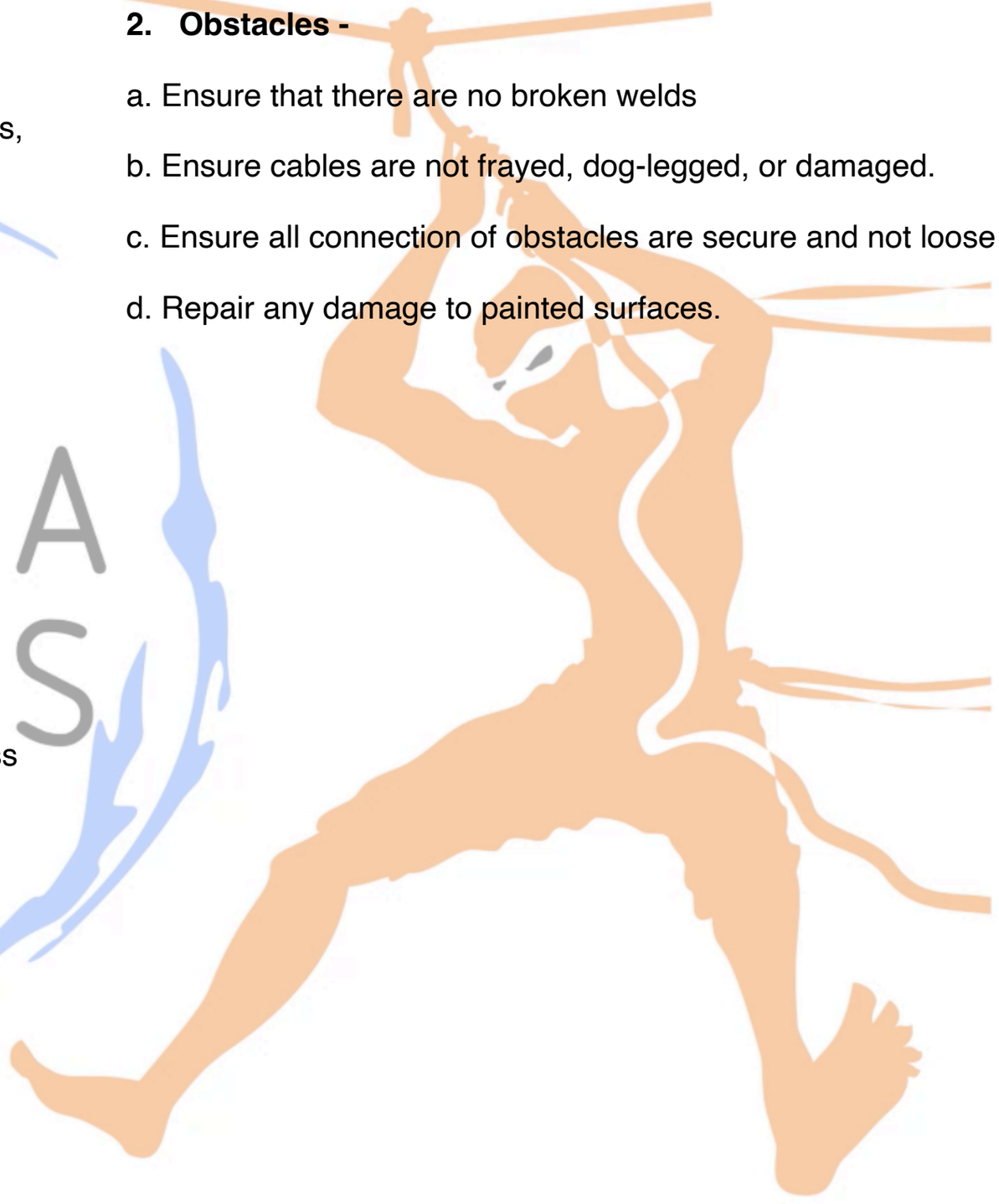
2. Obstacles -

a. Ensure that there are no broken welds

b. Ensure cables are not frayed, dog-legged, or damaged.

c. Ensure all connection of obstacles are secure and not loose

d. Repair any damage to painted surfaces.



Section 3

Yearly Inspection

All NinjaCross MiniNinja System components **SHALL** be inspected annually by NinjaCross Systems or an authorized representative. Failure to have the system inspected will result in NinjaCross Systems notifying all relevant inspection authorities that the system cannot be declared safe to use by manufacturer.

A minimum of 4-weeks' notice to NinjaCross Systems must be given for scheduling the annual inspection. Contact NinjaCross Systems via your sales contact or directly at Support@NinjaCrossSystems.com

Annual Inspection **SHALL** include and inspection of the following items to ensure the safe and proper working order of the NinjaCross MiniNinja System.

1. Obstacle Frame System including mounting plate
2. Obstacles
3. Inspection and Maintenance Logs

Section 4

Inspection Forms

Ninja*Cross* Systems has provided the following sample inspection forms for use or as a guideline to creating your own inspection forms. At minimum, all inspection forms must include the items including in each form.

Inspection Forms

Ninja*Cross* **:** Daily Pre-use Inspection

Description **:** Daily safety checks to be performed prior to use.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	Monthly
Obstacle Frame Mounting Plate																																
Obstacle Frame																																
Obstacle Ropes																																
Obstacles																																

Notes **:**

Inspection Forms

Ninja*Cross* **:** Quarterly Inspection

Description **:** System inspection every 3 months

	1st Quarter	2nd Quarter	3rd Quarter	4th Quarter
Obstacle Mounting Plate and Anchors				
Obstacle Frame Connections				
Obstacle Connections				
Obstacle Rope integrity				
Obstacle integrity				

Notes **:**

Certification and Training

Detailed instructions, extensive training, and actual operations specific to each NinjaCross MiniNinja System will be provided and addressed during the on-site Training and Certification conducted by The Owner/Operator of the MiniNinja System. The Owner/Operator will be responsible for instructing the operations team on Operations, Daily, Quarterly, Seasonal and Annual Inspections, Safe Usage, Procedures to follow in order to minimize possible injuries as well as cleaning and maintenance to

insure the longevity of your obstacle course & fitness system.

Personnel Training

(Please Note the Following Contains the Manufactures Minimum Recommendations but are Subject to Your Facilities Local and State Codes as well as contracted Third Party Organizations such as the American Red Cross)

Having properly trained and conscientious employees on site is the most important safety factor in the operation of the NinjaCross MiniNinja System.

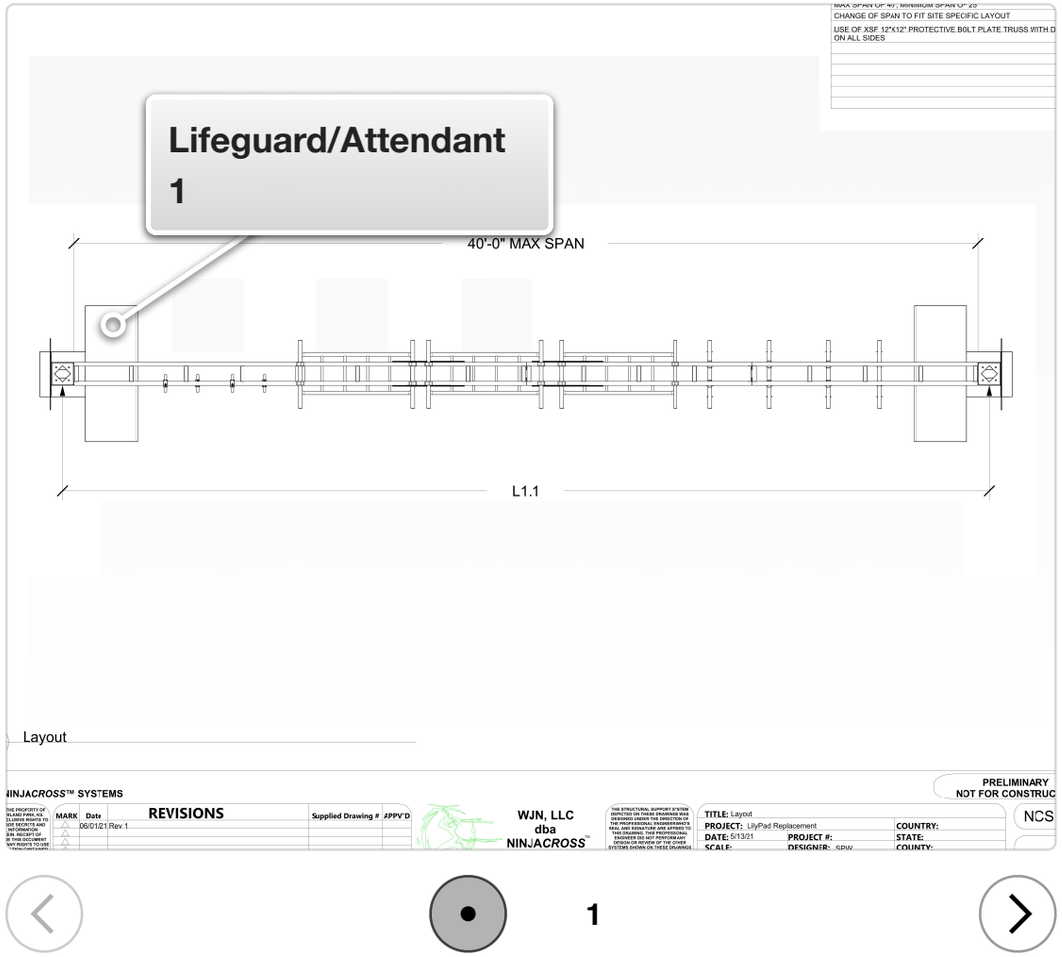
It is our recommendation that all employees who are responsible for the NinjaCross MiniNinja System operations be certified lifeguards and be qualified in both first-aid and life-saving techniques through the American Red Cross training or the equivalent. At least one person who has completed the Standard First Aid and Personal Safety course, as offered by the American Red Cross, or the equivalent should be on duty always during operating hours. This person should also be competent in carrying out any emergency procedures peculiar to the slide he or she is operating. Under most conditions, this is also a recommendation of the insurance carrier if applicable.

Each owner/operator shall have written operating procedures for the NinjaCross MiniNinja System, which are an integral part of their staff-training program. These procedures shall include but not be limited to:

Lifeguard/Attendant Station 1 - one trained lifeguard/attendant SHALL be stationed at the edge of the pool at the starting location. This staff duties are to ensure that all Participants start in the water, to ensure the proper spacing of Participants at the start, and to observe Participants at the start of the course.

All NinjaCross MiniNinja personnel should be alert to controlling crowd behavior and the proper entry rate into the pool; therefore, we recommend the line to participate be formed on the pool deck rather than the pool edge. One Participant may be stationed at the edge of pool to start the course, while any additional may be at a point away from the pool edge preparing to move into starting position at the command of the lifeguard/attendant. Once the Participant who is at edge of pool starts the course the Participant

Interactive 7.1 Lifeguard/Attendant locations



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on the deck enters the starting area at the edge of the pool then the line then moves up one position.

Lifeguards at the start of the course should address each and every Participant when it is their turn and then inform the Participant on the rules of the course prior to starting the course. All Participants should be instructed how to use the course and not allowed to run, jump, or leap into the pool. The Lifeguard(s) stationed at start will address each Participant first by asking that they follow their instructions and Do Not proceed into the pool until they are given the okay to do so.

Safe and orderly exit from the pool area helps reduce the risk of disoriented riders colliding with other pool guests. Lifeguards shall instruct Participants to exit the Designated Safety Area in the correct manner and direction.

An uninterrupted view of the pool and Obstacle Frame must be maintained at all times. It is recommended that all lifeguards be familiar with all the jobs related to the Obstacle Frame. Rotating lifeguards between positions keeps interest and attention high.

Section 2

Facility Requirements

Communications

Each facility shall ensure they have a communication plan in place for all staff working the NinjaCross MiniNinja System and have trained them in the proper use of signals, devices, or other methods.

Signage:

The owner/operator shall place signage as specified. These signs shall include safety, warning, and instructional signage reflecting manufacturer recommendations. Signage shall be prominently displayed at the course entrance or other appropriate area and shall include but not be limited to:

- **Instructions, which include:**
  - Expected participant conduct,
  - Dispatch procedures,
  - Exiting procedures, and
  - Obey attendant/lifeguard instructions.
- **Warnings, which include:**
  - NinjaCross MiniNinja characteristics, such as challenging & competitive
  - Water depth if not posted near pool edge already
- **Requirements which include:**
  - Participants being free of medical conditions, including but not limited to pregnancy and heart, back, or musculoskeletal problems,
  - Mental conditions that may prevent comprehension or adherence to posted rules,
  - Maximum/minimum height and weight, and
  - Any swimming or physical ability requirement or both.

Section 1

System Overview

Your Ninja*Cross* MiniNinja System is an indoor or outdoor system that includes the deck mounted anchor points and mounts. This section will give an overview of the different materials that make up the components of the system.

Stainless Steel Components

1. Bolting Hardware
2. Shackles

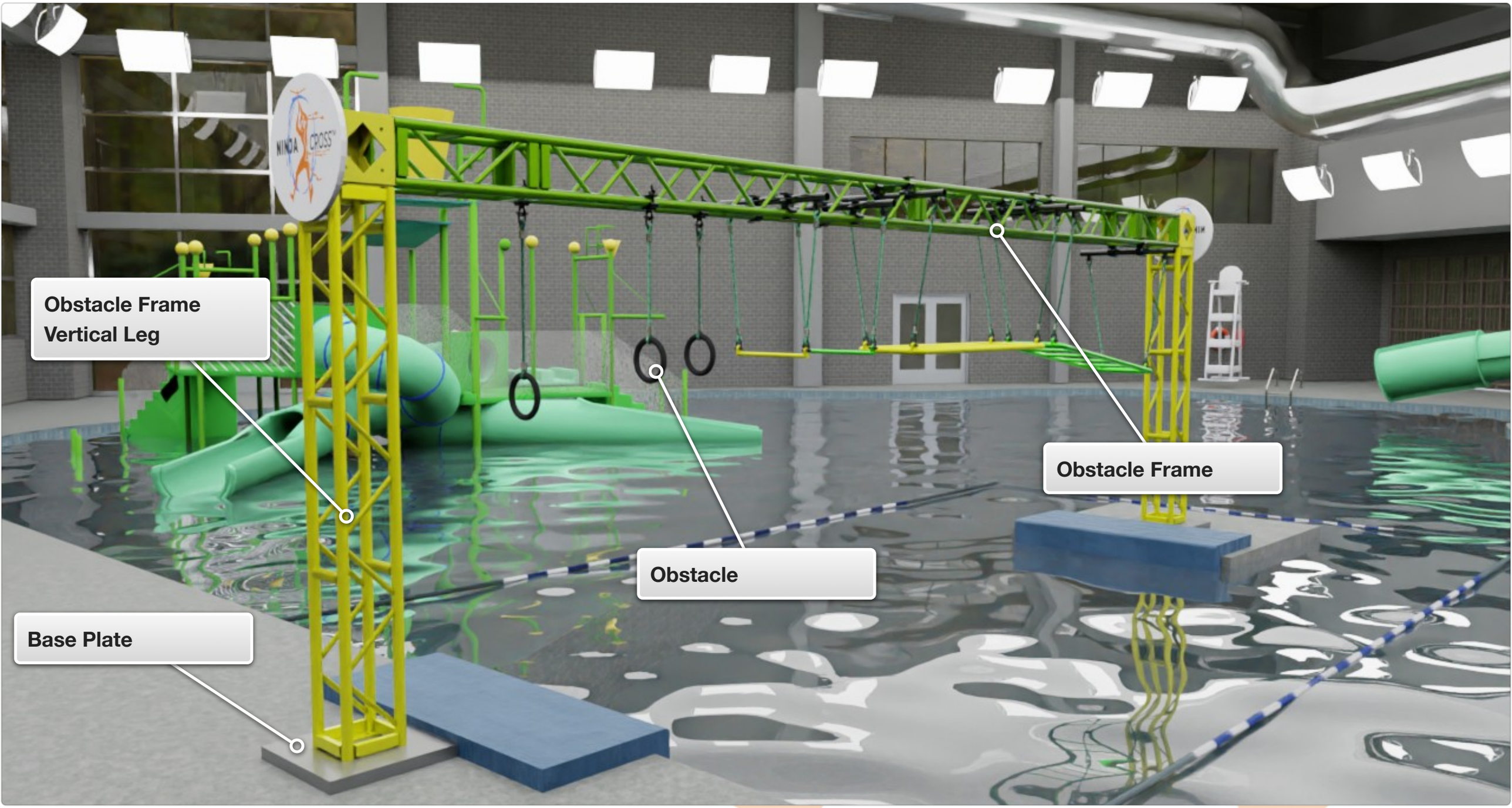
Aluminum Components

1. All metal Obstacles and OAB's
2. Obstacle Frame Truss and Corner Blocks
3. Truss Picks and Clamps

Other Materials

1. Signs - ABS
2. Backup System - powder-coated steel with galvanized cable
3. Ropes - InCord NetForm, Polyester Fiber Braided Steel Wire
4. Discs, Rings, and other Obstacles - HDPE

Interactive 8.1 System Overview



1 2 3 4

Section 2

Obstacle Frame Components

The Obstacle Frame consists of 3 primary components

1. 12”x12” Box Truss

2. 12” 6-way Corner Blocks

3. Mounting Plate

The parts of the Obstacle Frame System include:

1. **12”x12” Box Truss -** this aluminum box truss comprises the main structural component of the Obstacle Frame. Each section is at maximum 10’ long with the shortest being 2’ long. The type of Box Truss used is a bolt plate type that utilizes 5/8” bolt hardware.

2. **12”x12” 6-Way Corner Block -**is a 12” square block used to connect sections of Box Truss. The block is the only point where Static Lines are permitted to be installed.

3. **Mounting Plate -** this is a square aluminum plate designed to allow anchorage of the MiniNinja system to the concrete deck. The Mounting Plate is secured to the deck via wedge anchors and secured to the vertical Box Truss legs via bolting hardware.

Gallery 8.1 Obstacle Truss System

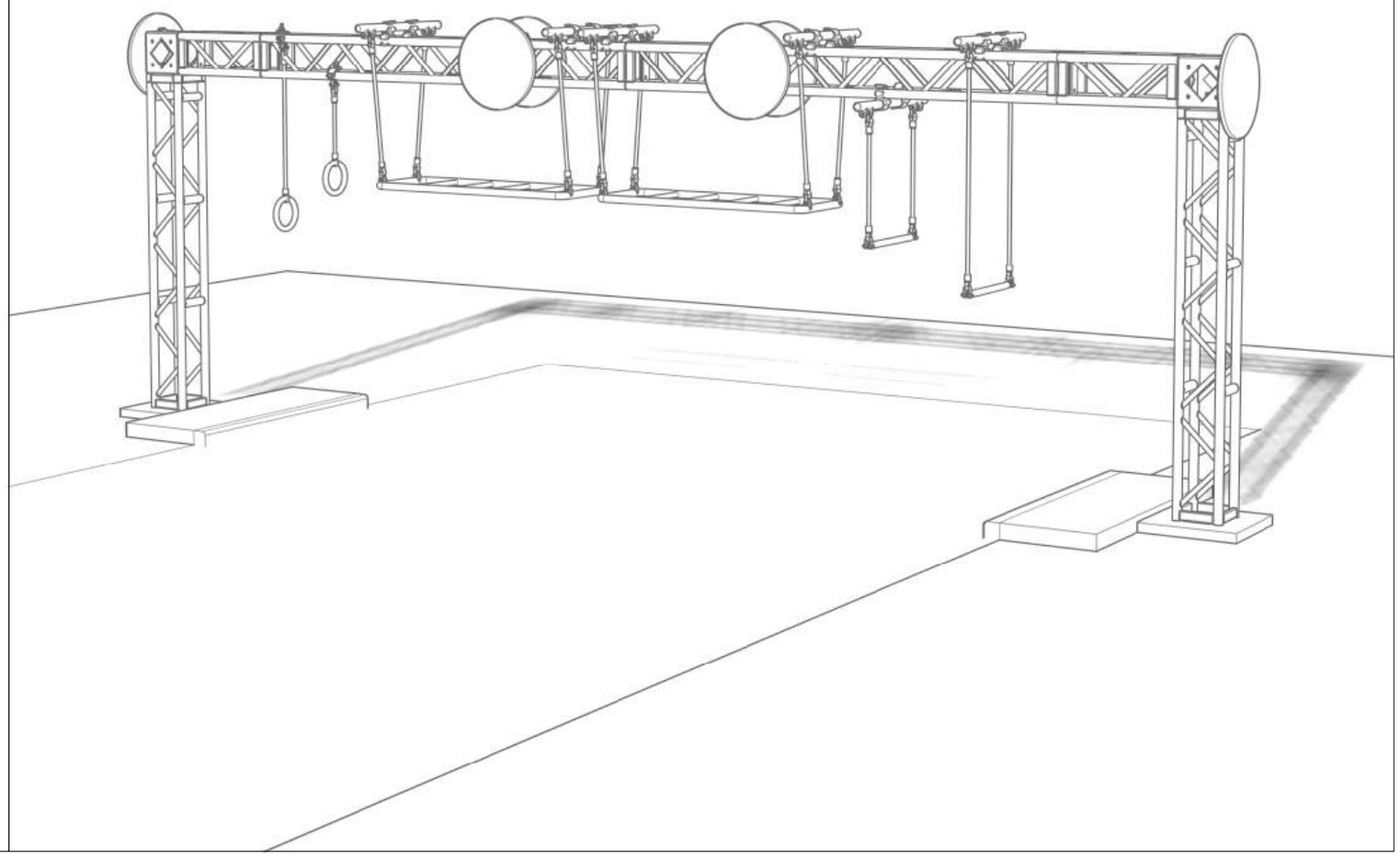
Corner Block

BASE PLATE AND ANCHOR NOTES:

FOR SLABS BETWEEN 4" AND 6" THICK:
INSTALL CUSTOM XSF BASE PLATE WITH (4) 5/8"Ø THREADED ROD ANCHORS IN AN 18" SQUARE PATTERN. EMBED 2¾" USING HILTI HIT-RE 500 V3 ADHESIVE.

FOR SLABS 6" THICK OR GREATER:
ANCHOR TRUSS DIRECTLY DOWN TO SLAB WITH (4) 5/8"Ø THREADED RODS AND HILTI HIT-RE 500 V3 ADHESIVE. USE A MINIMUM 4½" EMBEDMENT.

CONCRETE COMPRESSION STRENGTH SHALL BE 4000 PSI OR GREATER IN ALL CASES.



PRELIMINARY
NOT FOR CONSTRUCTION

MARK	Date	REVISIONS	Supplied Drawing #	APPV'D
1	06/01/21	Rev 1		
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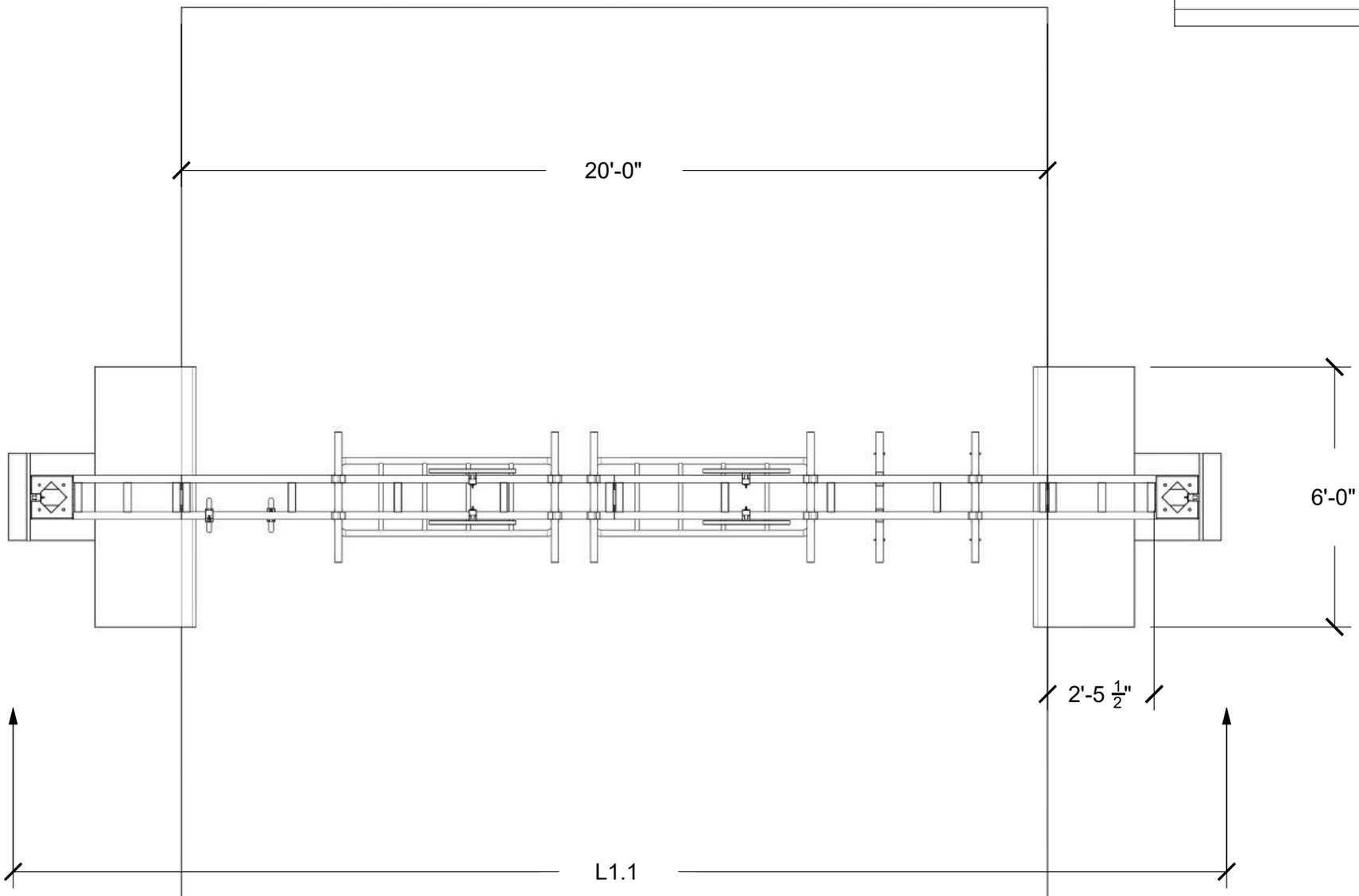
WJN, LLC
dba
NINJA*CROSS*™
SYSTEMS

TITLE: Cover Sheet		
PROJECT: LilyPad Replacement		**COUNTRY:**
DATE: 5/13/21	**PROJECT #:**	**STATE:**
SCALE:	**DESIGNER:** SPW	**COUNTY:**
CHECKED BY:	**APPROVED BY:**	**CITY:**

NCS CS

Comments/Notes
Make notes and comments here for review or to specify items



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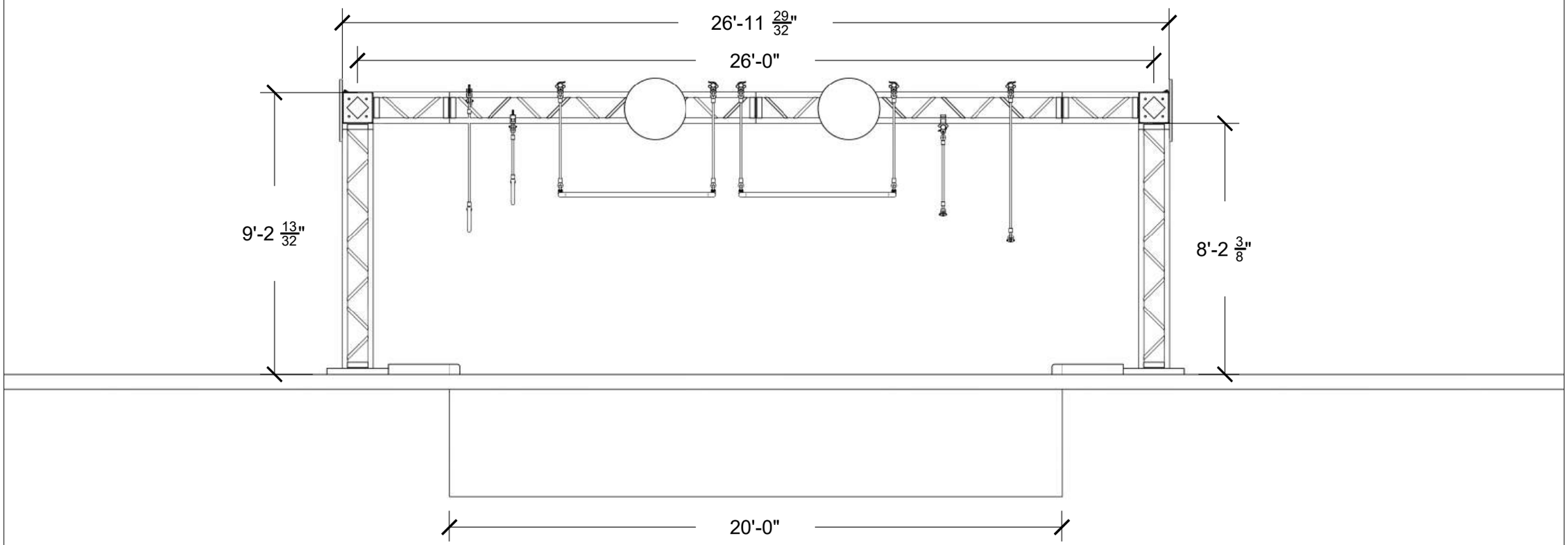
TITLE: Layout		
PROJECT: LilyPad Replacement		**COUNTRY:**
DATE: 5/13/21	**PROJECT #:**	**STATE:**
SCALE:	**DESIGNER:** SPW	**COUNTY:**
CHECKED BY:	**APPROVED BY:**	**CITY:**

NCS L1.0

Comments/Notes
Make notes and comments here for review or to specify items



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MARK	Date	REVISIONS	Supplied Drawing #	APPV'D
1	06/01/21	Rev 1		
2				
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SYSTEMS

THE STRUCTURAL SUPPORT SYSTEM DEPICTED ON THESE DRAWINGS WAS DESIGNED UNDER THE DIRECTION OF THE PROFESSIONAL ENGINEER WHO'S SEAL AND SIGNATURE ARE AFFIXED TO THIS DRAWING. THIS PROFESSIONAL ENGINEER DID NOT PERFORM ANY DESIGN OR REVIEW OF THE OTHER SYSTEMS SHOWN ON THESE DRAWINGS AND THEREFORE ASSUMES NO RESPONSIBILTY FOR THER ADEQUACY.

TITLE: Layout		
PROJECT: LilyPad Replacement		**COUNTRY:**
DATE: 5/13/21	**PROJECT #:**	**STATE:**
SCALE:	**DESIGNER:** SPW	**COUNTY:**
CHECKED BY:	**APPROVED BY:**	**CITY:**

NCS L1.1

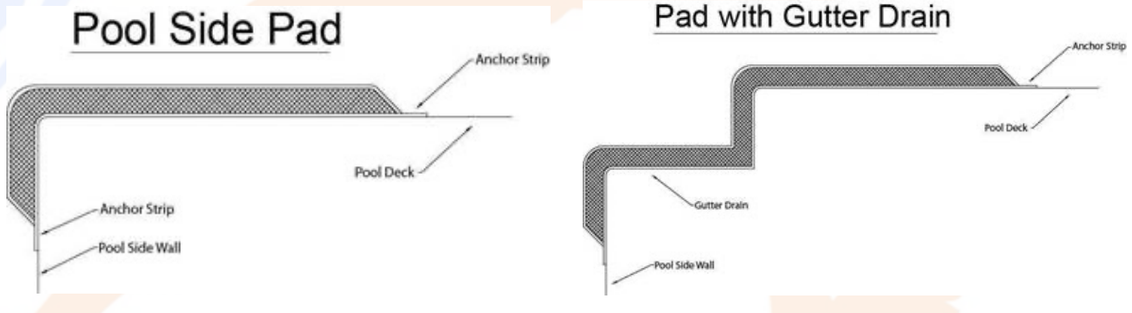
Disclaimers and Important Manufacturer Information

- **The NinjaCross MiniNinja System & ancillary components require installation by qualified personnel. Use of non-qualified trades' people or use of non-approved parts will void the manufacturer's Warranty.**

- **NinjaCross MiniNinja maintenance is the responsibility of the owner. It is recommended a maintenance log be kept documenting water quality including all performed maintenance. See suggested inspection check lists, water quality log, and maintenance section for guidelines on how to maintain the system, in addition to keeping your Warranty valid. These documents may be called on if warranty issues arise.**

- **When receiving manufacturer shipments, inspect all items for damage and quantity immediately. Failure to do so could result in costly repair or replacement costs at the expense of the owner/installer. When receiving any shipments, be sure to inform the driver of any discrepancies and report as indicated on the shipping documentation when signing for receipt of goods. All claims must be reported within 48 hours of receipt of goods. Claims reported outside of this time cannot be guaranteed. If nothing has been noted on the Bill of Lading a claim may not be accepted. If you are unable to inspect the shipment at time of receipt you must note on the Bill of Lading "Subject to inspection".**

- **NinjaCross Systems does not supply the Safety Padding. Safety Padding is the sole responsibility of the Owner/ Operator. Pool Side Pads are designed to be placed on the side of the pool to protect patrons as they enter and exit the MiniNinja area. Pads typically form an L-Shape covering the length of your area and protect the top walk area, the pool side wall and the pool edge. Pads can also be made in a "stair-step" shape to protect pool walls with drain gutters.**



WASHINGTON STATE BOARD OF HEALTH

Date: August 8, 2024

To: Washington State Board of Health Members

From: Kate Dean, Board Member

Subject: Rulemaking Petition – WAC 246-272A-0240, Holding Tank Sewage Systems – On-Site Sewage

Background and Summary:

On June 16, 2024, the Washington State Board of Health (Board) received a petition for rulemaking related to WAC 246-272A-0240, Holding Tank Sewage Systems. A holding tank sewage system is an on-site sewage system which 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.

The petition requests that the rule be changed to allow for external storage of septic waste in approved containers with a valid pumping contract. The petition states that this change is needed because too many people dump septic waste on the ground, and that the sanicans (portable toilets), which are allowed, are unsafe and inconvenient.

The rule prohibits a person from installing or using holding tank sewage systems for residential development or expansion of residences, whether seasonal or year-round, except under limited circumstances. The local health officer may approve the installation of holding tank sewage systems only for the following:

- Permanent uses limited to controlled, part-time commercial usage situations.
- Interim uses limited to handling of emergency situations (e.g., floods).
- For repairs as permitted under WAC 246-272A-0280.

The local health officer may grant a waiver from the above regulations under WAC 246-272A-0240 consistent with Department of Health (Department) guidance. The Department's guidance allows the regulation to be waived to allow a holding tank used for other than part-time, non-residential use, if it meets certain minimum criteria.

The minimum criteria established in the Department's guidance is important to maintain public health and safety when holding tank systems are used. A local health officer is not required to grant these waivers, and the use of waivers varies by county. The variability in county use of waivers may be due to local capacity to pump the tanks, capacity to enforce the waivers, and other reasons.

(continued on the next page)

Possible Board Motions:

The Board may wish to consider and amend, if necessary, one of the following motions:

The Board declines the petition for rulemaking to amend WAC 246-272A-0240- 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 246-272A-0240 to consider additional language related to holding tank sewage systems. 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

Shay Bauman
Policy Advisor

From: alyssaroyse@gmail.com <alyssaroyse@gmail.com>
Sent: Sunday, June 16, 2024 7:45 AM
To: Thompson, Tami (DOH) <tami.thompson@doh.wa.gov>
Subject: Petition for Rule Change

External Email

Hi Tami –

I've been engaged in marginally productive conversations with the Jefferson County Board of Health, trying to address illegal dumping of septic waste, for months now.

As a part time resident of Brinnon Washington, and a member of our HOA board for the Olympic Canal Tracts, I've talked to hundreds of people about the difficulty they have dealing with septic waste. Part of the problem – well, most of the problem – is that there is no good way to do it. There is, as you probably know, no sewage system in these beautiful rural areas. Most of them can't have septic systems because they are either too small, or too close to environmentally sensitive areas. Pumping small RV tanks weekly is cost-prohibitive. Sanicans are not a reasonable option because they are hard to keep clean and comfortable, especially if you're dealing with the weather that makes our area so beautiful. Not to mention that sanicans are hard to secure, and many of us don't feel safe having an open sanican on our property. As such, it's easier to simply dump waste on the ground, and that's what's happening. You can look at bacterial loads in local waterways for all the evidence you need that this is happening.

Nearly everyone I have talked to in Brinnon has said that the option to have an approved 200-500 external storage tank would be life-changing. (And I would personally work tirelessly to create a fund for subsidized pumping for those living in poverty, they deserve dignity and sanitation too!) There are many of those that simply slide under an RV, for safe storage, and would enable people to use their RVs – whether full time or as vacation getaways – in a way that is safe for both people and planet.

The Jefferson County BOH insists that state regulations do not allow this. Though I know many counties in Washington do, so I am not sure where the problem is.

In any event, they suggested that filing a petition for a rule change will require the state to address this within 60 days, so I'm hoping that – after months of only marginally productive conversation with JeffCo BOH – is true. This is such a serious issue, and there is such a simple solution that is already readily available. Even if it's only temporary, while we work to figure out how to serve rural communities indefinitely.

Please help!

I'm attaching the form that JeffCo BOH sent me. On the form it says to send to the appropriate state rules committee, and they offered me no guidance as to who that is. I'm hoping it's you, but if it's not, that you'll help me get to the right place.

I'm desperate to help both the people in my community and protect the natural beauty of it that I love so much. Thank you.

___

Alyssa Royse
(She / Her. Not into hugs.)
* Rocket Community Fitness
* Rocket on Instagram
* Speaking and Consulting

PETITION FOR ADOPTION, AMENDMENT, OR REPEAL OF A STATE ADMINISTRATIVE RULE

Print Form

In accordance with RCW 34.05.330, the Office of Financial Management (OFM) created this form for individuals or groups who wish to petition a state agency or institution of higher education to adopt, amend, or repeal an administrative rule. You may use this form to submit your request. You also may contact agencies using other formats, such as a letter or email.

The agency or institution will give full consideration to your petition and will respond to you within 60 days of receiving your petition. For more information on the rule petition process, see Chapter 82-05 of the Washington Administrative Code (WAC) at http://apps.leg.wa.gov/wac/default.aspx?cite=82-05.

CONTACT INFORMATION *(please type or print)*

Petitioner's Name: Alyssa Royse

Name of Organization: Part time Resident of the Olympic Canal Tracts in Brinnon WA

Mailing Address: 5315 55th Ave. S

City: Seattle State: WA Zip Code: 98118

Telephone: 206-931-9541 Email: alyssaroyse@gmail.com

COMPLETING AND SENDING PETITION FORM

- Check all of the boxes that apply.
- Provide relevant examples.
- Include suggested language for a rule, if possible.
- Attach additional pages, if needed.
- Send your petition to the agency with authority to adopt or administer the rule. Here is a list of agencies and their rules coordinators: http://www.leg.wa.gov/CodeReviser/Documents/RClist.htm.

INFORMATION ON RULE PETITION

Agency responsible for adopting or administering the rule: WA State / Jefferson County Board of Health

☐ **1. NEW RULE - I am requesting the agency to adopt a new rule.**

☐ The subject (or purpose) of this rule is: Allow secure external storage of septic waste that can be pumped by certified pumping companies.

☐ The rule is needed because: Currently, it is difficult and cost prohibitive for people without septic systems to manage their waste. This results in dumping directly on the ground. This is impacting both ground water and local waterways.

☐ The new rule would affect the following people or groups: This would be life changing for people who live in RVs and off-grid, as well as for people who have stationary RVs on vacation property that they use recreationally.

☐ **2. AMEND RULE - I am requesting the agency to change an existing rule.**

List rule number (WAC), if known: I don't know, but Jefferson County BOH said it's a state issue, so I'm trying.

☐ I am requesting the following change: Allow for external (above or in ground) storage of septic waste in APPROVED containers of at least 200 gallons, with a valid pumping contract. This is legal with sanicans, but those involve going outdoors, are unsafe and inconvenient.

☐ This change is needed because: Too many people simply dump septic waste on the ground because safe and sanitary solutions aren't allowed, and they don't want sanicans.

☐ The effect of this rule change will be: A HUGE reduction in people dumping septic waste directly on the ground.

☐ The rule is not clearly or simply stated: The rule is inconsistent. Mason county allows external storage, Jefferson does not. We need this in order to reduce illegal dumping, and allow more people to use their property in a safe and sanitary way.

In the Olympic Canal Tracts the lots are FAR too small for septic, and sanicans are inconvenient. So people dump. Allowing large storage, under RVs, and such, would be life-changing. People want this.

☐ **3. REPEAL RULE - I am requesting the agency to eliminate an existing rule.**

List rule number (WAC), if known: Allowing for external storage of septic waste, in approved containers, would reduce the amount of septic waste that is dumped directly on the ground. For people who use RVs either as permanent residents due to poverty, or recreationally on their own land, the small storage isn't enough. But frequent pumping is cost prohibitive.

(Check one or more boxes)

☐ It does not do what it was intended to do.

☐ It is no longer needed because: Sanicans are an option for some people. But for many, they aren't. Sanicans are harder to keep clean and secure, not to mention they often involve going out in the weather. They can also be tipped over by vandals, which makes everything worse.

☐ It imposes unreasonable costs: Meanwhile, frequent pumping of small RV tanks is expensive.

☐ The agency has no authority to make this rule: Allowing people the clean, safe, dignity of large external storage for waste, with a valid pumping contract (as is the current sanican regulation) would encourage safe septic practices. It would be safer, simpler, more convenient and feasible for many people.

☐ It is applied differently to public and private parties: If Sanicans are allowed, there is no good reason for even safer external storage not to also be allowed.

☐ It conflicts with another federal, state, or local law or rule. List conflicting law or rule, if known: Whether it was the plan or not, more people are moving to remote areas permanently, or spending more time there because remote work is possible. Given that full sewage systems are not feasible, and in many cases septic is not possible, this is truly the only responsible way to protect both people and the environment. This would eliminate the primary reason that people dump their septic waste illegally.

☐ It duplicates another federal, state or local law or rule. List duplicate law or rule, if known:

☐ Other (please explain): PLEASE help us protect our beloved environment by allowing the safe, affordable, simple storage of septic waste that can be pumped and managed by companies equipped to do so.

I WOULD LOVE TO TALK TO ANYONE WHO CAN HELP WITH THIS! I've been working with my neighbors and it matters so much to us. I am putting a lot of time into trying to solve this problem, but I need help!

Petition for Rulemaking

WAC 246-272A-0240, Holding Tank Sewage Systems – On-Site Sewage

Shay Bauman, Policy Advisor – August 7, 2024

WASHINGTON STATE BOARD OF HEALTH

Petition for Rulemaking

- Requests that the rule allow for external storage of septic waste in approved containers of at least 200 gallons with a valid pumping contract.
  - States that this is legal with sanicans (portable toilets), but that sanicans are unsafe, inconvenient, and harder to keep clean and secure.
  - Claims that this change is needed because too many people illegally dump waste.
  - States that the rule is inconsistent, as some counties allow this, but others do not.

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)(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.

Class A Waivers – Department of Health Guidance

Local Health Officers **may** grant waivers to WAC 246-272A-0240(2)

- “Holding tank used for other than part-time, non-residential use”

- Minimum criteria includes:
  - Holding tank design criteria – must be consistent with recommended standards and on the “approved list”

  - Performance assurance of system – Established management program, which assures the on-going proper operation and maintenance of the system

Recommendation

Decline the petition for rulemaking to amend WAC 246-272A-0240.

- Department of Health guidance provides a process for these to be allowed, with necessary protections and broad discretion granted to county health officials.
- Previous versions of the rule allowed these systems for seasonal use, and there were many problems that led to the current restrictions.
- Ongoing maintenance is impractical and expensive.
- Further research on capacity to pump these tanks is underway.

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.

An Application Guide for

Granting Waivers from State On-Site Sewage System Regulations, Chapter 246-272A WAC

April 2017

Washington State Department of Health

An Application Guide for

Granting Waivers from State On-Site Sewage System Regulations, Chapter 246-272A WAC

April 2017

Washington State Department of Health

For information or additional copies of this report contact:
Wastewater Management Program
Physical address: 101 Israel Road SE, Tumwater, WA 98501
Mailing Address: PO Box 47824, Olympia, Washington 98504-7824

Phone: (360) 236-3330
FAX: (360) 236-2257
Webpage: www.doh.wa.gov/wastewater
Email: wastewatermanagement@doh.wa.gov

Umair A. Shah, MD, MPH
Secretary of Health

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Para solicitar este documento en otro formato, llame al 1-800-525-0127. Clientes sordos o con problemas de audición, favor de llamar al 711 (servicio de relé de Washington) o enviar un correo electrónico a civil.rights@doh.wa.gov.

DOH 337-021

Contents

List of Tables

Introduction

As a result of State Board of Health rule (Chapter 246-272 WAC) adoption in 1995, and the incorporation of the waiver requirements into statute (RCW 70.05), the Department of Health (DOH) developed a process by which waivers may be granted from the state on-site sewage regulations. The revision to the on-site sewage system rules in 2005 (WAC 246-272A) retains the same waiver process to assure that all waivers granted by the local health officer are consistent with the standards in, and intent of, the state board of health rules. The procedural framework maintains public health protection at least equal to the level established by the provisions in Chapter 246-272A WAC On-Site Sewage Systems.

This manual is furnished to serve as a guide to local health department staff who are involved in evaluating and granting waivers from state regulations, and to clarify the review process and reporting requirements. The standards that are referenced in the manual for approved mitigation measures are performance-based or design-specific technical specifications and related management practices for on-site sewage systems and their components. These standards are intended to provide, as far as practicable, uniformity of practice. They are based on standard engineering practice, and are deemed the best technical documents based on available information.

Technical questions pertaining to DOH waiver requirements, as well as questions regarding waiver process contact:

Leslie Turner
Wastewater Management Specialist
Washington State Department of Health
P.O. Box 47824
Olympia, WA 98504-7824
Phone: 360/236-3043 Fax: 360/236-2257
E-mail: leslie.turner@doh.wa.gov

Section 1: Background

Brief History

Waivers from the state on-site sewage system regulations have been considered in the same manner since the State Board of Health (SBOH) passed an emergency rule revision on December 14, 1994. The emergency rule was passed in response to a specific request the SBOH received from three Washington State Senators concerning issues expressed by some constituents in their legislative districts. The current language has gone from the adopted 1994 rule, into law (RCW 70.05.072 on May 5, 1995) and back to rule (WAC 246-272A on July 13, 2005).

In 1994, these parts of the rule were kept:

- The local health officer has the authority to grant waivers.
- Waivers must be consistent with the purpose and objectives of the rules to meet the public health intent.
- DOH has to concur with the local health officer's decision on the waiver.
- DOH does the waivers for Large On-Site Sewage Systems (systems with design flows over 3,500 gallons per day).
- Waivers are still considered on a "Site-by-Site" basis.

The emergency rule added:

- Local health officers must report to DOH every three months on the waivers they have approved or denied.

These things were removed:

- A Site-by-Site Waiver processing procedure that directly involved the citizen applicant, the local health officer and DOH, and the payment of a fee to cover the cost of the departmental review and concurrence.
- A waiver could no longer be granted that would cover multiple sites at once, each site had to be considered separately.

On May 5, 2005, enacted legislation placed the waiver provisions found in rule into statue (RCW 70.05). The statue paralleled waiver language in WAC 246-272, although not including reference to DOH "concurrence", it clarified the process, which involves DOH oversight and technical assistance, which is currently followed to assure concurrence. It also provided suspension of waiver authority if problems are not corrected after DOH technical assistance is provided.

On July 13, 2005, the SBOH adopted revisions to the on-site sewage systems rules (WAC 246-272A). The rule revisions incorporated the waiver statue language into the rules so that the waiver rule provisions are consistent with the statue. Section 5,

Appendix A- RCW 70.05.072 and WAC 246-272A-0420 is the exact language of the law and the rule that are being used now.

Section 2: Basic Concepts

Key Elements

The Department's of Health's process for granting a waiver is consistent with the basic concepts and general direction provided in the rules and statue (See Appendix A- RCW 70.05.072 and WAC 246-272A-0420). These key elements provides a framework to understand the process:

- The local health officer has the authority to grant waivers.
- Waivers may be considered and granted only on an individual, "site-by-site" basis.
- Only those waivers that are consistent with the public health protection provided by the state rules may be granted.
- The local health officer must report each quarter to DOH about any waivers approved or denied
- The local health officer's authority to grant waivers may be suspended if inconsistencies are not corrected after DOH technical assistance is provided.

Statewide Standards for Public Health Protection

The Washington State Board of Health (SBOH) On-Site Sewage System rules (Chapter 246-272A WAC) encompasses the minimum statewide standards for public health protection. Implemented by local health jurisdictions and by the state department of health, these rules are developed for statewide application.

The on-site sewage system rules provide minimum standards and operational framework for on-site sewage treatment and effluent dispersal, including technical specifics for siting, use, design, installation, permitting, repair of failures, minimum land area, and operation and maintenance. These standards and requirements are established to assure safe treatment and dispersal of sewage, providing protection of public health and water quality. As it is unlikely that the rules apply equally well to all sites encountered in the state, DOH has developed the process with assurance and oversight in this manual so that the rules may be waived.

Mitigation-Based Waiver

Waivers of state regulations may be granted only when the local health officer determines that the requested waiver is consistent with the standards in, and the intent of, the public health protection purpose and objectives of the rules. As the rules provide the minimum standards for public health and water quality protection, any waiver, or "set-aside" of any portion of the rules must provide a corresponding mitigation measure(s) to assure that public health and water quality protection at least equal to that established by the rules, is provided. Only in rare instances, where the resulting risk to public health or water quality is not increased, is waiving minimum standards allowed without appropriate mitigation measures.

Conceptual Framework for Waiver Process

The following conditions must be met by the local health jurisdiction to maintain consistency between the waivers granted and the standards in, and intent of, WAC 246-272A:

- **Site-by-Site application of the state rules, review and granting of waivers.** (Each site and proposed design / development must be considered independently. Local waiver judgment is to be made on a site-by-site basis, as opposed to, for example, “all 45 lots in this subdivision”).

- **Local waiver decisions made by qualified and authorized personnel.** (These persons must have knowledge of the principles, and the state / local processes for “mitigation-based” waivers, and specific written authorization by the Local Health Officer.)

- **Waivers based on the criteria established, and guidance materials provided by DOH.** (This will help assure that an equal level of protection of public health and water quality is provided throughout the statewide network of 34 local health jurisdictions).

- **Timely, complete, and accurate reporting to DOH.** (Local record keeping and documentation of waiver activity, needs to be filed for easy retrieval and open to local program quality assurance review by DOH).

Functional Framework for Waiver Process

In overview, the process for granting waivers from state on-site sewage system regulations involves the following steps (See Figure 1 for a schematic of the process):

- The local health officer reviews a waiver request for a site / development, and decides whether the proposed waiver request is consistent with the public health protection intent of the rules.

- The local health officer chooses a waiver from one of three classifications established by DOH (See Section 3, Classes of Waivers):

  Class A - DOH has specific evaluation criteria and mitigation measures already in place for state-wide use.

  Class B - The local health jurisdiction, with agreement by DOH, has established how the waiver will be evaluated and what mitigation measures are necessary to protect public health under local conditions in a jurisdictional area.

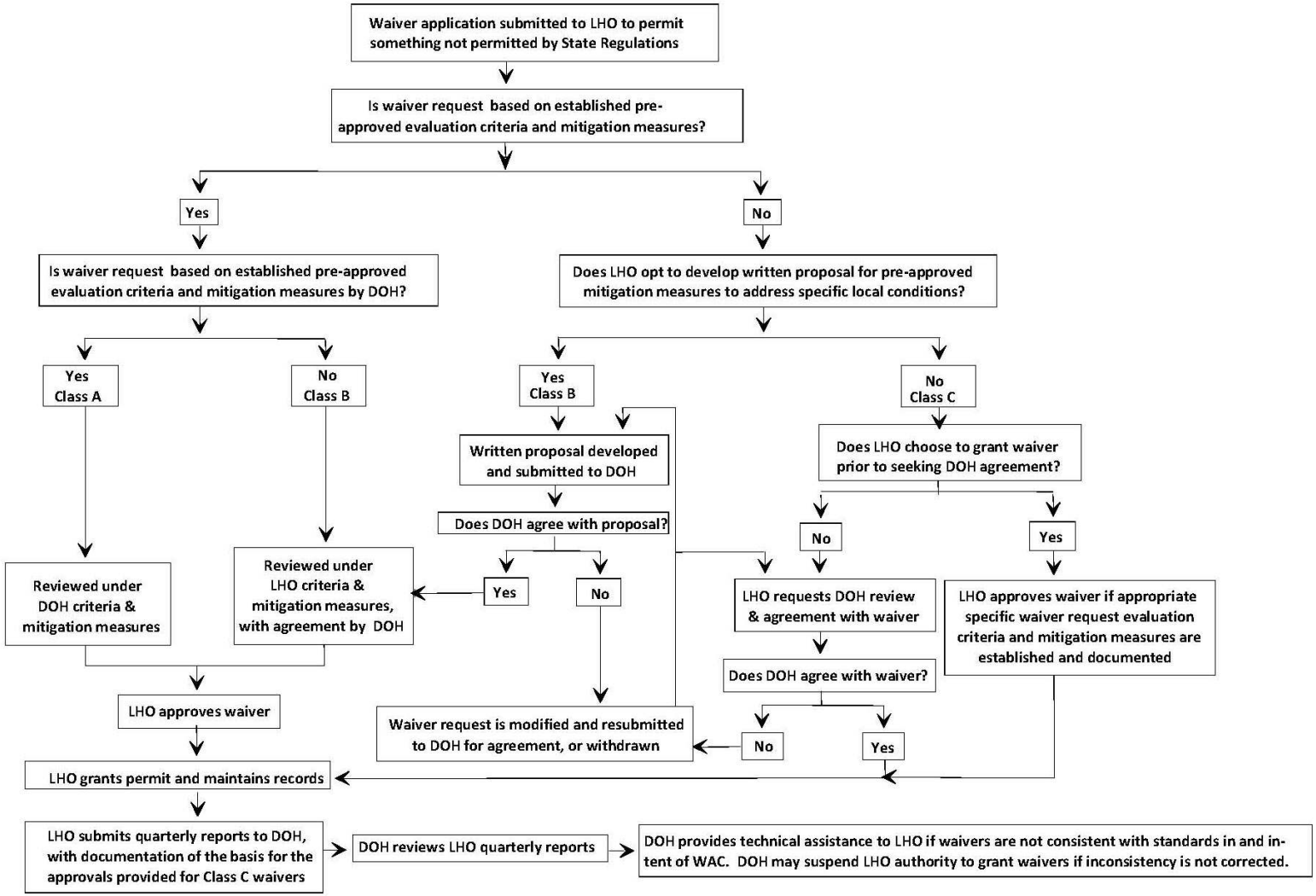
  Class C - There are no pre-approved standards that cover the situation. Before a Class C waiver request is decided, the local health officer must establish appropriate specific waiver request evaluation criteria against which proposed mitigation measures are judged. While not required, local health officer consultation with DOH prior to granting a Class C waiver is strongly recommended.

- The local health officer assures that local waivers will be consistent with the rules by applying specific evaluation criteria and mitigation measures. All local health jurisdiction staff who reviews waiver requests should have wastewater management training and maintain continuing education in this area. At a minimum, this includes ensuring staff that are involved in reviewing waivers complete this Application Guide for Granting Waiver from State On-Site Sewage Systems Regulations, and hold an Inspector Certificate of Competency from the Washington State Department of Licensing.

- The local health officer provides quarterly reports to DOH on the waiver of state regulation activity in his/her jurisdiction. DOH provides waiver forms to ensure that all local health jurisdictions are reporting information in the same format and at the same time. (See Section 5, Appendix B - Waiver Forms). The reports are reviewed by DOH with technical assistance provided for oversight and assurance of local waiver activities.

- If DOH finds any inconsistency between the waiver grants and the state standards, the department will provide technical assistance to the local health officer. If the inconsistencies are not corrected, the department may suspend the authority of the local officer to grant waivers. Once the inconsistencies have been corrected, DOH has the option to allow the local health officer to grant waiver again.

Figure 1. Evaluation of Site-By-Site Waiver Requests of State Regulations



Section 3: Classes of Waivers

Class A

A waiver for which review criteria and mitigation measures have been pre-approved by the Department of Health on a statewide basis (See Tables 1-4. Class A - Pre-Approved Review Criteria and Mitigation Measures on pages 9-14).

- DOH agreement with individual waivers approved by qualified/authorized local health department practitioners can be assumed if pre-approved review criteria and mitigation measures are applied.
- Records of Class A waivers will be maintained by local health agencies and made available to DOH upon request.
- Local health departments will submit quarterly reports concerning all Class A waivers requests and the review criteria and mitigation measures applied.

Class B

A waiver for which a local health agency and DOH have established pre-approved review criteria and mitigation measures to address specific local conditions or issues in an individual county or jurisdictional area.

- DOH agreement with individual waivers approved by qualified /authorized local health agencies practitioners can be assumed if pre-approved review criteria and mitigation measures are applied.
- Class B Waivers, with their review criteria and mitigation measures, are proposed by a local health agency and reviewed and approved by DOH, prior to their application,
- DOH wastewater program staff are available for consultation to assist the development of Class B Waivers and appropriate review criteria and mitigation measures. The amount of proposal-support documentation will vary with the complexity of the issues surrounding the specific waiver. Prior to DOH approval and local health agency application of a Class B Waiver, a written proposal is developed by the local health agency and submitted to DOH.

A proposal must describe the specific requirements to be waived, the review criteria to be used and site/design/administrative mitigation measures to be employed to provide an equal level of public health protection, and technical / public health protection justification for the proposed actions. Also, provide, if applicable, the anticipated methods of verification that the mitigation measures proposed/used provide the level of public health protection needed.

- Based on discussions with the local health agency and review of the written proposal, DOH will either agree with the proposal, request additional information, or determine that waivers advocated by the proposal would be inconsistent with the intent of the State Board of Health on-site sewage regulations. Class B waivers may be granted by the local health officer only after DOH agrees with the proposed review criteria and mitigation measures. Denial of proposal may be appealed.

- Records of Class B waivers will be maintained by local health agencies and available to DOH upon request.

Local health agencies will submit quarterly reports concerning the Class B waivers requests and the review criteria and mitigation measures applied.

Class C

A waiver for which no pre-approved review criteria and mitigation measures have been developed; Department of Health approval for review criteria and mitigation measures can be secured on a case-by-case basis.

- DOH must grant agreement for each waiver individually. The agreement may be obtained either prior to local health agency approval in consultation with DOH or after local health agency granting through the quarterly reporting process. (Advance agreement is strongly recommended.)

- Local health jurisdictions may consult with DOH regarding a waiver/review criteria and mitigation proposal to discuss the adequacy of technical justification, review criteria, site/design/administrative mitigation measures, and verification methods. DOH may agree with the proposal, request additional information or determine that the proposed waiver and review criteria and mitigation measures would be inconsistent with the intent of the State Board of Health on-site sewage regulations.

- A local health jurisdiction may grant a waiver prior to securing agreement by DOH. In such instances, the local health jurisdiction must submit, with the next quarterly report, complete documentation of the basis for the waiver including, as applicable, technical justification, review criteria, site/design/administrative mitigation measures, and proposed methods of verification.

Class C Waivers, with their review criteria and mitigation measures, upon agreement by DOH, may be considered for inclusion on the local/state pre-approved Class B Waiver options list for the health jurisdiction. Any subsequent application for waiver for the same portion of the state regulations could then be treated as a Class B waiver application within the applicable county or jurisdiction.

Table 1. CLASS A - NONPERFORATED DISTRIBUTION LINE HORIZONTAL SEPARATIONS (6/07)

WAC Section	Specific Item Waived	Extent or Degree Waived	Minimum Issues/Criteria to Consider	Approved Mitigation Measures*
246-272A-0210(1) Table IV	Pressure sewer transport line 10 feet from surface water	Subaqueous crossing of pressure sewer transport line or down to 0 feet horizontally	**1)** Extra protection of integrity of line at crossing	**1a)** Transport line installed in a casing of at least Schedule 40 steel or ductile iron pipe within 10 feet on each side of the crossing. Transport line uniformly supported by pressure-grouting annular space with sand-cement grout or bentonite, or casing spacers or skids installed consistent with AWWA PVC Pipe Design and Installation Manual M23. Underground installation of line consistent with ASTM D 2774.
				1b) Transport line buried at least 3 feet below the bottom of the water body's bed.
				1c) Transport line within 10º of the perpendicular direction of the stream.
			2) Performance testing of line	**2a)** Transport line leakage test consistent with ASTM D 2774, except line should be pressurized to 150% of the system's design operating pressure, but not less than 70 psi, and pressure must hold for 1 hour.
			3) Determination of applicable aquatic resource permitting requirements	**3a)** Submit JARPA application to appropriate review agencies.
246-272A-0210(1) Table IV	Pressure sewer transport line 10 feet from surface water	Aerial crossing of pressure sewer transport line or down to 0 feet horizontally	**1)** Extra protection of integrity of line at crossing	**1a)** Transport line installed in a casing of at least Schedule 40 steel or ductile iron pipe within 10 feet on each side of the crossing. Transport line uniformly supported by casing spacers or skids installed consistent with AWWA PVC Pipe Design and Installation Manual M23.
				1b) Transport line crossing designed by an engineer to prevent freezing, leaking, settlement, lateral movement, and damage from expansion/contraction.
				1c) Transport line located with proper clearance above floodwater conditions.
			2) Performance testing of line	**2a)** Transport line leakage test consistent with ASTM D 2774, except line should be pressurized to 150% of the system's design operating pressure, but not less than 70 psi, and pressure must hold for 1 hour.
			3) Determination of applicable aquatic resource permitting requirements	**3a)** Submit JARPA application to appropriate review agencies.

*** The local health officer may require additional site-specific issues and criteria to consider, and mitigation measures.**

Table 1. CLASS A - NONPERFORATED DISTRIBUTION LINE HORIZONTAL SEPARATIONS (6/07)

WAC Section	Specific Item Waived	Extent or Degree Waived	Minimum Issues/Criteria to Consider*	Approved Mitigation Measures*
246-272A-0210(1) Table IV	Pressure sewer transport line 50 feet from non-public well or suction line	Down to 25 feet	**1)** Extra protection of integrity of line within 50 feet of well	**1a)** Transport line installed in a casing of at least Schedule 40 PVC within 50 feet of well. Transport line uniformly supported by pressure-grouting annular space with sand-cement grout or bentonite, or casing spacers or skids installed consistent with AWWA PVC Pipe Design and Installation Manual M23. Underground installation of line consistent with ASTM D 2774.
			2) Performance testing of line	**2a)** Transport line leakage test consistent with ASTM D 2774, except line should be pressurized to 150% of the system's design operating pressure, but not less than 70 psi, and pressure must hold for 1 hour.
			3) Determination of any existing covenants or easements for maintaining a sanitary control area	**3a)** Notify well owner of proposed encroachment if there are no existing covenants or easements establishing a control area.
246-272A-0210(1) Table IV	Building sewer, collection, non-pressure non-perforated distribution line 50 feet from non-public well or suction line	Down to 25 feet	**1)** Extra protection of integrity of line within 50 feet of well	**1a)** Line installed in a casing of at least Schedule 40 PVC pipe within 50 feet of well. Transport line uniformly supported by pressure-grouting annular space with sand-cement grout or bentonite, or casing spacers or skids installed consistent with AWWA PVC Pipe Design and Installation Manual M23.
				1b) Underground installation of line consistent with ASTM D 2321.
			2) Performance testing of line	**2a)** Line leakage test consistent with ASTM F 1417 or exfiltration test consistent with WSDOT 7-17.3(2)B.
			3) Determination of any existing covenants or easements for maintaining a sanitary control area	**3a)** Notify well owner of proposed encroachment if there are no existing covenants or easements establishing a control area.

*** The local health officer may require additional site-specific issues and criteria to consider, and mitigation measures.**

Table 2. CLASS A - SEWAGE TANK HORIZONTAL SEPARATIONS (6/07)

WAC Section	Specific Item Waived	Extent or Degree Waived	Minimum Issues/Criteria to Consider*	Approved Mitigation Measures*
246-272A-0210(1) Table IV	Sewage tank 50 feet from surface water	Down to 25 feet	**1)** Extra protection of integrity of tank and joints	**1a)** Waterproof surface barrier applied to concrete tank consistent with Manual of Concrete Practice ACI 515.1R. Flexible rubber boots or compression seals meeting ASTM C 1644, or flexible couplings meeting ASTM C 1173 used for inlet and outlet connections to provide flexibility in case of tank settlement while still maintaining a watertight seal. An approved double-wall fiberglass tank may be used in lieu of a concrete tank.
			2) Performance testing of tank	**2a)** Concrete tank tested for water-tightness consistent with ASTM C 1227. Fiberglass tank tested for water-tightness consistent with IAPMO/ANSI Z1000-2007.
			3) Accessibility of tank for ease of operation and maintenance	**3a)** Access openings at or above finished grade with lockable lids or secured to prevent unauthorized entry.
246-272A-0210(1) Table IV	Sewage tank 50 feet from non-public well or suction line	Down to 25 feet	**1)** Extra protection of integrity of tank and joints	**1a)** Waterproof surface barrier applied to concrete tank consistent with Manual of Concrete Practice ACI 515.1R. Flexible rubber boots or compression seals meeting ASTM C 1644, or flexible couplings meeting ASTM C 1173 used for inlet and outlet connections to provide flexibility in case of tank settlement while still maintaining a watertight seal. An approved double-wall fiberglass tank may be used in lieu of a concrete tank.
			2) Performance testing of tank	**2a)** Concrete tank tested for water-tightness consistent with ASTM C 1227. Fiberglass tank tested for water-tightness consistent with IAPMO/ANSI Z1000-2007.
			3) Accessibility of tank for ease of operation and maintenance	**3a)** Access openings at or above finished grade with lockable lids or secured to prevent unauthorized entry.
			4) Determination of any existing covenants or easements for maintaining a sanitary control area	**4a)** Notification of proposed encroachment to the well owner if there are no existing covenants or easements establishing a control area.
246-272A-0210(1) Table IV	Sewage tank 10 feet from pressured water supply line	Down to 2 feet	**1)** Extra protection of integrity of tank and joints	**1a)** Waterproof surface barrier applied to concrete tank consistent with Manual of Concrete Practice ACI 515.1R. Flexible rubber boots or compression seals meeting ASTM C 1644, or flexible couplings meeting ASTM C 1173 used for inlet and outlet connections to provide flexibility in case of tank settlement while still maintaining a watertight seal. An approved double-wall fiberglass tank may be used in lieu of a concrete tank.
			2) Performance testing of tank	**2a)** Concrete tank tested for water-tightness consistent with ASTM C 1227. Fiberglass tank tested for water-tightness consistent with IAPMO/ANSI Z10000-2007..
			3) Accessibility of tank for ease of operation and maintenance	**3a)** Access openings at or above finished grade with lockable lids or secured to prevent unauthorized entry.
			4) Extra protection of integrity of water line	**4a)** Water line installed in casing of at least Schedule 40 PVC within 10 feet of the tank. Water line uniformly supported by pressure-grouting annular space with sand-cement grout or bentonite, or casing spacers or skids installed consistent with AWWA PVC Pipe Design and Installation Manual M23-7-2.

*** The local health officer may require additional site-specific issues and criteria to consider, and mitigation measures.**

Table 3. CLASS A - SOIL DISPERAL COMPONENT HORIZONTAL SEPARATIONS (6/07)

WAC Section	Specific Item Waived	Extent or Degree Waived	Minimum Issues/Criteria to Consider*	Approved Mitigation Measures*
246-272A-0210(4)	Soil dispersal component 75 feet from surface water	Down to 50 feet, except not in Soil Type 1	**1)** Enhanced treatment performance	**1a)** Treatment component or sequence listed as meeting Treatment Level B. Disinfection can not be used to achieve the fecal coliform limit of the treatment level. The soil dispersal component maintaining at least 3 feet vertical separation; i.e., sand filter followed by a gravity distribution SSAS with at least 3 feet of vertical separation or by a pressure distribution SSAS with at least 2 feet of vertical separation. . A mound system with 2 feet of sand media may be allowed, if there is at least 3 feet of available soil depth.
			2) Performance assurance of treatment system	**2a)** Management program established, which assures the on-going proper operation and maintenance of the system.
			3) Hydrogeologic susceptibility	**3a)** Adequate protective site specific conditions existing, such as physical settings with low hydrogeologic susceptibility from contaminant infiltration; i.e. evidence of excessive depth to groundwater, down-gradient contaminant source, or outside a sensitive area.
246-272A-0210(4)	Soil dispersal component 75 feet from non-public well or suction line	Down to 50 feet, except not in Soil Type 1	**1)** Enhanced treatment performance	**1a)** Treatment component or sequence listed as meeting Treatment Level B. Disinfection can not be used to achieve the fecal coliform limit of the treatment level. The soil dispersal component maintaining at least 3 feet vertical separation; i.e., sand filter followed by a gravity distribution SSAS with at least 3 feet of vertical separation or by a pressure distribution SSAS with at least 2 feet of vertical separation. A mound system with 2 feet of sand media may be allowed, if there is at least 3 feet of available soil depth.
			2) Performance assurance of treatment system	**2a)** Management program established which assures the on-going proper operation and maintenance of the system.
			3) Hydrogeologic susceptibility	**3a)** Adequate protective site specific conditions existing, such as physical settings with low hydrogeologic susceptibility from contaminant infiltration; i.e. evidence of confining layers and or aquitards separating potable water from the OSS treatment zone, excessive depth to groundwater, down-gradient contaminant source, or outside the zone of influence.
			4) Determination of any existing covenants or easements for maintaining a sanitary control area	**4a)** Notification of proposed encroachment to the well owner if there are no existing covenants or easements establishing a control area.

*** The local health officer may require additional site-specific issues and criteria to consider, and mitigation measures.**

Table 3. CLASS A - SOIL DISPERSAL COMPONENT HORIZONTAL SEPARATIONS (6/07)

WAC Section	Specific Item Waived	Extent or Degree Waived	Minimum Issues/Criteria to Consider	Approved Mitigation Measures*
246-272A-0210(1) Table IV	Soil dispersal component 10 feet from pressurized water supply line	Down to 5 feet	**1)** Extra protection of integrity of water line	**1a)** Water line installed in casing of at least Schedule 40 PVC within 10 feet of the dispersal component. Water line uniformly supported by pressure-grouting annular space with sand-cement grout or bentonite, or casing spacers or skids installed consistent with AWWA PVC Pipe Design and Installation Manual M23.
			2) Performance testing of water line	**2a)** Water line leakage test consistent with WSDOT 7-09.3(23) Hydrostatic Pressure Test.
			3) Hydrogeologic susceptibility	**3a)** Adequate protective site specific conditions existing, such as physical settings with low hydrogeologic susceptibility from contaminant infiltration; i.e. deep, well-drained soils or down-gradient contaminant source.
246-272A-0210(1) Table IV	Soil dispersal component 30 feet from interceptor/ curtain drains/ drainage ditches down-gradient	Down to 15 feet, except not in Soil Type 1	**1)** Enhanced treatment performance	**1a)** Treatment component or sequence listed as meeting Treatment Level B. Disinfection not used to achieve the fecal coliform limit of the treatment level. The soil dispersal component with pressure distribution and maintaining at least 2 feet vertical separation; i.e. sand filter followed by a pressure distribution drainfield with at least 2 feet vertical separation.
			2) Performance assurance of treatment system	**2a)** Management program established which assures the on-going proper operation and maintenance of the system.
246-272A-0210(1) Table IV	Soil dispersal component 25 feet from down-gradient cuts or banks with at least 5 feet of original soil above a restrictive layer	Down to 12 feet, except not in Soil Type 1	**1)** Enhanced treatment performance	**1a)** Treatment component or sequence listed as meeting Treatment Level B. Disinfection not used to achieve the fecal coliform limit of the treatment level. The soil dispersal component with pressure distribution and maintaining at least 3 feet vertical separation; i.e. sand filter followed by a pressure distribution drainfield with at least 3 feet vertical separation.
			2) Performance assurance of treatment system	**2a)** Management program established, which assures the on-going proper operation and maintenance of the system.
			3) Stability of bank or cut	**3a)** Evidence of slope stability.
246-272A-0210(1) Table IV	Soil dispersal component 50 feet from down-gradient cuts or banks with less than 5 feet of original soil above a restrictive layer	Down to 25 feet, except not in Soil Type 1	**1)** Enhanced treatment performance	**1a)** Treatment component or treatment sequence listed as meeting Treatment Level B. Disinfection not used to achieve the fecal coliform limit of the treatment level. The soil dispersal component with pressure distribution and maintaining at least 2 feet vertical separation; i.e. sand filter followed by a pressure distribution drainfield with at least 2 feet vertical separation.
			2) Performance assurance of treatment system	**2a)** Management program established which assumes the on-going proper operation and maintenance of the system.
			3) Stability of bank or cut	**3a)** Evidence of slope stability.

*** The local health officer may require additional site-specific issues and criteria to consider, and mitigation measures.**

Table 4. CLASS A - MISCELLANEOUS DESIGN PROVISIONS (6/07)

WAC Section	*Specific Item Waived*	*Extent or Degree Waived*	*Minimum Issues/Criteria to Consider**	*Approved Mitigation Measures**
246-272A-0240(2)	Holding tank used only for permanent uses limited to controlled, part-time, commercial usage situations.	Holding tank used for other than part-time non-residential use	**1)** Holding tank design criteria	**1a)** Design criteria consistent with the Recommended Standards and Guidance for Holding Tank Sewage Systems, and tank on current "Approved List".
			2) Performance assurance of system	**2a)** Management program established, which assures the on-going proper operation and maintenance of the system.
246-272A-0234(3)(a)	SSAS infiltrative surface depth shall not exceed 10 feet from the finished grade	Down to 20 feet in depth	**1)** Enhanced treatment performance	**1a)** Treatment with greater than 3 feet of sand-lined bed/trench media and the soil dispersal component's infiltrative surface is installed in suitable native soil consistent with the Recommended Standards and Guidance for Sand Lined Trench Systems.
			2) Performance assurance of treatment system	**2a)** Management program established, which assures the on-going proper operation and maintenance of the system.
			3) Hydrogeologic susceptibility	**3a**) Adequate protection site specific conditions existing, such as physical setting with low hydrogeologic susceptibility from contaminant infiltration. The point where the treated wastewater is applied to the native soil for dispersal must be within the zone of aeration.
246-272A-0234(3)(b)	All SSAS must have a minimum of six inches of sidewall located in original undisturbed soil	SSAS sidewall installed in unoriginal disturbed soil (installed in fill)	**1)** Enhance treatment performance	**1a)** Treatment component or sequence listed as meeting Treatment Level B without disinfection to achieve the fecal coliform limit. The soil dispersal component maintaining at least 3 feet separation between the bottom of the infiltrative surface and the highest seasonal water table, a restrictive layer or soil type 7 i.e. sand filter followed by gravity distribution SSAS with a least 3 feet separation or by pressure distribution SSAS with a least 2 feet of separation.
246-272A-0234(4)(b)	The sidewall below the invert of the distribution pipe is located in original, undisturbed soil	SSAS sidewall installed in unoriginal disturbed soil (installed in fill)	**2)** Performance assurance of treatment system	**2a)** Management program established, which assures the on-going proper operation and maintenance of the system.
			3) Hydrogeologic characteristics	**3a)** Evidence of soil stability, and soil (fill material) displays suitable hydraulic conductivity.
246-272A-0234(3)(c)	SSAS beds are only designed in Soil Types 1, 2, 3, or in fine sands with a width not exceeding 10 feet	Allow bed in Soil Type 4-6, with a width not exceeding 10 feet	**1)** Enhance treatment performance	**1a)** Treatment component or sequence listed as meeting Treatment Level B without disinfection to achieve the fecal coliform limit. The SSAS bed maintaining at least 3 feet vertical separation; i.e. sand filter followed by gravity distribution bed with a least 3 feet vertical separation (pressure distribution with 2 feet of vertical separation allowed). A pressure distribution bed with at least 4 feet of vertical separation may be substituted for a Treatment level B treatment component/sequence.
			2) Performance assurance of treatment system	**2a)** Management program established, which assures the on-going proper operation and maintenance of the system.
			3) Extra protection of soil during construction to limit damage to infiltrative surface	**3a)** Site preparation, excavation, placement of gravel, and backfilling operations done with the proper equipment and care. Only low load-bearing construction equipment to be used in the bed area to limit soil compaction.
				3b) Construction proceeds only during low soil moisture content conditions (below its plastic limit). Once exposed, infiltrative surface covered within 12 hours to prevent desiccation or before periods of precipitation to prevent puddling.

*** The local health officer may require additional site-specific issues and criteria to consider, and mitigation measures.**

Section 4: Waiver Reporting

Waiver Approval Form

This single page document is the primary waiver recording form and contains the following information (See Appendix B - Request for Wavier from State Regulations form):

- Basic permit data (applicant name, site address, designer name, etc.);
- Specific rule/requirement waived (section and subsection of Chapter 246-272A WAC);
- Site/design/administrative mitigation measures proposed and any additional evaluation criteria and/or mitigation measures employed; Type of Waiver (Class A, B, or C);
- Confirmation of adjacent or affected property owner notification (if appropriate); and
- Approval signature (by qualified/authorized local health agency personnel).

This form is completed whenever a waiver of state regulations is requested.

Local Record-Keeping / Data Management

The local health officer is required to maintain complete and retrievable records of all waivers reviewed, granted or denied. Individual waiver request forms / records are, at minimum, to be filed with the sewage system permit records. A copy of the waiver request form may also be filed in a separate file as an on-going record of waivers reviewed, approved or denied. Electronic record keeping may also be used to track and retrieve information regarding waivers.

Quarterly Reports Form Local Health Officer to DOH

Both the statue and WAC 246-272A requires that the local health officer report quarterly to DOH regarding the waiver request activity within their jurisdiction. This involves submitting all waiver requests (any waiver approved or denied) to DOH by their quarterly due date.

Report Schedule

First Quarterly (January - March)	Due April 15th
Second Quarterly (April - June)	Due July 15th
Third Quarterly (July - September)	Due October 15th
Fourth Quarter (October - December)	Due January 15th

Report Format

Each Quarterly Report is to consist of the following items:

- Copies of each complete waiver application (See Section 5, Appendix B - Request for Wavier from State Regulations form) acted on during the time-period of the report. "acted on" means reviewed and either approved or denied. Waiver requests received but pending review or decision will be reported in subsequent reports.

- A Quarterly Report Coversheet, with the signature of the local health officer or authorized local health agency supervisory personnel, to indicate that the local health officer is adequately informed regarding waiver activity (See Section 5, Appendix B - Quarterly Report).

DOH Review / Technical Assistance / Assurance

The Department of Health is available for consultation and technical assistance at any point in the local health officer review and decision-making processes. Inquiry and discussion prior to granting waivers is encouraged when questions or issues arise. This is particularly true for Class C waivers for which no specific waiver request evaluation criteria or pre-approved mitigation measures have been developed.

The DOH has a principal role in the assurance of consistent and appropriate extension of public health protection in all local health jurisdictions. To that end, DOH will review the local health officer's quarterly reports regarding their waiver review and granting activity. It is anticipated that a more comprehensive oversight will be provided through periodic local on-site sewage program reviews, as opposed to response to received problems or complaints. DOH, however, will respond to non-agreement or non-compliance issues as they arise.

Assembly / How the Components Link Together

- Flow Chart: A comprehensive Flow Chart presents the three primary process routes for waiver of state regulations. As this chart presents all of the information together to show the interrelationships, the reader is encouraged to study the chart section-by-section, by class of waiver. The chart format is a "decision-tree" -- that is, a question leads the reader depending upon the answer, "Yes" or "No" (See Figure 1 - Evaluation of Site-By-Site Waiver Requests of State Regulations).

Reference Materials: The various reference materials provided in the **Referenced Standards and Technical Material for On-Site Sewage Systems** notebook supports the Class A mitigation measures found in Tables 1-4. Most of the materials are technical in nature, which are based on standard engineering and industry practice, intended to provide uniformity of practice. In addition, terms used in this document which need definition or clarification are provided in Appendix C of Section 5- Glossary of Terms.

Section 5: Appendixes

Appendix A - Statutory Authority and Regulations Pertaining To Waivers

RCW 70.05.072 Local health officer–Authority to grant waiver from on-site sewage system requirements. The local health officer may grant a waiver from specific requirements adopted by the state board of health for on-site sewage systems if:

(1) The on-site sewage system for which a waiver is requested is for sewage flows under three thousand five hundred gallons per day;

(2) The local health officer on an individual, site-by-site basis evaluates the waiver request;

(3) The local health officer determines that the waiver is consistent with the standards in, and the intent of, the state board of health rules; and

(4) The local health officer submits quarterly reports to the department regarding any waivers approved or denied.

Based on review of the quarterly reports, if the department finds that the waivers previously granted have not been consistent with the standards in, and intent of, the state board of health rules, 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.

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, the department may suspend the authority of the local health officer to grant waivers under this section until such inconsistencies have been corrected.

WAC 246-272A-0420 Waiver of state regulations. (1) The local health officer may grant a waiver from specific requirements of this chapter if:

(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) The local health officer submits quarterly reports to the department regarding any waivers approved or denied; and

(d) Based on review of the quarterly reports, if the department finds that the waivers previously granted have not been consistent with the standards in, and the intent of these rules, 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. 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, the department may suspend the authority of the local health officer to grant waivers under this section until such inconsistencies have been corrected.

(2) The department shall develop guidance to assist local health officers in the application of waivers.

Appendix B – Waiver Forms

On-Site Sewage Systems (Chapter 246-272A WAC)
Waivers from State Regulations

Quarterly Report

TO: Washington State Department of Health
On-Site Sewage System Waivers
P.O. Box 47824
Olympia WA 98504-7824

FROM: _____

Copies of Waiver Request Forms and this transmittal sheet are to be submitted by the local health officer to the Washington State Department of Health for each quarter of the year. Submittal of this information is part of the process required under 70.05 RCW for waivers of state regulations granted by the local health officer.

Year:

☐ 2017 ☐ 2018
☐ 2019 ☐ 2020
☐ 2021 ☐ 2022
☐ 2023 ☐ 2024
☐ 2025 ☐ 2026

Quarter:

☐ 1st (January – March) [Due April 15]
☐ 2nd (April – June) [Due July 15]
☐ 3rd (July – September) [Due October 15]
☐ 4th (October – December) [Due January 15]

With this transmittal sheet are copies of the Requests For Waiver From State Regulations for On-Site Sewage Systems (Chapter 246-272A WAC) received and either approved or denied during the indicated year and quarter.

These waiver requests were reviewed, and approved or denied in full compliance with the provisions of the Washington State Board of Health's on–site sewage system rules.

Where waivers have been granted, the conditions, comments, requirements and mitigation measures have been evaluated for their ability to provide public health protection at least equal to that provided by Chapter 246-272A WAC On-Site Sewage Systems.

All waivers granted under these provisions have been evaluated and approved either by the local health officer or persons specifically authorized by the local health officer.

_____ _____
Local Health Officer **Date**

On-Site Sewage Systems (Chapter 246-272A WAC)
Request for Waiver from State Regulations

Section I.	*(completed by applicant)*
Name: ***(1)***	Local Health Department / District ***(2)*** *(see instructions)*
Address:	
Telephone: ( )	
Signature:	
Property Identification: ***(3)***	

Section II.	*(completed by applicant)*	
WAC Number**:** ***(4)***	WAC Requirement: ***(5)***	Waiver Sought: ***(6)***
246-272A —		
Subsection:		
Justification *(mitigation measures to be provided)*: ***(7)***		

Section III.	*(completed by health officer)*
Review Criteria: ***(8)***	Mitigation Measures *(in addition to those proposed)*: ***(9)***
Comments / Conditions: ***(10)***	
Type of Waiver: ***(11)*** **[ ]** Class A **[ ]** Class B **[ ]** Class C — Request DOH review <u>before</u> granting? Yes ___ No ___	
Neighbor Notification: ***(12)*** Required? Yes ___ No ___ *If needed, are agreements, easements, etc. properly filed?* Yes ___ No ___	

Section IV.	*(completed by health officer)*

This Request For Waiver From State Regulations has been reviewed according to the provisions of Chapter 246-272A WAC On-Site Sewage Systems. The review criteria applied, and the mitigation measures proposed and/or required, have been evaluated for their ability to provide public health protection at least equal to that provided by this chapter WAC.

[ ] Denied **[ ] Approved / Granted** —Subject to all comments, conditions and requirements noted in Sections II and III.

Local Health Officer ***(13)*** _____ **Date:** _____

Instructions for Completion

Sections I and II are to be completed by the Applicant.
Sections III and IV are to be completed by the local health officer or his/her authorized representative.

Most items in each Section are followed by a number in ( ). The instructions below are listed by these numbers:

(1) Individual requesting waiver. (Presumed to be property owner..., indicate if not.) Be sure to include mailing address and phone number.

(2) Local Health Department. Usually this will be "filled in" by the local health jurisdiction for their use.

(3) Property Identification: Provide the address, parcel number, permit application number or other identifying description of the property for which a waiver is being requested. A full legal description is not required.

(4) WAC Number. Specify the particular WAC number from Chapter 246-272A WAC for which a waiver is being sought, such as "WAC 246-272A-0210(1)".

(5) WAC Requirement. State the requirement in the specified WAC for which a waiver is being sought, such as "100 foot setback from soil dispersal component to a well".

(6) Waiver Sought. Briefly describe the waiver sought, such as "Reduction of setback to 70 feet".

(7) Justification. Provide the rationale for the waiver request. What site conditions, system design characteristics, etc. mitigate the concerns that resulted in the requirements in the WAC? Technical justification should include supporting data, plat plans, device or treatment methodology proposed, possible mitigating site characteristics, gross land area, other options explored, and any other pertinent data. Possible mitigation measures may include system design, site requirements, or administrative approaches. Attach additional pages, if necessary to provide the local health officer adequate information upon which to make an informed decision.

(8) Review Criteria. Indicate when specific criteria were used in the review of the proposed waiver and mitigation measures.

(9) Mitigation Measures. Indicate any mitigation measures required in addition to those proposed by the applicant.

(10) Comments / Conditions. Briefly describe any concerns or issues regarding the waiver request, mitigation measures, or related issues.

(11) Type of Waiver. Indicate which category of waivers this particular request is in. For Class C Waivers, indicate if DOH review is to be requested before a decision is made to grant the request.

(12) Neighbor Notification. Are there any aspects of this waiver request for which notification to and/or permission by, adjoining or nearby property owners / dwellers would be appropriate?

(13) Local Health Officer. This is where the local health officer, or his/her authorized representative, by checking the appropriate box and signing, grants or denies the requested waiver.

Assistance for applicants requesting a "Waiver From State Regulations" may be obtained from the Local Health Department or District.

Local Health Department / District Health Officers may obtain assistance from the Washington State Department of Health in their review of proposed "Waiver From State Regulations":
(360) 236-3043 / Leslie Turner

Appendix C - Glossary of Terms

ACI: American Concrete Institute.

ASTM: American Society for Testing and Materials.

Aquitard: A semi-permeable (low porosity) or impermeable geologic layer that impedes vertical movement of groundwater and acts as a confining layer to an aquifer. It may include the following materials: hardpan, clay, till, or massive bedrock.

AWWA: American Water Works Association.

Bed: A soil dispersal component consisting of an excavation with a width greater than three feet.

Casing: A metal or plastic pipe where a PVC pressure transport or gravity collection line is installed inside for additional protection in case of pipe failure or leakage.

Casing Spacers / Skids: Pipe fittings that provide long-term support around the circumference of a PVC pressure transport or gravity collection pipe within a casing. Skids may extend the full length of the pipe encased, with the exception of the bell and spigot position, or may be spaced at intervals inside a casing.

Confining Layer: A layer of impermeable material adjacent to an aquifer that hampers the movement if water into or out of the aquifer.

Desiccation: Thorough removal of water from a soil by drying.

Flexible Coupling: A device used to form a leakproof joint between sections of plain end pipe or fittings of the same or different materials, of the same or different size, or any combination of materials or pipe sizes.

Hydraulic Conductivity: The ability of soil to transmit liquids through pore spaces in a specified direction, e.g., horizontally or vertically.

Hydrogeologic Characteristics: Characteristics that describe the hydrology (the distribution of water on the surface and below the ground) and the geology (the structure and content of the earth) at a site. Hydrogeologic characteristics include soil type, depth to ground water, soil permeability, and ground-water recharge rate. These properties control the entrance of water to the subsurface and the capacity to hold, transmit, and deliver water.

Hydrogeologic Susceptibility: Hydrogeologic characteristics that would either impede or enhance the movement of contaminants from the land surface into groundwater or surface water.

Hydrostatic Pressure: The pressure per unit area exerted by water at rest.

IAPMOSPS: International Association of Plumbing & Mechanical Officials Material & Property Standard for Prefabricated Septic Tanks.

Infiltrative Surface: The surface within a treatment component or soil dispersal component to which is applied and thorough which effluent moves into original, undisturbed soil or other porous treatment media.

JARPA: Joint Aquatic Resource Permits Application. Fill out a JARPA to apply for Hydraulic Project Approvals, Shoreline Management Permits, Water Quality Certifications, and U.S. Army Corps of Engineers Section 404 and Section 10 permits.

Load-bearing: The ability to support superimposed loads without shear failure or excessive deformation within the soil mass.

Local Health Officer: 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.

Maintenance: The actions necessary to keep the on-site sewage system components functioning as designed.

Monitoring: The periodic or continuous checking of an on-site sewage system, 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.

Performance Standard: A standard used to judge whether predetermined requirements have been met, such as the necessary level of treatment for waste stream, after the completion or initiation of operation. Performance standards generally are in the form of a pre-determined level or concentration of a particular compound or constituent that is allowed in a waste effluent.

Plastic Limit: The moisture content at which a soil changes from a semisolid to plastic consistency; characterized by a soil just beginning to crumble when rolled into a wire approximately 1/8 in. in diameter.

Pressure Distribution: A system of small diameter pipes equally distributing effluent throughout a SSAS, as described in the department's "Recommended Standards and Guidance for Pressure Distribution Systems". A subsurface drip system may be used wherever the chapter requires pressure distribution.

Puddling: Act of destroying soil structure, usually by disturbing or compacting the soil at high water content, thereby reducing porosity and permeability.

Registered List: "List of Registered On-site Treatment and Distribution Products", developed and maintained by the department and containing a list of treatment and distribution products that meets the requirements for product registration in WAC 246-272A.

Sanitary Control Area: A horizontal protective radius around a well, which excludes major potential contaminant sources.

Sewage Tank: A prefabricated or cast-in-place septic tank, pump tank/dosing chamber, holding tank, grease interceptor, recirculating filter tank or any other tanks as they relate to on-site sewage systems including tanks for use with proprietary products.

Slope Stability: The resistance of an inclined surface to failure by sliding or collapsing.

Soil Compaction: Increasing the soil bulk density, and concomitantly decreasing the soil porosity, by the application of mechanical forces to the soil. Results in a soil that retains less water and resists root penetration. Soils with high clay content are more easily compacted than sandy soils.

Soil Dispersal Component: A technology that releases effluent from a treatment component into the soil for dispersal, final treatment and recycling.

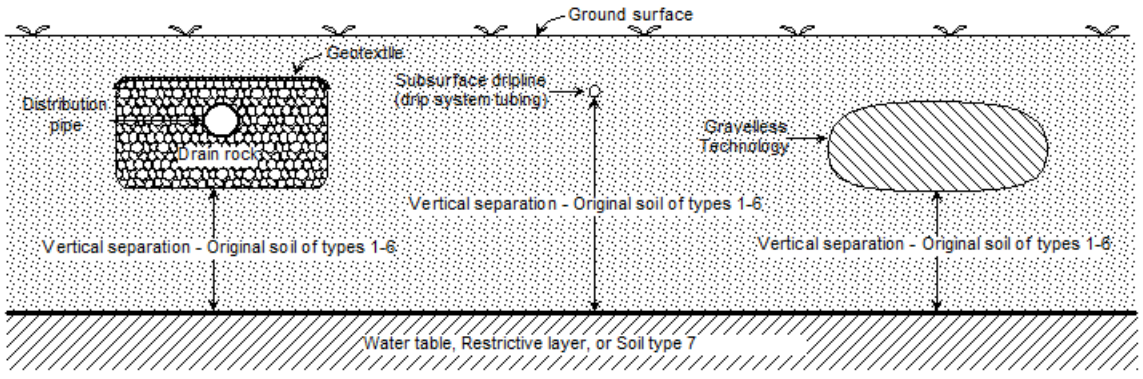
Timed Dosing: The delivery of discrete volumes of sewage at prescribed time intervals.

Treatment Component: A technology that treats sewage in preparation for further and/or dispersal into the soil environment. Some treatment components, such as mound systems, incorporate soil dispersal components in lieu of separate treatment and soil dispersal components.

Treatment level: One of six levels (A, B, C, D, E, & N) 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: Any series of treatment components that discharges treated sewage to the soil dispersal component.

Vertical Separation: The depth of unsaturated, original, undisturbed soil of Soil Types 1-6 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:



Waterproof Surface Barrier: A barrier material applied for treating concrete surfaces to prevent leakage into a retaining structure or to prevent loss of water from a retaining structure.

Well: 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 ground water for agricultural, municipal, industrial, domestic, or commercial use. Excluded are:

(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.

WSDOT: Washington State Department of Transportation.

Zone of Aeration: That part of the ground in which the voids are not continuously saturated.

Zone of Influence: The area surrounding a pumping well within which the water table or potentiometric surfaces have been changed due to groundwater withdrawal.

Section 6: References

1. ***Criteria for Sewage Works Design, October 2007***, Publication No. 98-37 WQ Washington State Department of Ecology, P.O. Box 47600, Olympia, WA 98504-7600. www.ecy.wa.gov/biblio/9837.html

2. ***On-Site Sewage System Management Plan Guidance for the Twelve Puget Sound Counties, June 2006,*** Washington State Department of Health, P.O. Box 47824, Olympia, WA 98504-7824. www.doh.wa.gov/Portals/1/Documents/Pubs/337-084.pdf

3. ***Handbook of PVC Pipe Design & Construction, Forth Edition, 2001***, Uni-Bell Plastic Pipe Association, 2655 Villa Creek Drive, Suite 155, Dallas, TX 75234-7362

4. ***Management Options for Unstable Bluffs in Puget Sound, Washington, Coastal Erosion Management Studies Volume 8,*** Shorelands and Water Resources Program, Washington Department of Ecology, P.O. Box 47600, Olympia, WA 98504-7600. www.ecy.wa.gov/biblio/94081.html

5. ***PVC Pipe - Design and Installation, Manual of Water Supply Practices M23, Second Edition, 2002.*** American Water Works Association, 6666 West Quincy Avenue, Denver, CO 80235.

6. ***Testing Reinforced Concrete Structures for Watertightness, ACI 350.1R-93/AWWA 400-93,*** ACI Committee 350 report/AWWA Committee 400, American Concrete Institute, P.O. Box 9094 Farmington Hills, MI 48333.

7. **2006** ***Uniform Plumbing Code,*** International Association of Plumbing and Mechanical Officials. 20001 Walnut Drive South, Walnut, CA 91789-2825.

8. ***WSDOT 2006 Standard Specifications for Road, Bridge, and Municipal Construction M41-10,*** Department of Transportation, P.O. Box 47408, Olympia, WA 98504-7408. www.wsdot.wa.gov/Publications/Manuals/M41-10.htm

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 s 37; 2011 c 27 s 1; 2009 c 495 s 1; 2007 c 343 s 11; 1993 c 492 s 489; 1992 c 34 s 4. Prior: 1989 1st ex.s. c 9 s 210; 1989 c 207 s 1; 1985 c 213 s 1; 1979 c 141 s 49; 1967 ex.s. c 102 s 9; 1965 c 8 s 43.20.050; prior: (i) 1901 c 116 s 1; 1891 c 98 s 2; RRS s 6001. (ii) 1921 c 7 s 58; RRS s 10816.]

Explanatory statement—2021 c 65: See note following RCW 53.54.030.

Effective date—2009 c 495: "Except for section 9 of this act, this act is necessary for the immediate preservation of the public peace, health, or safety, or support of the state government and its existing public institutions, and takes effect immediately [May 14, 2009]." [2009 c 495 s 17.]

Findings—1993 c 492: "The legislature finds that our health and financial security are jeopardized by our ever increasing demand for health care and by current health insurance and health system practices. Current health system practices encourage public demand for unneeded, ineffective, and sometimes dangerous health treatments. These practices often result in unaffordable cost increases that far exceed ordinary inflation for essential care. Current total health care expenditure rates should be sufficient to provide access to essential health care interventions to all within a reformed, efficient system.

The legislature finds that too many of our state's residents are without health insurance, that each year many individuals and families are forced into poverty because of serious illness, and that many must leave gainful employment to be eligible for publicly funded medical services. Additionally, thousands of citizens are at risk of losing adequate health insurance, have had insurance canceled recently, or cannot afford to renew existing coverage.

The legislature finds that businesses find it difficult to pay for health insurance and remain competitive in a global economy, and that individuals, the poor, and small businesses bear an inequitable health insurance burden.

The legislature finds that persons of color have significantly higher rates of mortality and poor health outcomes, and substantially lower numbers and percentages of persons covered by health insurance than the general population. It is intended that chapter 492, Laws of

1993 make provisions to address the special health care needs of these racial and ethnic populations in order to improve their health status.

The legislature finds that uncontrolled demand and expenditures for health care are eroding the ability of families, businesses, communities, and governments to invest in other enterprises that promote health, maintain independence, and ensure continued economic welfare. Housing, nutrition, education, and the environment are all diminished as we invest ever increasing shares of wealth in health care treatments.

The legislature finds that while immediate steps must be taken, a long-term plan of reform is also needed." [1993 c 492 s 101.]

Intent—1993 c 492: "(1) The legislature intends that state government policy stabilize health services costs, assure access to essential services for all residents, actively address the health care needs of persons of color, improve the public's health, and reduce unwarranted health services costs to preserve the viability of nonhealth care businesses.

(2) The legislature intends that:

(a) Total health services costs be stabilized and kept within rates of increase similar to the rates of personal income growth within a publicly regulated, private marketplace that preserves personal choice;

(b) State residents be enrolled in the certified health plan of their choice that meets state standards regarding affordability, accessibility, cost-effectiveness, and clinical efficaciousness;

(c) State residents be able to choose health services from the full range of health care providers, as defined in RCW 43.72.010(12), in a manner consistent with good health services management, quality assurance, and cost effectiveness;

(d) Individuals and businesses have the option to purchase any health services they may choose in addition to those included in the uniform benefits package or supplemental benefits;

(e) All state residents, businesses, employees, and government participate in payment for health services, with total costs to individuals on a sliding scale based on income to encourage efficient and appropriate utilization of services;

(f) These goals be accomplished within a reformed system using private service providers and facilities in a way that allows consumers to choose among competing plans operating within budget limits and other regulations that promote the public good; and

(g) A policy of coordinating the delivery, purchase, and provision of health services among the federal, state, local, and tribal governments be encouraged and accomplished by chapter 492, Laws of 1993.

(3) Accordingly, the legislature intends that chapter 492, Laws of 1993 provide both early implementation measures and a process for overall reform of the health services system." [1993 c 492 s 102.]

Short title—Savings—Reservation of legislative power—Effective dates—1993 c 492: See RCW 43.72.910 through 43.72.915.

Severability—1992 c 34: See note following RCW 69.07.170.

Effective date—Severability—1989 1st ex.s. c 9: See RCW 43.70.910 and 43.70.920.

Savings—1985 c 213: "This act shall not be construed as affecting any existing right acquired or liability or obligation incurred under the sections amended or repealed in this act or under any rule, regulation, or order adopted under those sections, nor as affecting any proceeding instituted under those sections." [1985 c 213 s 31.]

Effective date—1985 c 213: "This act is necessary for the immediate preservation of the public peace, health, and safety, the support of the state government and its existing public institutions, and shall take effect June 30, 1985." [1985 c 213 s 33.]

Severability—1967 ex.s. c 102: See note following RCW 43.70.130.

Rules and regulations—Visual and auditory screening of pupils: RCW 28A.210.020.

Health Impact Reviews: FY 2024 Update

Cait Lang-Perez (she/her), Miranda Calmjoy (she/they), Lindsay Herendeen (she/her)

August 7, 2024

Health Impact Reviews

- **Objective**, **nonpartisan**, **evidence-based analysis**
- Determine how a legislative or budgetary change may impact **health** and **equity**
- Prospective tool
- Requested by any Legislator or the Governor
- Can be requested for any bill topic
- Must be completed in 10 days during legislative session

(Authorizing law: RCW 43.20.285)

HIR Process

Review Bill

Determine how provisions in the bill may change status quo:

- Review the bill
- Interview agencies responsible for implementation

Explore Pathways

Explore potential connections to health:

- Conduct initial literature reviews
- Review public testimony and relevant documents
- Draft a logic model

Review Literature

Conduct specific reviews of literature to determine:

- How provisions may impact health
- Who is most likely to be impacted
- How the change may impact equity

Engage People

Talk with people who have content and context expertise to:

- Verify understanding of the bill
- Refine pathway to health and equity
- Consider impacts comprehensively

Types of Key Informant Engagement

Strength-of-Evidence Criteria

Ratings are based on criteria which consider:

- the amount of research
- appropriateness of study design
- study execution
- generalizability

VERY STRONG EVIDENCE

STRONG EVIDENCE

A FAIR AMOUNT OF EVIDENCE

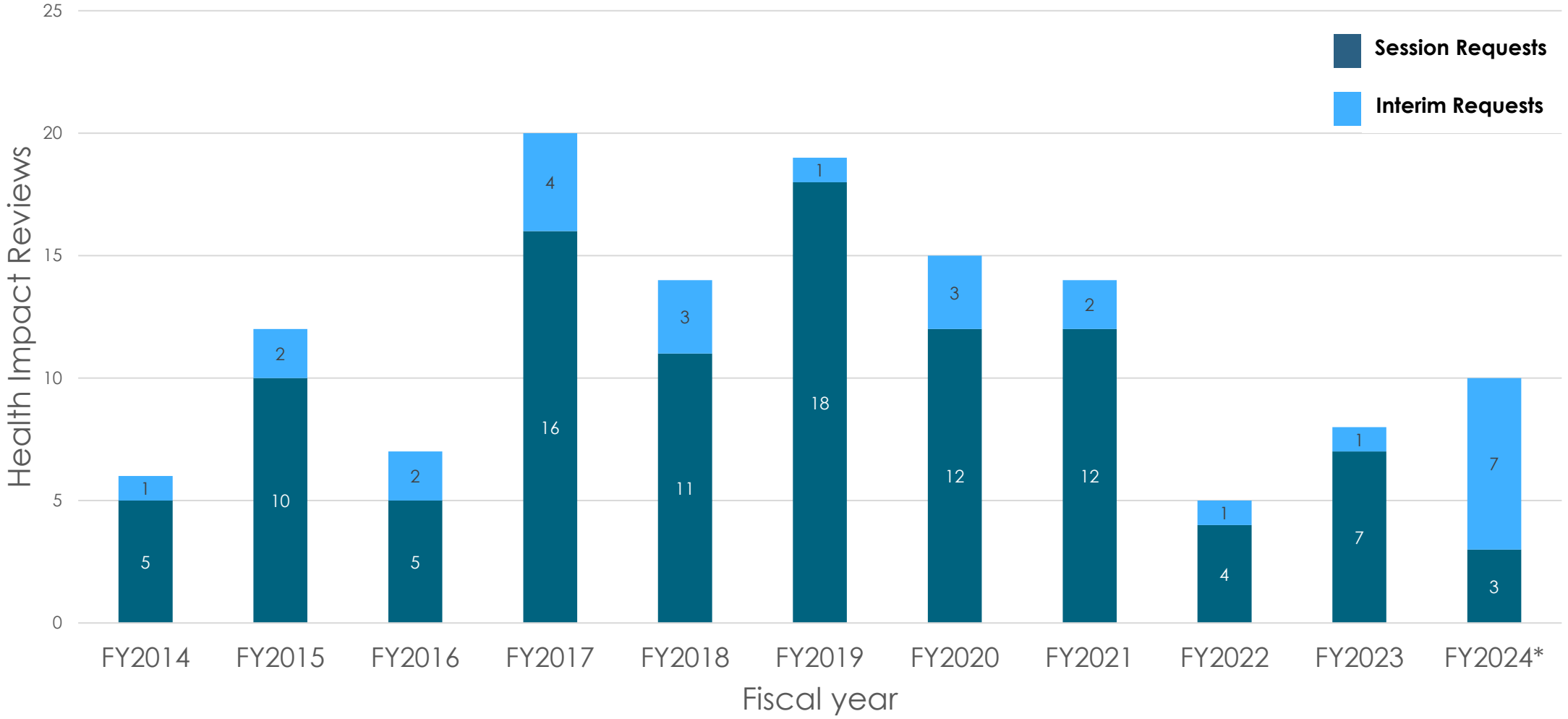
EXPERT OPINION

INFORMED ASSUMPTION

NOT WELL RESEARCHED

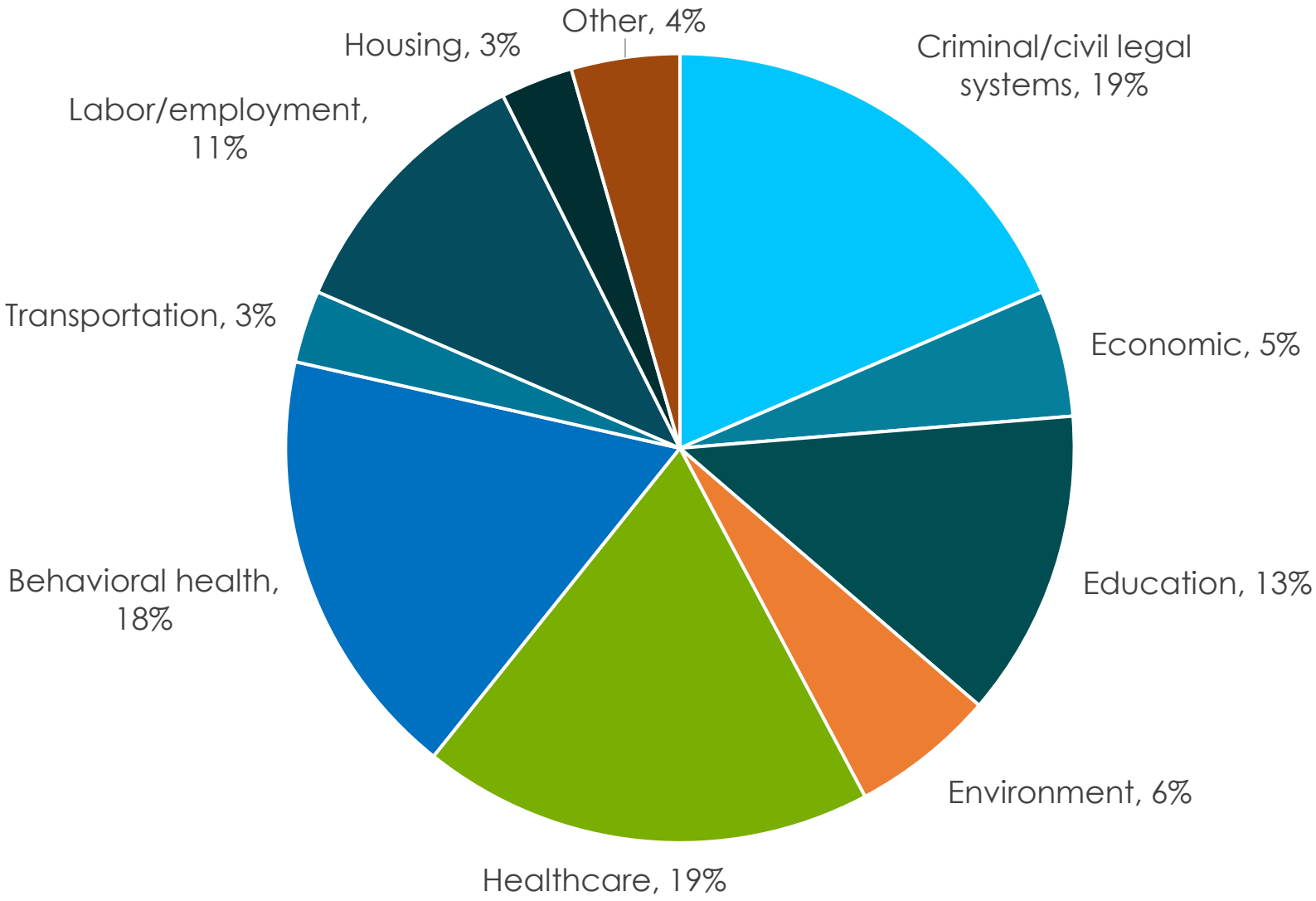
UNCLEAR

129 Completed HIRs



*Includes 1 HIR in progress

HIR Requests by Topic Area



2024 Legislative Session, By the Numbers

9 The **number of Health Impact Reviews** completed during Fiscal Year 2024.	**119** The **number of key informants** engaged during Fiscal Year 2024.
7 The **number of HIRs requested during the Interim,** the most requests ever made outside of Legislative Session in a single fiscal year.	**38%** The **percentage of HIR requests made by a first-time requester** during Fiscal Year 2024.
19% The **percentage of HIR requests received since 2014 that have been related to criminal/civil legal system-related topics**, tying it with healthcare as the most requested policy topic area.	**2** The **total number of HIR-related bills that passed** the Legislature in 2024 and were signed into law by the Governor.

How HIRs Inform Policy

Requesters have used HIRs to understand:

- The **evidence base** for a proposal
- If a bill will have the **intended impact**
- Potential **unintended consequences**
- **Equity implications**

Requesters have used HIR findings to:

- Talk with colleagues about a bill
- **Refine a policy**
- Discuss the bill on the floor
- Develop points for **budget negotiations**
- Inform members' votes

2024 Key Informant Interviewee Feedback

Survey Participants	
Role	**Total**
WA State Agency	**13**
Agency outside of WA	**2**
Researcher	**2**
Community-based organization	**1**
Individual, community member	**1**
Professional Association	**1**
Total	**20 (17%)**

Of key informants who responded…

- **95%** said participation with the HIR team was productive and meaningful
- **50%** said they used HIR findings

Ways HIR findings were used:

- Education
- Advocacy
- Dissemination

Most useful about HIR findings:

- Thorough distillation of information
- Findings provide a deep understanding of the policy topic and equity impacts, and a different way to think about the policy at hand
- The executive summary
- HIRs are objective, non-partisan, impartial

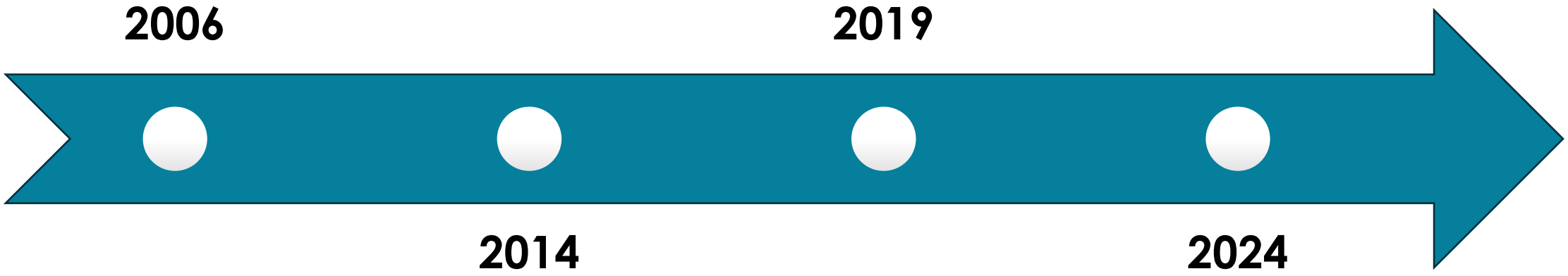
Work during the Interim

- Completing Health Impact Reviews.
- Holding outreach meetings with legislators and the Governor's policy advisors.
- Continuing to refine equity approach.
- Continuing to explore opportunities to provide compensation for key informants.
- Developing an outreach toolkit for Board and Council Members to use to discuss Health Impact Reviews.

Providing compensation for key informants

- Health Impact Reviews began including key informant interviews in 2018.
    - Since then, HIR requests have increased in number and complexity.
    - Key Informants have suggested compensation for their time.
- State Board of Health provided the opportunity for all eligible key informants to receive compensation for their time on HIRs in Fiscal Year 2024.
    - 12 eligible key informant interviewees received compensation.
    - Each received a $100 electronic gift card.

Revising the HIR Strength-of-Evidence criteria



Strength-of-Evidence Criteria

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- the amount of research
- appropriateness of study design
- study execution
- generalizability

VERY STRONG EVIDENCE

STRONG EVIDENCE

A FAIR AMOUNT OF EVIDENCE

EXPERT OPINION

INFORMED ASSUMPTION

NOT WELL RESEARCHED

UNCLEAR

Strength-of-Evidence: Multiple types of evidence

"Published" *Rated for association, design, execution, generalizability*	**Primary research** *Conducted by SBOH**; rated for association, design, execution, generalizability***

• Published literature
  ○ Quantitative
  ○ Qualitative
  ○ Reviews/meta-analyses
• Data (published, unpublished)
• Reports (published, unpublished)
	• Key informant interviews
	  ○ Washington State agencies
	  ○ Researchers
	  ○ Agencies in states outside of Washington State
	  ○ Professional associations
	  ○ Community organizations
	  ○ People with lived experience most likely to be directly impacted by the bill

From HIR Strength-of-Evidence Criteria_V12_2024, Appendix A

Strength-of-Evidence: Example considerations

Strength-of-Evidence ratings are based on criteria which consider:	Example of current considerations for rating published qualitative research	Example of draft considerations for rating information from key informants based on critical reflection of HIR staff methods
Association	Is the evidence generally in alignment or conflicting?	Is information from key informants generally in alignment or conflicting?
Appropriateness of study design	Methodology (e.g., interviews, focus groups)	Did staff identify key informants reflecting various communities, experiences, perspectives, locations?
Study execution	Steps taken to verify accuracy of themes with community	Did staff provide opportunity for key informants to review content?
Generalizability	Do study populations include or reflect Washington State or groups impacted by the bill?	Does information from key informants generally reflect all sides of a policy topic?

Strength-of-Evidence: Next steps

- **Now: Participate in a feedback session with the HIR Team**
    - Learn more about the draft process for information from key informants to contribute to the Strength-of-Evidence.
    - Ask additional questions.
    - Provide feedback about HIR methodology.
- **July 2024-June 2025: Implement a draft process and continue to refine it**
- **July 2025: Implement a final process**

Contact the HIR Team

Miranda Calmjoy (she/they)
Lindsay Herendeen (she/her)
Cait Lang-Perez (she/her)

hir@sboh.wa.gov
360-819-0750

Completed Health Impact Reviews can be found on the Washington State Board of Health website:
https://sboh.wa.gov/health-impact-reviews

THANK YOU

WASHINGTON STATE BOARD OF HEALTH

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

WASHINGTON STATE BOARD OF HEALTH

Date: August 7, 2024

To: Washington State Board of Health Members

From: Kelly Oshiro, Board Member

Subject: Petition – Chapter 246-650 WAC, Newborn Screening, Request to add Wilson's Disease

Background and Summary:

The Administrative Procedures Act (RCW 34.05.330) allows any person to petition a state agency to request 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 July 26, 2024, the Washington State Board of Health (Board) received a rulemaking petition requesting to amend chapter 246-650 WAC to add Wilson's Disease as a condition for newborn screening. The petition states that early diagnosis of Wilson's Disease and pre-emptive treatment prior to symptom onset can prevent life-threatening complications.

Wilson's Disease is a rare and inherited metabolic disease that prevents the body from properly eliminating copper, causing it to accumulate in body tissues, especially the liver, brain and corneas of the eyes. Wilson's Disease is caused by two copies of an abnormal gene (*ATP7B*) that would normally help transport copper out of liver cells, allowing copper to be eliminated from body.[2,5] Excess copper then builds up in the liver and eventually in the blood and other organ systems.[3] Too much copper is toxic to the body's tissues and can lead to serious damage to the liver, nervous system, and other organ systems.[3]

Symptoms of Wilson's Disease vary widely and can occur anytime after age 3 through the age of 70.[1,3] People with Wilson's Disease may develop symptoms related to liver dysfunction initially, such as yellowing of skin and eyes, swelling, vomiting, and fatigue. Other people with Wilson's Disease may only develop brain-related symptoms such as behavioral changes, difficulty swallowing, muscle rigidity, difficulty with speech, and lack of coordination.[2,3]

Wilson's Disease is not being screened by any other state currently and has yet to be added to the Federal Recommended Uniform Screening Panel (RUSP). However, Washington's newborn screening lab has been working on a pilot project in partnership with Key Proteo in which 50,000 de-identified samples from residual newborn screening

(continued on the next page)

specimens have been tested to demonstrate the feasibility of a population-based screening test for Wilson's Disease.[4]

The Board has the authority under RCW 70.83.050 to adopt rules for screening Washington-born infants for hereditary conditions. WAC 246-650-010 defines the conditions, and WAC 246-650-020 lists the conditions on the state's required newborn screening panel.

The Board has a process it follows when considering new conditions to include in the state's newborn screening panel. To determine which conditions to include in the panel, the Board may convene an advisory committee to evaluate candidate conditions using guiding principles and an established set of criteria. Before an advisory committee is convened, there should be sufficient scientific evidence available to apply the Board's criteria for inclusion. This may require a preliminary review.

I have invited Kelly Kramer, Board staff, and John Thompson, Director of the Department of Health's Newborn Screening Program, to provide an overview of the Board's process for adding a condition to the panel, the petition request, and a brief overview of Wilson's Disease

Recommended Board Actions:

The Board may wish to consider one of the following motions:

The Board declines the petition for rulemaking to add Wilson's Disease as a condition for newborn screening in Chapter 246-650 WAC, and directs staff to work with the Department of Health to perform a preliminary review of the condition for inclusion in WAC 246-650-020 and then report back to the Board so the Board can determine whether to establish a technical advisory committee to evaluate Wilson's Disease against the Board's criteria for adding conditions to the newborn screening rule.

OR

The Board declines the petition for rulemaking to add Wilson's Disease as a condition for newborn screening in Chapter 246-650 WAC, and directs staff to work with the Department of Health to move forward with convening a technical advisory committee to evaluate Wilson's Disease using the Board's process and criteria to evaluate conditions for inclusion in WAC 246-650-020 and then make a recommendation to the Board.

OR

The Board accepts the petition for rulemaking to amend Chapter 246-650 WAC to add Wilson's Disease as a condition for newborn screening. The Board directs staff to notify the requestor of its decision and to file a CR-101, Preproposal of Inquiry, under its authority in RCW 70.83.050.

(continued on the next page)

Staff
Kelly Kramer

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

1. National Institutes of Health National Library of Medicine. Wilson Disease: GeneReviews. Published January 2023. Accessed July 22, 2024. https://www.ncbi.nlm.nih.gov/books/NBK1512/#wilson
2. National Organization for Rare Disorders. Wilson Dease. Published March 2018. Accessed July 22, 2024. https://rarediseases.org/rare-diseases/wilson-disease
3. National Institutes of Health National Library of Medicine. Wilson Disease: StatPearls . Published August 2023. Accessed July 22, 2024. https://www.ncbi.nlm.nih.gov/books/NBK441990/
4. Key Proteo. Key Proteo Pilots Newborn Screening in Washington State. Published Mar 2022. Accessed July 22, 2024.
5. National Institutes of Health National Library of Medicine. Epidemiology, diagnosis, and treatment of Wilson's disease. Published November 2017. Accessed July 24, 2024. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5735277/

PETITION FOR ADOPTION, AMENDMENT, OR REPEAL OF A STATE ADMINISTRATIVE RULE

Print Form

In accordance with RCW 34.05.330, the Office of Financial Management (OFM) created this form for individuals or groups who wish to petition a state agency or institution of higher education to adopt, amend, or repeal an administrative rule. You may use this form to submit your request. You also may contact agencies using other formats, such as a letter or email.

The agency or institution will give full consideration to your petition and will respond to you within 60 days of receiving your petition. For more information on the rule petition process, see Chapter 82-05 of the Washington Administrative Code (WAC) at http://apps.leg.wa.gov/wac/default.aspx?cite=82-05.

CONTACT INFORMATION *(please type or print)*

Petitioner's Name: Dr. Sihoun Hahn, M.D, Ph.D.

Name of Organization: Key Proteo, Inc.

Mailing Address: 720 Broadway

City: Seattle State: WA Zip Code: 98122

Telephone: 206-399-7515 Email: steve.hahn@keyproteo.com

COMPLETING AND SENDING PETITION FORM

- Check all of the boxes that apply.
- Provide relevant examples.
- Include suggested language for a rule, if possible.
- Attach additional pages, if needed.
- Send your petition to the agency with authority to adopt or administer the rule. Here is a list of agencies and their rules coordinators: http://www.leg.wa.gov/CodeReviser/Documents/RClist.htm.

INFORMATION ON RULE PETITION

Agency responsible for adopting or administering the rule: Washington State Board of Health

☒ **1. NEW RULE - I am requesting the agency to adopt a new rule.**

☒ The subject (or purpose) of this rule is: Institute newborn screening of Wilson Disease for Washington State.

☒ The rule is needed because: NBS and early diagnosis for Wilson Disease is currently an unmet need.

☒ The new rule would affect the following people or groups: Newborns with Wilson Disease; Parents and families negatively impacted by association.

☐ **2. AMEND RULE - I am requesting the agency to change an existing rule.**

List rule number (WAC), if known: _____

☐ I am requesting the following change: _____

☐ This change is needed because: _____

☐ The effect of this rule change will be: _____

☐ The rule is not clearly or simply stated: _____

☐ **3. REPEAL RULE - I am requesting the agency to eliminate an existing rule.**

List rule number (WAC), if known: _____

(Check one or more boxes)

☐ It does not do what it was intended to do.

☐ It is no longer needed because: _____

☐ It imposes unreasonable costs: _____

☐ The agency has no authority to make this rule: _____

☐ It is applied differently to public and private parties: _____

☐ It conflicts with another federal, state, or local law or rule. List conflicting law or rule, if known: _____

☐ It duplicates another federal, state or local law or rule. List duplicate law or rule, if known: _____

☐ Other (please explain): _____

[Supplemental Documents]

1. Criteria Justification
2. Petition Letter
3. WA State Pilot Study Overview Slide deck
4. NBS Kit validation for FDA submission Slide deck
    a. IFU
    b. Analytical & Clinical Validation data for FDA submission
5. References
    a. APHL abstract 2024
    b. Ann. N.Y. Acad. Sci: Population screening for Wilson disease
    c. Clinical and Translational Perspectives on Wilson disease. Edited by Nanda Kerkar and Eve A. Roberts
        i. Chapter 26. Population screening for Wilson disease
        ii. Chapter 17. Wilson disease in Infancy through Adolescence
    d. Handbook of Clinical Neurology. Wilson Disease Edited by Anna Czlonkowska and Michael L. SChilsky
        i. Chapter 3.The genetics of Wilson Disease
    e. J. Proteome Res. 2017: Quantification of ATP7B Protein in Dried Blood Spots by Peptide Immuno-SRM as a Potential Screen for Wilson's Disease
    f. Gastroenterology 2021: Direct Measurement of ATP7B Peptides Is Highly Effective in the Diagnosis of Wilson Disease
        i. Editorial comments: Expanding the Diagnostic Toolkit of Wilson Disease with ATP7B Peptides
    g. JPGN 2020: Management of Wilson Disease Diagnosed in Infancy: An Appraisal of Available Experience to Generate Discussion
    h. GeneReviews: Wilson disease: Wilson Disease - GeneReviews® - NCBI Bookshelf (nih.gov)
6. Letter of Support
    a. Wilson Disease Association, U.S.A.
    b. Wilson Disease Association, Spain
    c. Experts
        i. Dr. Karl Weiss in Germany
        ii. Dr. Michael Schilsky at Yale University
    d. Family members:
        i. Letters from US

- Alice William Family
- Christopher and Rachel Johnson
- Nora Closser
- Erin Brooks
- Dr. Kirk Vestal
- Thomas Sandall
- Marilee and Gary Wolter
- Maxine and Michael Bonn
- Janet Laubgross

ii. Letter from Poland

- Anna Aniol

THREE GUIDING PRINCIPLES: Three guiding principles govern all aspects of the evaluation of a candidate condition for possible inclusion in the NBS panel.
• Decision to add a screening test should be driven by evidence. For example, test reliability and available treatment have been scientifically evaluated, and those treatments can improve health outcomes for affected children.
• All children who screen positive should have reasonable access to diagnostic and treatment services.
• Benefits of screening for the disease/condition should outweigh harm to families, children and society

[Criteria for Testing]

Available Screening Technology: Sensitive, specific and timely tests are available that can be adapted to mass screening.

The base technology is tandem mass spectrometry (LC-MS/MS), which is already being used for NBS conditions such as LSD (Lysosomal Storage Disorders) and XLD (X-linked Adrenoleukodystrophy) in Washington State. Quantification of ATP7B protein using LC-MS/MS utilizes trypsin to digest protein and quantify the resulting signature surrogate peptides of the target protein, ATP7B. It has been well demonstrated that the assay can provide the diagnosis and newborn screening of Wilson disease with sensitivity and specificity both above 92%. To date, over 25,000 newborns have been successfully tested in Washington State. A total of 4 presumptive positive cases have been detected. Two of them were false positive (no variants or carriers) and two of them were likely true positive. The sample to sample run time is less than 3 minutes which is appropriate for high throughput analysis.

Diagnostic Testing and Treatment available: Accurate diagnostic tests, medical expertise, and effective treatment are available for evaluation and care of all infants identified with the condition.

Biochemical and genetic testing are widely available to confirm the diagnosis. Treatment includes a copper restricted diet, zinc or chelating agents such as penicillamin or syprine. Gene therapy is currently under clinical trial. Liver transplantation is required in advanced patients with decompensated liver cirrhosis and acute liver failure. The cost for the maintenance treatment with zinc is a penny a day ($.01/day).

Prevention Potential and Medical Rationale: The newborn identification of the condition allows for early diagnosis and intervention. Important considerations:

• *There is sufficient time between birth and onset of irreversible harm to allow for diagnosis and intervention.*
The newborn identification of the condition allows for early diagnosis and preemptive intervention. There has been a strong need for NBS of Wilson disease (WD), which progresses to an irreversible multi-systemic disease. The patient mortality rate is 100% if left untreated. The copper accumulation starts right after birth but as the disease progress is slow, WD is usually diagnosed in adolescents after significant complications occur. Nonetheless, it is now well established that Wilson disease can present with clinical disease, mainly hepatic, in very young children <5 years old. Since the first detailed and well-documented report of a 3-year-old with cirrhosis, numerous reports have appeared providing additional evidence to institute NBS for Wilson disease.

Unlike other metabolic disorders, infants with Wilson disease do not present with any acute deterioration in the first few years of life. It is generally recommended to start the zinc maintenance

treatment during 12-24 months of life and monitor the labs every 6 months or a year. There is ample time to allow for diagnosis, counseling and initiation of treatment.

• *The benefits of detecting and treating early onset forms of the condition (within one year of life) balance the impact of detecting late onset forms of the condition.*
The symptoms of Wilson disease are frequently nonspecific. Approximately one-third of children with clinically evident WD are present with cirrhosis. Acute liver failure poses significant diagnostic problems. Although neurologic Wilson disease is less common in pediatric patients with Wilson disease, it has been reported in children as young as 6 years old. Initial neurological symptoms could be extremely nonspecific further delaying the diagnosis and appropriate management. Treatment for neurological Wilson disease is challenging as they could develop further deterioration with chelating agents.

The cost for zinc maintenance treatment is only a penny a day ($.01/day) when started before any complications occurred and the patients live a completely normal life. The benefits of detecting and treating the condition before symptoms occurred have been well described in many literatures.

• Newborn screening is not appropriate for conditions that only present in adulthood.
As many genetic disorders have a spectrum of clinical severity, some patients with Wilson disease may not have the symptoms until later in life. However, all those biochemically and genetically confirmed Wilson disease patients should start the treatment regardless of the symptoms or lab results at the time of the diagnosis.

Public Health Rationale: Nature of the condition justifies population-based screening rather than risk-based screening of other approaches.

Effective treatment for Wilson disease is widely available and affordable, highlighting the importance of early detection and intervention. These treatments are highly effective in preventing the downstream of morbidity and early mortality associated with Wilson disease. While there are effective treatments available, it is unfortunate that most patients come to the clinic after developing serious complications. Furthermore, many patients suffer from delayed diagnosis due to the nature of nonspecific and considerably variable clinical presentations. Half of the affected patients with neurological symptoms may develop permanent neurological damage. Patients with chronic liver disease may progress to cirrhosis and hepatocellular carcinoma.

Importantly, patients with Wilson disease can live a completely normal life when the treatment is started early in life. Early initiation of therapy before the onset of symptoms makes a critical difference in their outcome. The cost for zinc treatment is nominal compared to other cell based or gene therapies. The best clinical outcome could be achieved only through NBS.

Cost-benefit/Cost-effectiveness: The outcome outweighs the costs of screening. All outcomes, both positive and negative, need to be considered in the analysis. Important considerations to be included in economic analyses include:

Newborn screening of Wilson disease will change the diagnostic course and clinical outcome of WD patients. As many WD patients undergo liver transplantation, this opportunity for early initiation of therapy before the onset of symptoms will significantly make a critical and huge difference in their long-term clinical outcome. Ultimately, we anticipate there will be savings that come from converting patients who would otherwise be consumers of high-intensity medical care, long-term convalescent care, and disability resources, to functional individuals capable of holding jobs and contributing to the tax base.

- *The prevalence of the condition among newborns.*

The prevalence of WD is approximately 1 in 30,000 newborns, with a carrier frequency of 1 in 90 (higher in certain populations). However, regional variations exist. Costa Rica, Sardinia, the Canary Island and Crete have all reported to have increased incidence.

• *The positive and negative predictive values of the screening and diagnostic tests.*

ATP7B 887 analysis was found to have a sensitivity of 91.2%, specificity of 98.1%, positive predictive value of 98.0%, and a negative predictive value of 91.5%. ATP7B 1056 showed a positive predictive value of 96.1% and negative predictive value of 91.3%.

• *Variability of clinical presentation by those who have the condition.*

Wilson disease presents a variety of symptoms depending on which organs are more affected. Wilson disease can lead to hepatic failure and/or severe neurological disability and ultimately death if untreated.

• *The impact of ambiguous results. For example, the emotional and economic impact on the family and medical system.*

The current pilot study in Washington State indicates the false positive rate is extremely low compared to the other conventional newborn screening methods.

• *Adverse effects or unintended consequences of screening.*

As with other screening programs, there could be false negative cases from this screening.

720 Broadway
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KEY PROTEO

To: Washington State Department of Health
Board of Directors

From: Sihoun Hahn, MD, PhD
Professor of Pediatrics, University of Washington
Seattle Children's Hospital
Founder and CMO, Key Proteo, Inc

Re: Letter from Petitioner

Date: July 26, 2024

Dear Washington State Department of Health Board of Directors,

On behalf of Key Proteo, I am writing to request your review of our Petition for Rulemaking to adopt a new administrative rule to screen for Wilson Disease (WD). Within our petition package you will find evidence to support Washington States Qualifying Assumption, evidence to support the Three Guiding Principles, as well as evidence to support the 5 Criteria used to evaluate conditions for possible inclusion in the newborn screening panel.

Wilson Disease is an autosomal recessive disease of copper metabolism caused by pathogenic mutations in the *ATP7B* gene, a copper-transporting ATPase. About 1 out of 30,000 individuals are affected by WD. Early diagnosis and pre-emptive therapy before the manifestation of symptoms are crucial for successful clinical outcomes that can lead to a completely normal life with a cost of a penny a day ($.01/day) with zinc treatment. Without early intervention, individuals can develop significant life-threatening complications, including liver cirrhosis, acute kidney failure, and brain and nerve damage. Key Proteo's Immuno-Selected Reaction Monitoring (SRM) platform is a novel proteomic screening solution that uniquely delivers rapid, scalable, and cost-effective detection of extremely low abundance peptide biomarkers in newborn dried blood spot specimens with high accuracy, effectiveness, and efficiency via familiar mass spectrometry workflows currently being used in the Washington State Newborn Screening Laboratory. By quantifying the ATP7B peptide, Key Proteo's technology provides accurate results to identify affected patients allowing them to be treated clinically leading to healthier outcomes. We have been successfully running a pilot study in collaboration with WA State DOH Newborn Screening Laboratory (Director Dr. John Thompson) for over two years screening ~25,000 newborn babies for Wilson disease. The progress report is included in the supplemental package.

Our petition package includes letters of support from the Wilson Disease Association (US), Wilson Disease Association (Spain), experts in the field such as Drs. Karl Heinz Weiss and Michael Schilsky, and families from the U.S., and Poland providing additional support as well as sharing their stories. We've also included evidence to support the need to adopt this test including validation data, Instructions for Use, criteria justification, as well as literature references.

It is our immense hope that Washington State will adopt and require this test for all newborns screened in the newborn screening program which will be the first model to all other states and the world. Together we can provide the community with early diagnosis, healthier outcomes, and relieve the heavy

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KEY PROTEO

burden these families carry when confronted with unexpected but life-saving decisions due to late diagnosis.

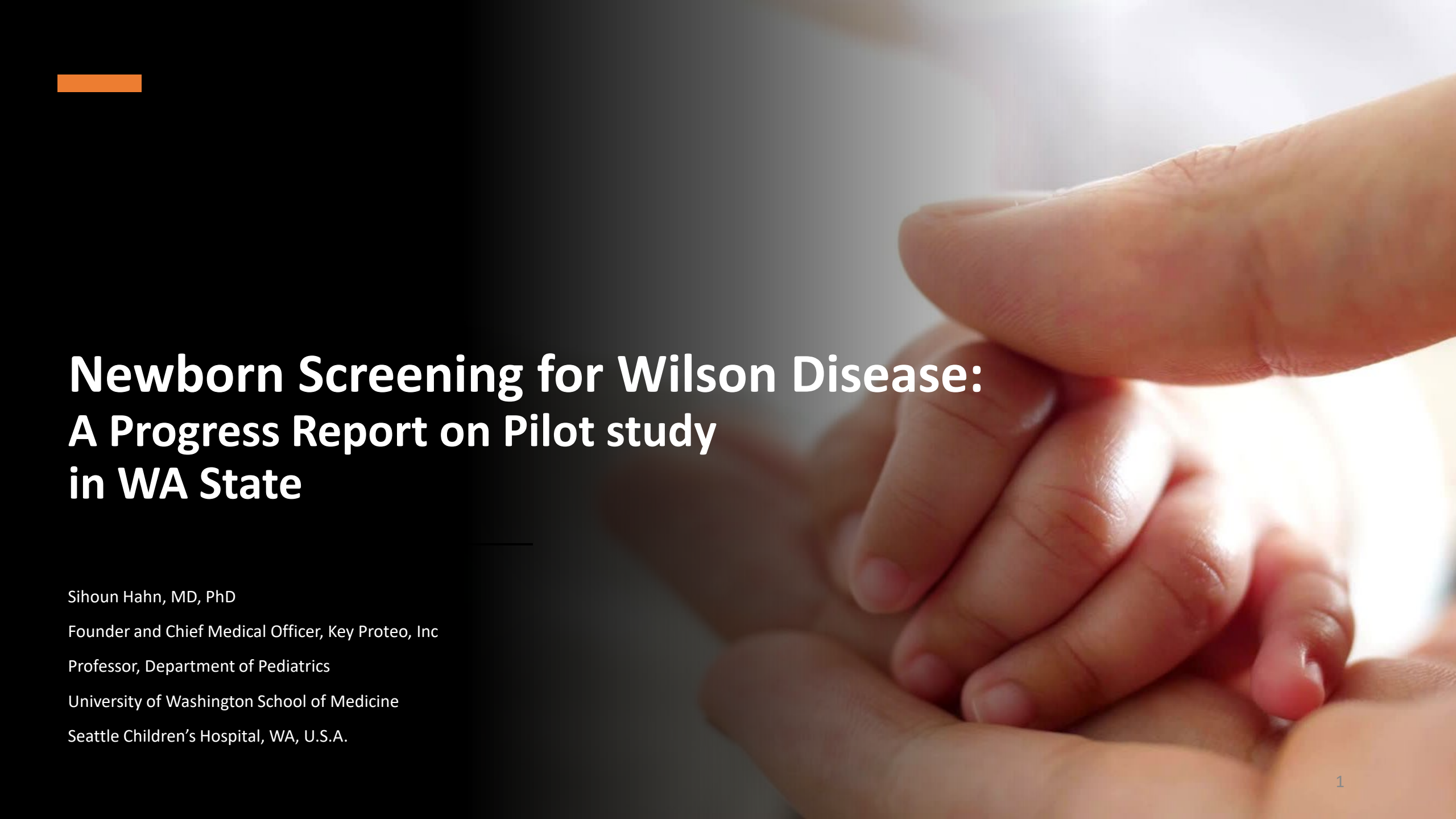
We appreciate your consideration and look forward to your review. Please do not hesitate to reach out with any questions whatsoever.

Sincerely,

Sihoun Hahn

Sihoun Hahn, MD, PhD
Director, Center of Excellence for Wilson disease
Seattle Children's Hospital
Founder and CMO, Key Proteo, Inc

Newborn Screening for Wilson Disease:
A Progress Report on Pilot study in WA State



Sihoun Hahn, MD, PhD

Founder and Chief Medical Officer, Key Proteo, Inc

Professor, Department of Pediatrics

University of Washington School of Medicine

Seattle Children's Hospital, WA, U.S.A.

FINANCIAL DISCLOSURES

Sihoun Hahn, MD, PhD, is a member of the Seattle Children's Hospital workforce and is serving as Chief Medical Officer of Key Proteo, Inc. He is an inventor of intellectual property that has been licensed to Key Proteo, Inc. Dr. Hahn is the founder of Key Proteo, Inc. and has ownership equity interests in the company.

Topics to discuss

- Background of Wilson disease
- Proof of evidence study on a large-scale patient cohort
- Manufactured kit validation for public health transition
- Pilot study in WA state

Background of Wilson Disease

Wilson Disease

- Autosomal Recessive Copper Transport Disorder
- Incidence: ~1/30,000
- Early diagnosis and pre-emptive treatment can lead to a normal quality of life

Dr. Samuel A. K. Wilson (1878-1937)

- Progressive, fatal disease if untreated
- ***ATP7B* gene**: ATPase, copper transporter
  - >1700 variants (Varsome)
  - p.H1069Q : ~35-45% in European origin
  - p.R778L: ~30% in Far East Asian origin

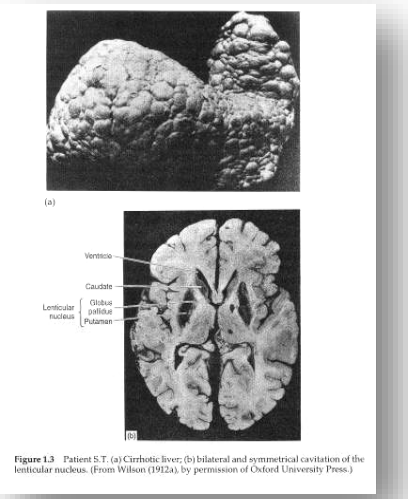


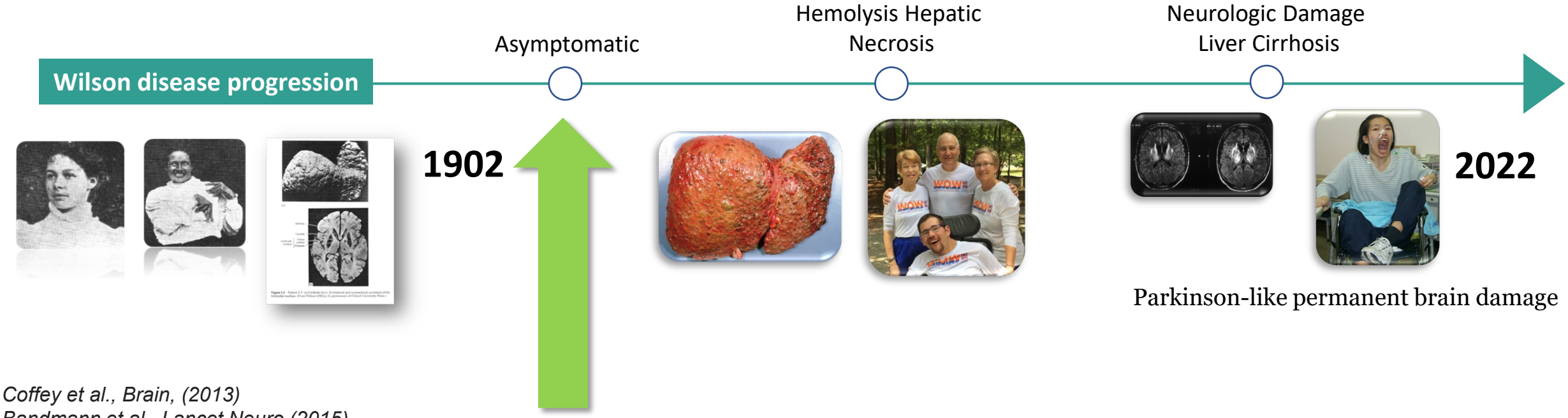
Figure 1.3 Patient S.T. (a) Cirrhotic liver; (b) bilateral and symmetrical cavitation of the lenticular nucleus. (From Wilson (1912a), by permission of Oxford University Press.)

Hoogenraad TU, Wilson's disease, Intermed Medical Publishers

- Diagnosis: high copper in the liver/urine, low serum ceruloplasmin, KF ring in the cornea
- Treatment: Chelators (Trientine), Zinc salts, Gene therapy under trial
- ***Immuno-SRM analysis is promising for NBS***
  - ***Majority of mutations results in markedly decreased level of ATP7B protein due to enhanced degradation, absence or decay of mRNA*** *(Hepatology, 2009; Proc Natl Acad Sci U S A, 1998; Gastroenterology, 2007; Curr Issues Mol Biol, 2001; Proteins, 2008; Genetics, 1998; Blood, 2005; Mol Genet Metab, 2005; BMC Gastroenterol, 2010; Nat Genet, 2004; Annu Rev Biochem, 2007)*

With early intervention, we can change the clinical course

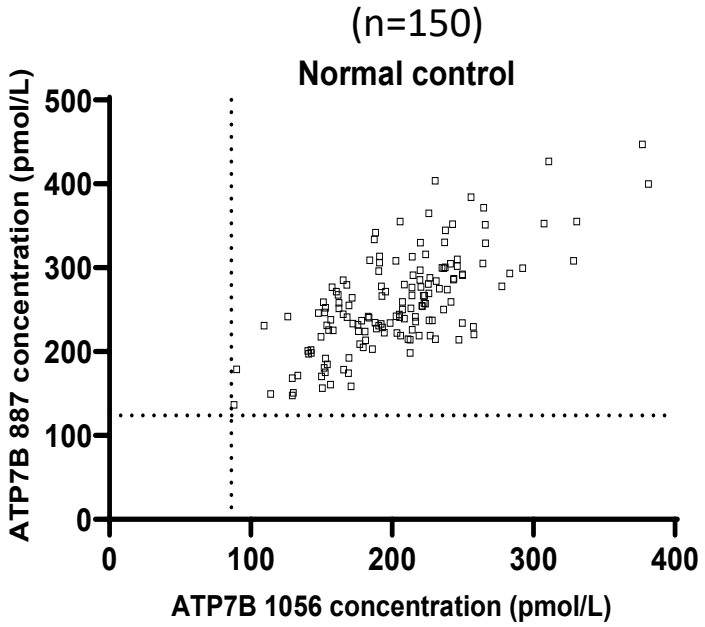
- Unrecognized in a substantial portion of affected individuals [1-2]
  (At least half of patient with WD are **never diagnosed** and die of untreated disease)
- Late diagnosis is the most common cause of death [3]
- Early diagnosis improves clinical outcome [4]
- Currently, there is no screening method while effective treatments are available

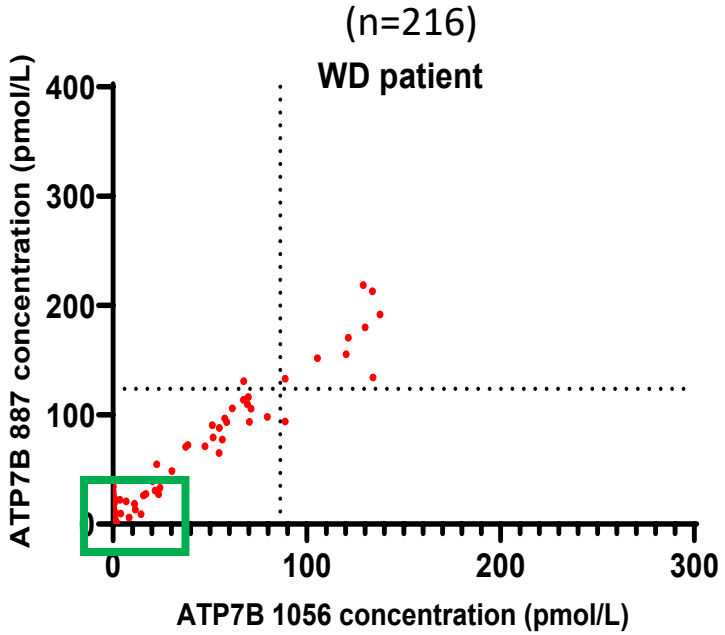


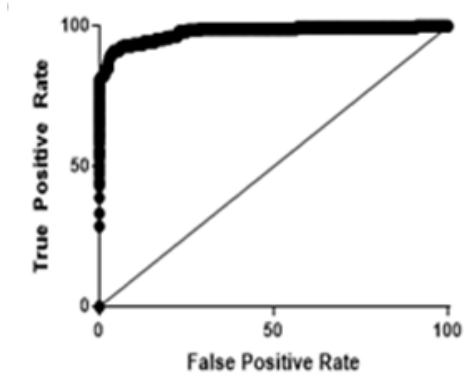
1. *Coffey et al., Brain, (2013)*
2. *Bandmann et al., Lancet Neuro (2015)*
3. *Walshe . Mov Disord. (2007)*
4. *Beinhardt et al, Clini Gastroenterol Hepatol (2014)*

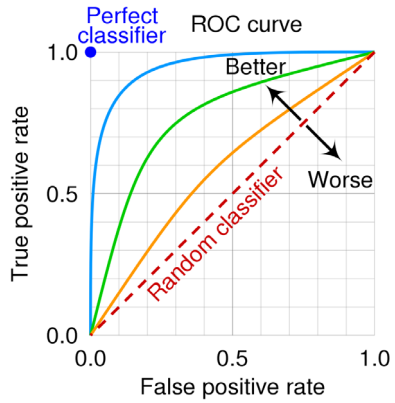
Proof-of-Evidence Study on a Large-Scale Patient Cohort

Wilson Disease: Most patients are deficient in ATP7B peptides







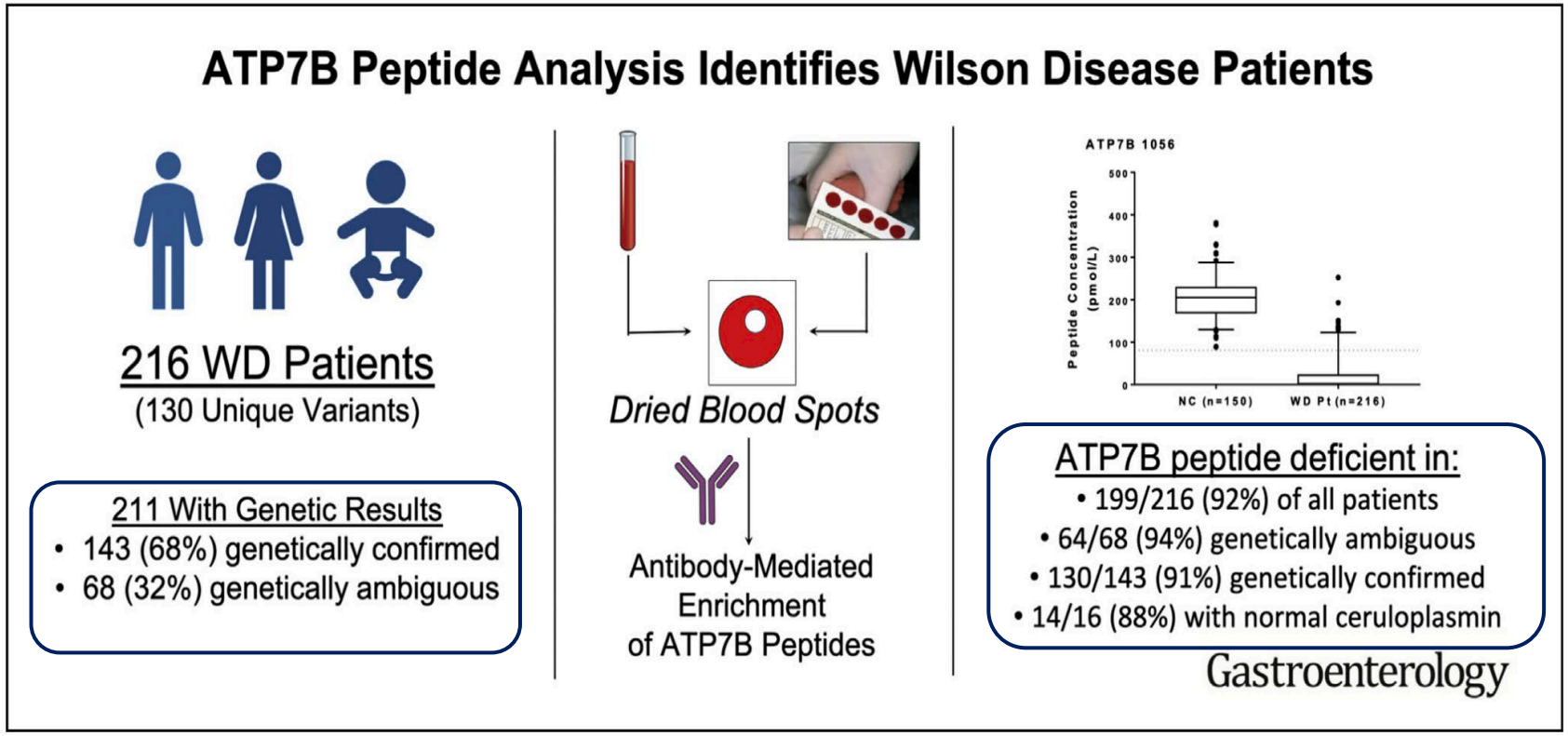


- **Large Retrospective Study**
  - 216 WD patients, 48 carriers,150 healthy controls
  - ATP7B peptides enriched from DBS
- 199 / 216 patients (92.1%) had at least 1 ATP7B peptide below diagnostic cutoff.
  - ~80% of patients had ATP7B < 32 pmol/L and <56 pmol/L for ATP7B 1056 and ATP7B 887 respectively
- ROC curve shows AUC 0.98, sensitivity 91.2%, specificity 98.1%, PPV 98.0% and NPV 91.5%

Collins C.J. et al. Gastroenterology. (2021)

Wilson Disease: Direct measurement of ATP7B peptides reduces clinical ambiguity

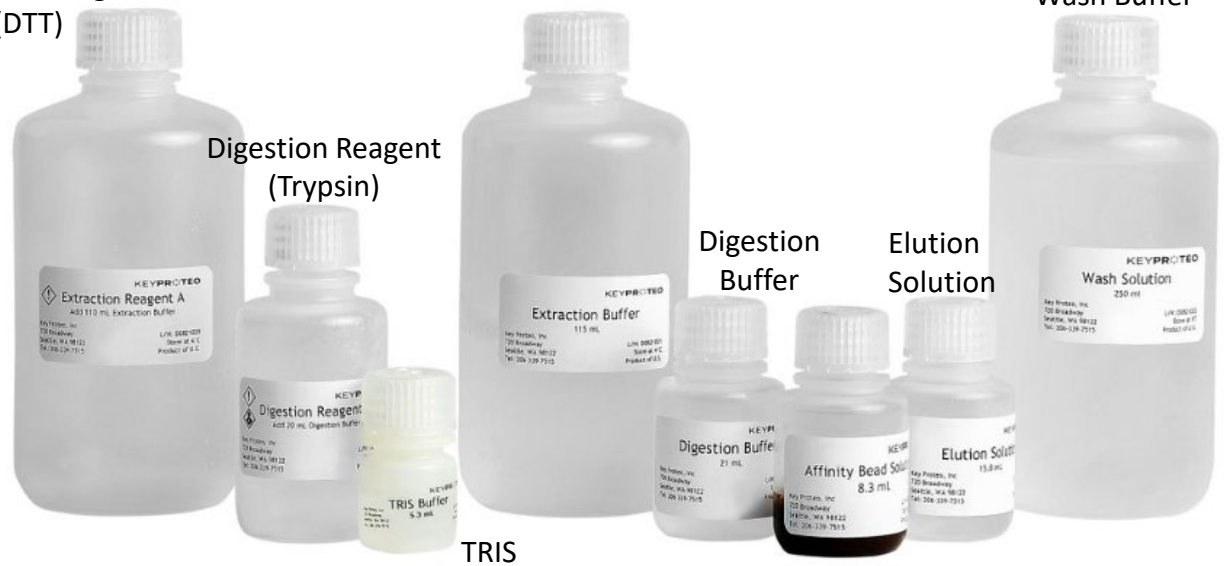
- *Collins C.J. et al. Gastroenterology. 2021, 160(7):2367-2382*
- *Editorial comment. Expanding the Diagnostic Toolkit of Wilson Disease with ATP7B Peptides*



Manufactured Kit Validation for Public Health Transition

Public Health Transition: Prototype 4-plex Screening Reagents

- Prototype reagents for assay performance have been manufactured at GMO facility
- Affinity beads mixed into a single solution
- Internal Standard Peptides lyophilized into 96-well plates
- DBS Control Cards
  - 3 levels of DBS spots
  - Negative, Low, High

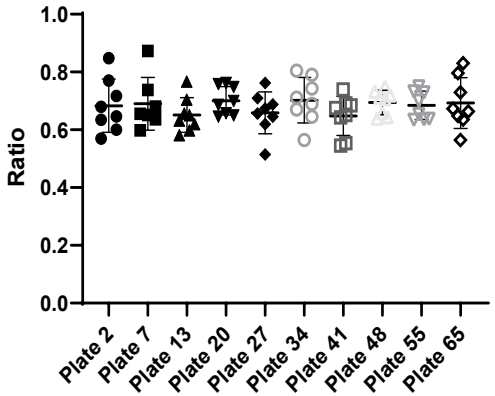


Internal Standard Plates

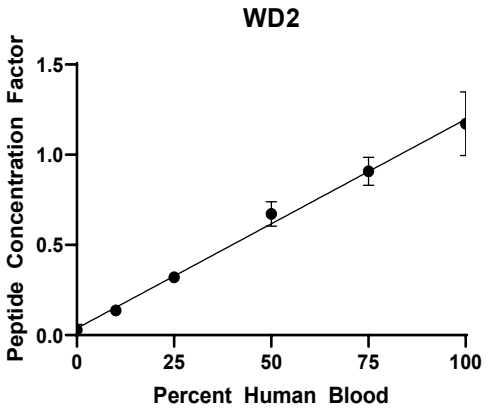
Control Cards

Prototype reagents are not FDA approved and currently intended for investigational use only

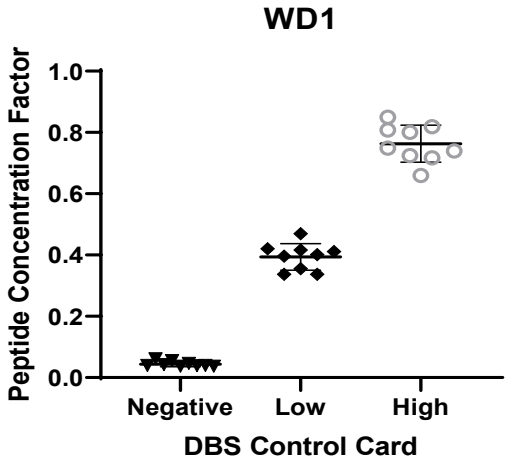
Performance: *Full assay performance and DBS control cards*



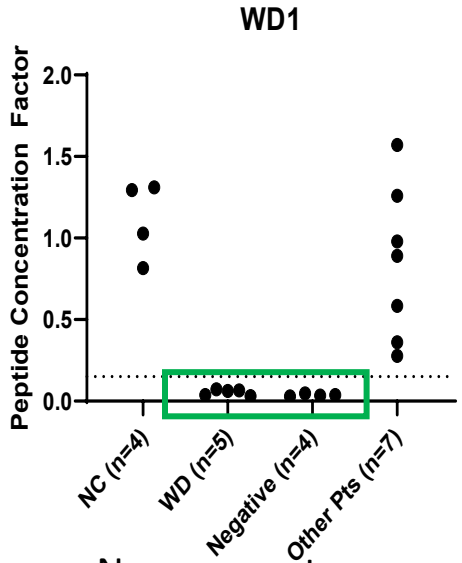
- *IS Plates ensured consistent peptide measurements and maintain consistent cutoff*
  - No dissolution/dilution of IS stock



- *Linear response from decreasing percentage of human blood.*
  - 0, 10, 25, 50, 75 and 100% human



- *DBS control cards perform as expected.*
  - Linear change in measured concentration



- New reagents, workflows, and analysis positively identify blinded patient samples.

Pilot Study in WA State

WA State Pilot Study

In Q1 2022, a large-scale pilot study began in WA State

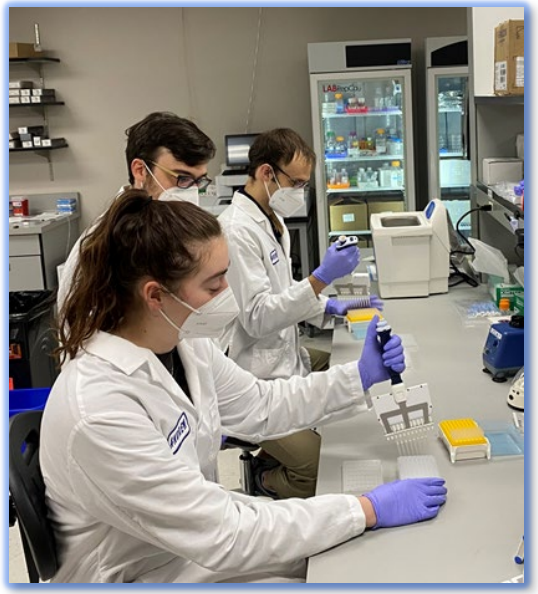
- In conjunction with the ***WA State Public Health Newborn Screening Laboratory***
- De-identified newborn DBS collected through routine course

Multiplexed screening utilizing the Neo-WA kit reagents

- Screening for WD, WAS, XLA, and ADA Deficiency
- 50,000-75,000 newborn samples targeted

Samples below initial cut-offs are sequenced for confirmation

WA PHL beginning KP Pilot



Analyzing KP Pilot Samples

WA Pilot Study of Newborn Screening:
Cut-off as of 7/2024

- Initially, the ionKey Microfluidic Separation Chromatography was used, which was later changed to more affordable low-flow ESI probe assembly fitted with a narrow bore capillary suitable for use with flow rates from 5 µL/min to 100 µL/min

- *Ionkey (plates 1-166)*

	ATP7B 887	ATP7B 1056
MEDIAN	359.8	292.4
SD	149.7	99.2
CV	41.6	33.9
Number	14,607	14,607
CUTOFF	68.4	55.5

- *ESI Low Flow (plates 179-current)*

	ATP7B 887	ATP7B 1056
MEDIAN	272.0	270.2
SD	98.1	90.3
CV	36.1	33.4
Number	10,139	10,141
CUTOFF	76.2	75.7

*cutoff is tentative as for now

WA Pilot Study of Newborn Screening: *Demographics*

- A total of ~25,000 newborns have been analyzed to date (7/2024).

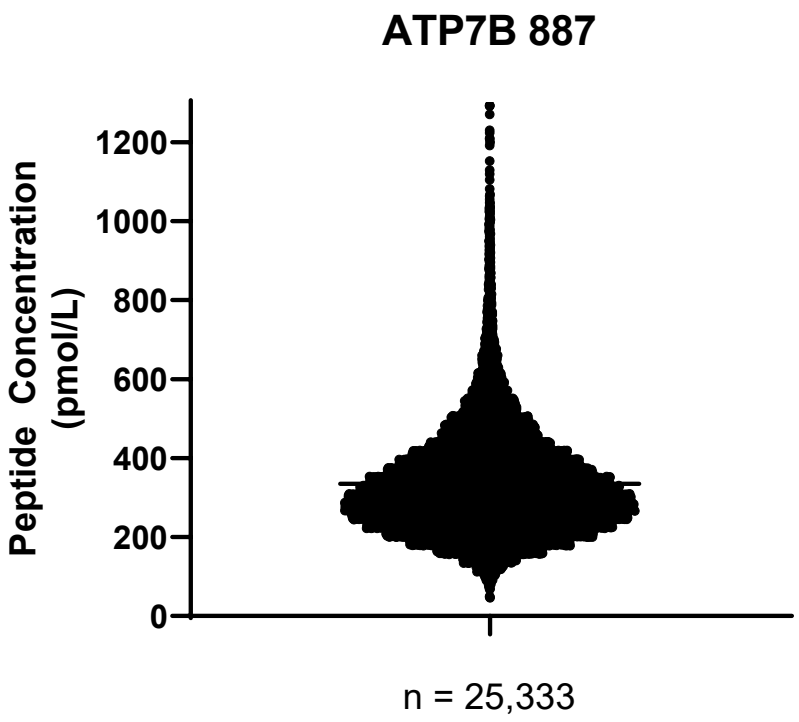
Category	Number
Male	13,035
Female	12,298
< 1500 g BW	258
1500 - 2500 g BW	1,533
2501 – 3500 g BW	13,995
> 3500 g BW	9,547

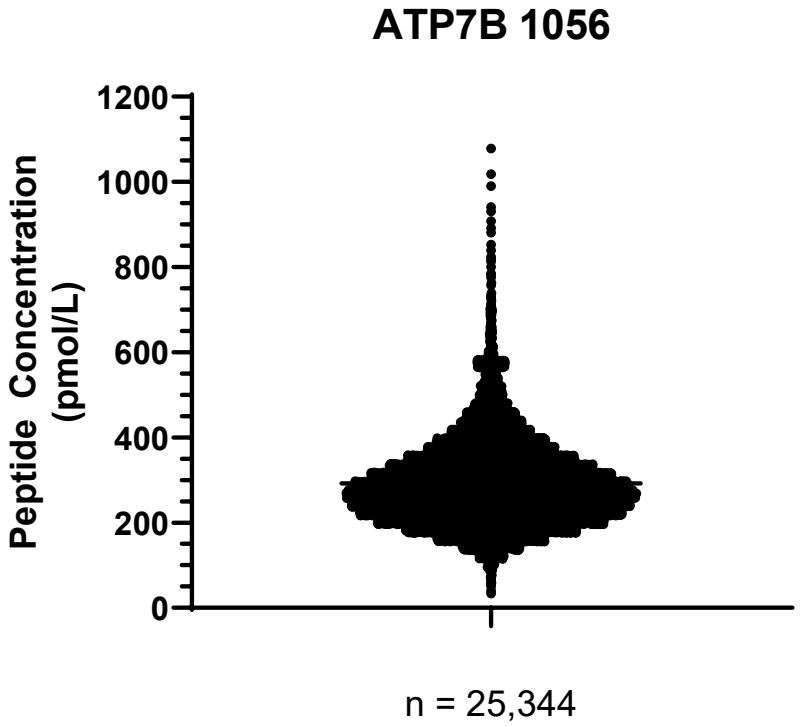
DOC After Birth	Number
0 day (< 24 hours)	4,044
1 day	10,345
2 day	5,608
3 day	3,285
4 day	1,165
5 day	479
6 - 14 days	407

Ethnicity	Number
White	15,603
Hispanic	4,967
Asian	2,813
Black	1,803
Native American	518
Other	910

WA Pilot Study of Newborn Screening: *Peptide Conc.*

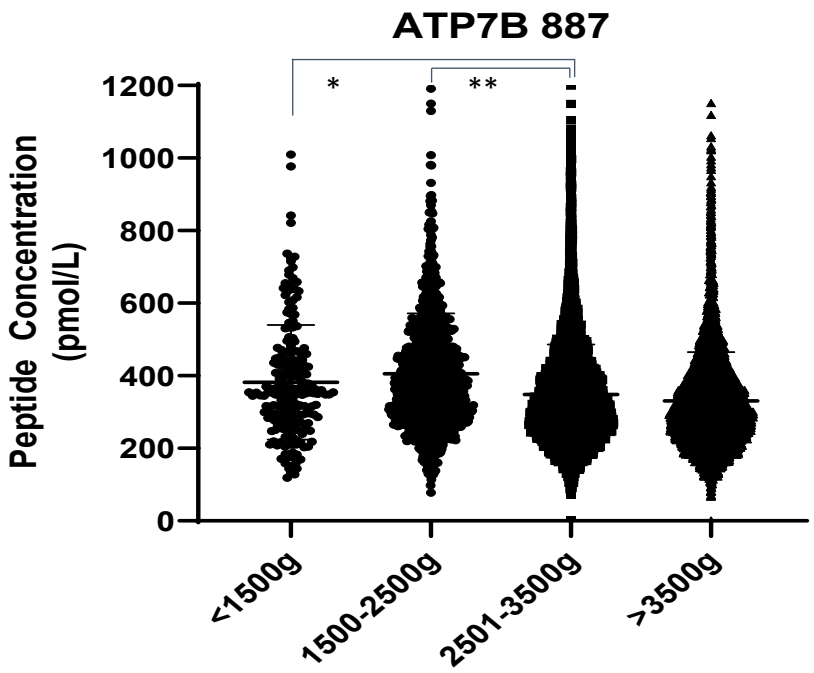
- *Two peptide concentration distributions were identical*

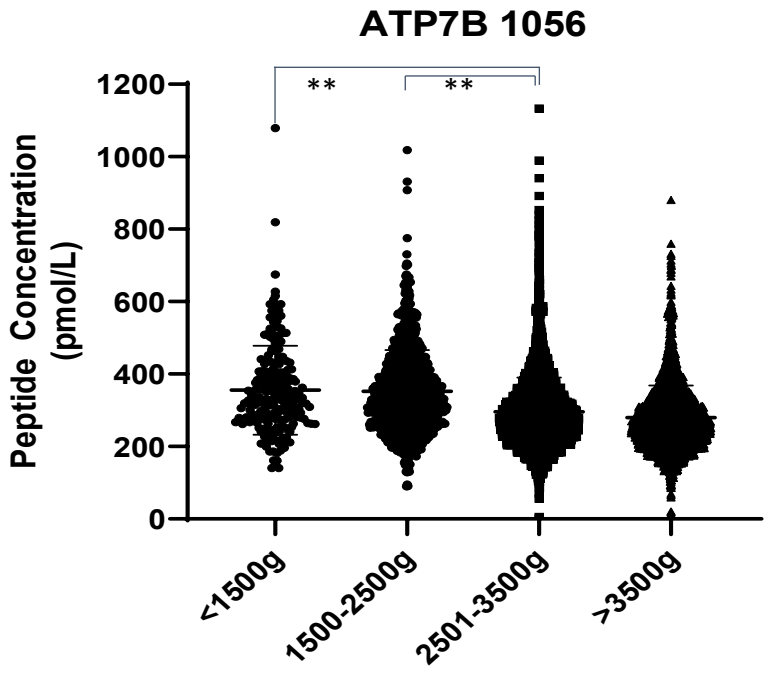




WA Pilot Study of Newborn Screening : *Birth Weight*

- *Peptide concentration was slightly higher in low birth weight but did not impact the overall cutoff range*





*P<0.01
**p<0.001

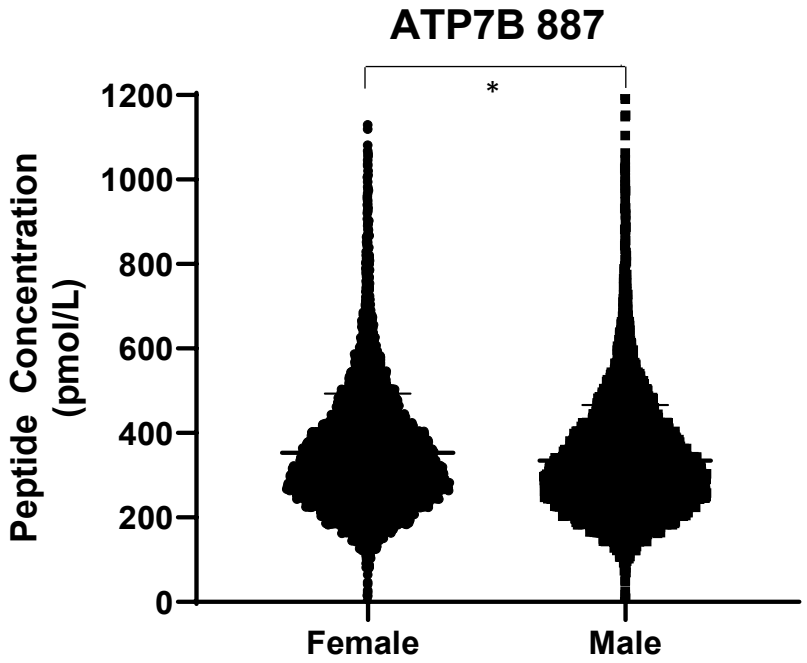
WA Pilot Study of Newborn Screening : *Birth Weight*

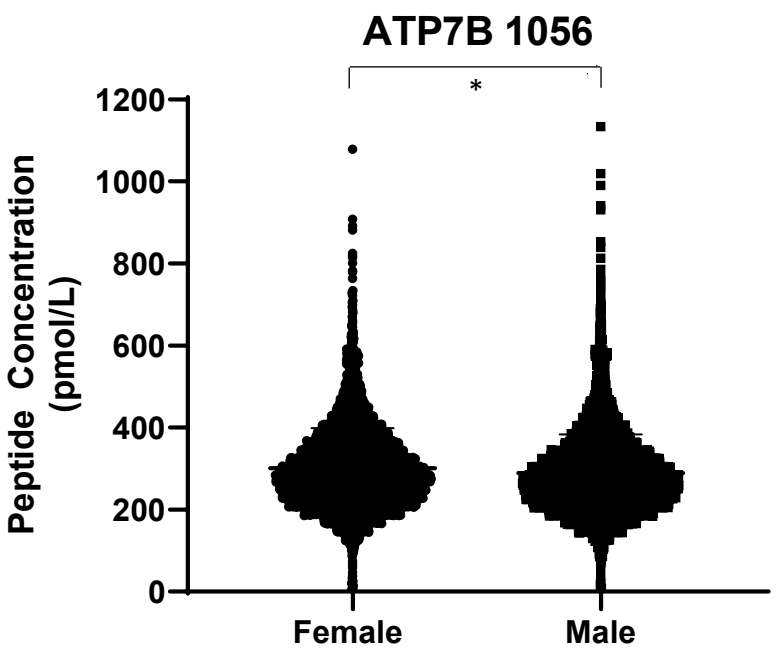
Statistical Analysis

Peptide	<1500g mean (pmol/L)	1500-2500g mean (pmol/L)	>2500g Mean (pmol/L)	p-val <1500g vs >2500g	p-val 1500-2500g vs. >2500g
887	382.0	405.6	341.6	0.0008	<0.0001
1056	355.3	352.4	289.6	<0.0001	<0.0001

WA Pilot Study of Newborn Screening : *Gender*

- *Difference between gender is minimal though statistically significant due to high number of samples tested*





*P<0.001

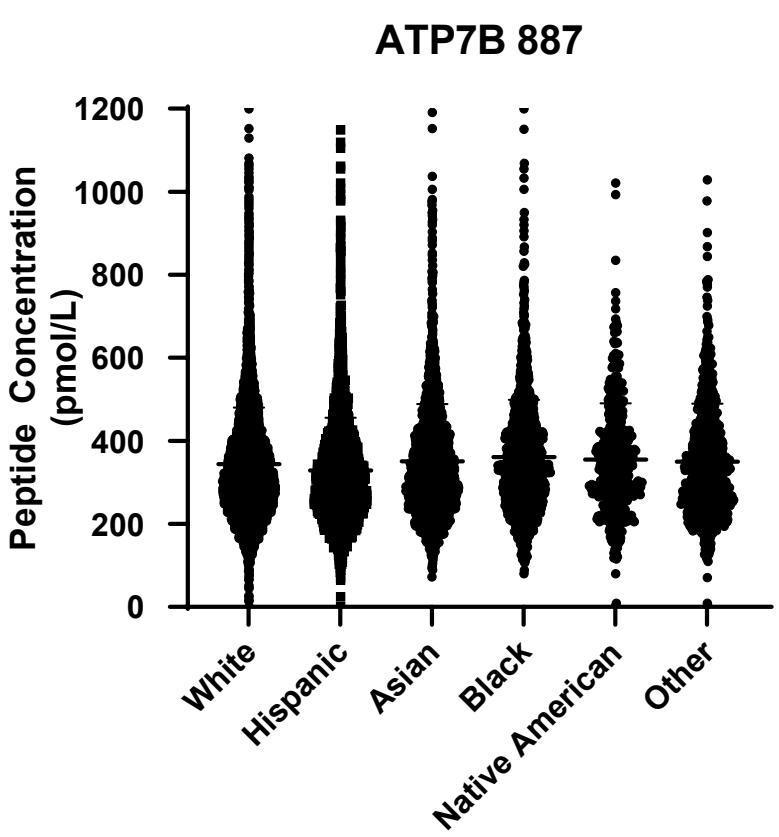
WA Pilot Study of Newborn Screening : *Gender*

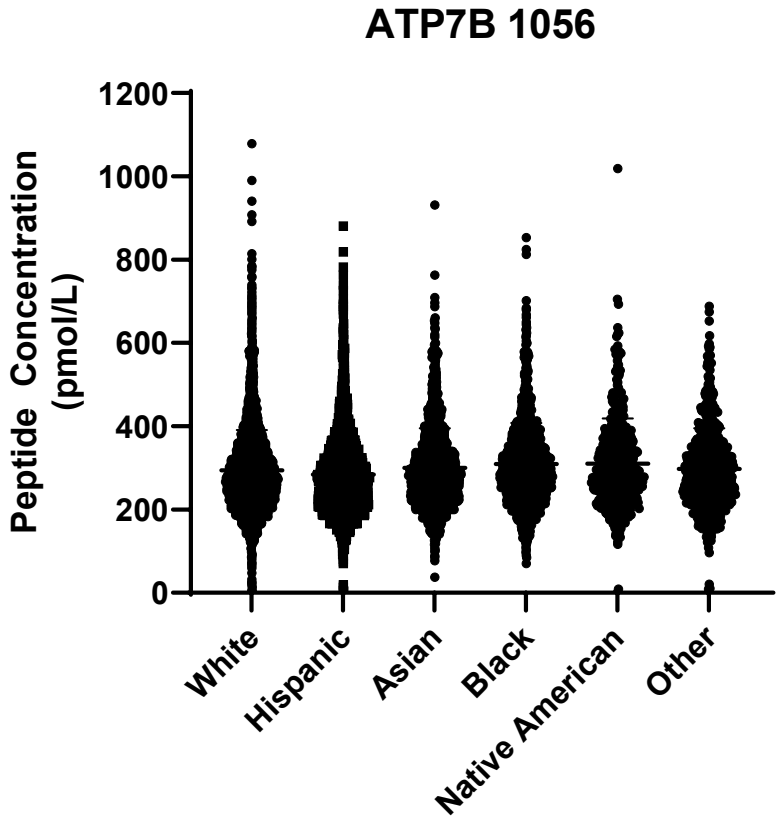
Statistical Analysis

	Female mean (pmol/L)	Male mean (pmol/L)	Difference between means	p-value
887	352.2	334.4	18.4	<0.0001
1056	288.7	301.1	12.4	<0.0001

WA Pilot Study of Newborn Screening : *Ethnicity*

- *No difference in various ethnicities*





Four presumptive positive cases detected

- Please note that current cut-off was tentatively set which can be readjusted in the future
- Two False positive (2/25,000 = 0.008%)
- One likely true positive with two VUS
- One likely true positive DNA pending

	BW (g)	Sex	ATP7B 887 (pmol/L)	ATP7B 1056 (pmol/L)	Genotype	Note
1	3690	M	67.6	134.5	c.3402del/no second variant	carrier
2	4105	M	71.0	96.5	p.Pro610Leu/p.Arg1224Leu	Presumptive Positive
3	3870	M	66.4	70.0	NO VARIANTS	Not affected
4	3840	F	64.3	59.2	PENDING	DNA pending

1: IonKey column (cutoff 68.4 and 55.5 respectively)

2-4: ESI low flow column (cutoff 76.2 and 75.7 respectively)

Summary

- These studies highlight the use of a novel IVD assay demonstrating the feasibility of LC-MS/MS proteomics for NBS of Wilson disease
- The false positive rate is extremely low
- FDA study shows the performance and precision of the manufactured kit were reliable, highly sensitive, and specific for targeted peptides as surrogate markers for ATP7B protein (please see separate PowerPoint slide)
- LC-MS/MS has been adopted globally in clinical laboratories and newborn screening. We anticipate the assay kit can be successfully utilized for clinical/public practice
- Other global feasibility studies are on going:
    - Sardinia (Dr. Georgios Loudianos): 41/44 cases below cutoff (manuscript in preparation)
    - Grand Canary Island, Spain (Drs. Luis García Villarreal/Antonio Tugores)
    - Costa Rica (Dr. Monica Portman Penon)

Acknowledgements

- **Key Proteo, Inc**

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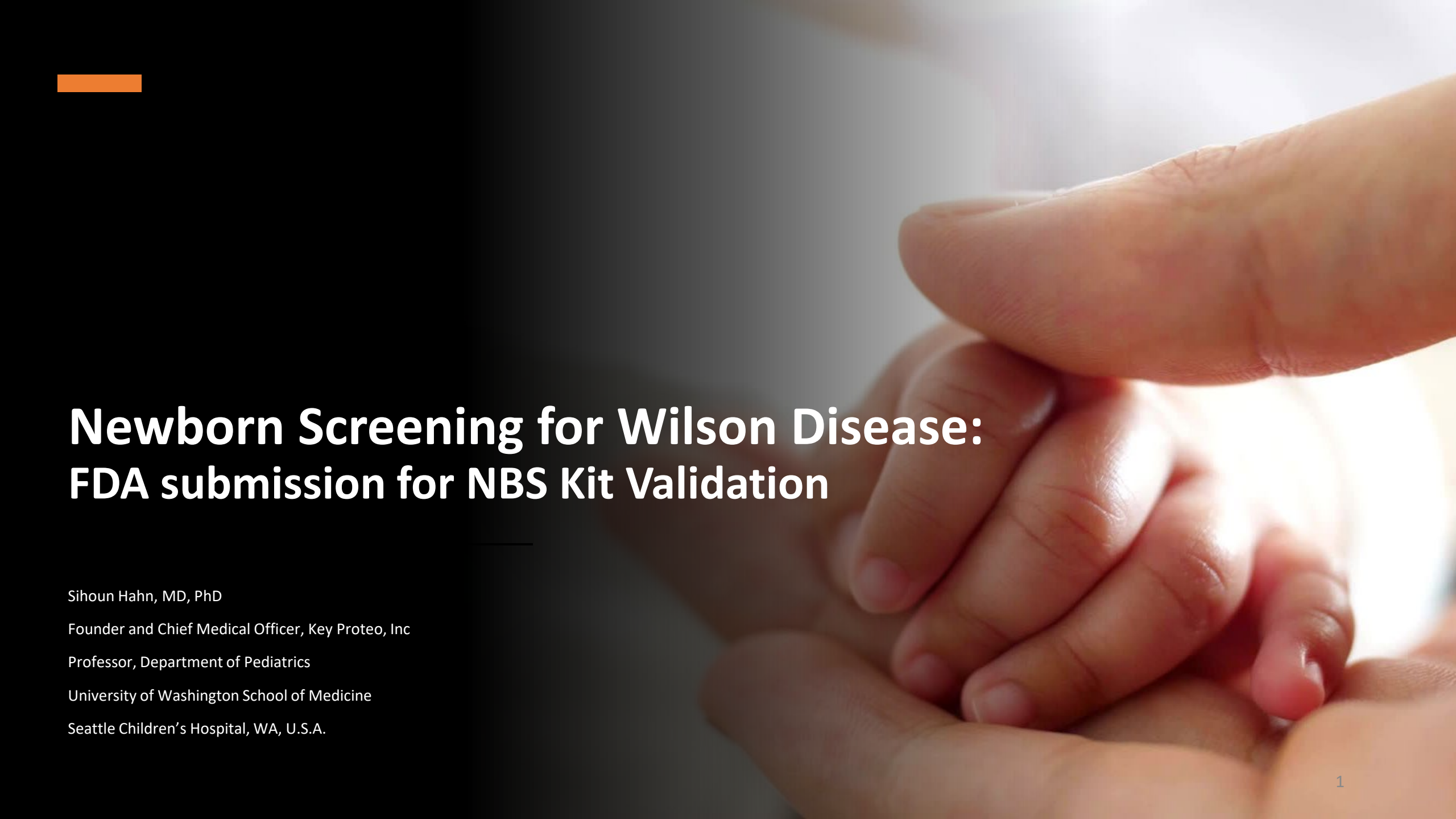
Newborn Screening for Wilson Disease:
FDA submission for NBS Kit Validation



Sihoun Hahn, MD, PhD

Founder and Chief Medical Officer, Key Proteo, Inc

Professor, Department of Pediatrics

University of Washington School of Medicine

Seattle Children's Hospital, WA, U.S.A.

FINANCIAL DISCLOSURES

Sihoun Hahn, MD, PhD, is a member of the Seattle Children's Hospital workforce and is serving as Chief Medical Officer of Key Proteo, Inc. He is an inventor of intellectual property that has been licensed to Key Proteo, Inc. Dr. Hahn is the founder of Key Proteo, Inc. and has ownership equity interests in the company.

Key Proteo Newborn Screening Kit for Wilson Disease

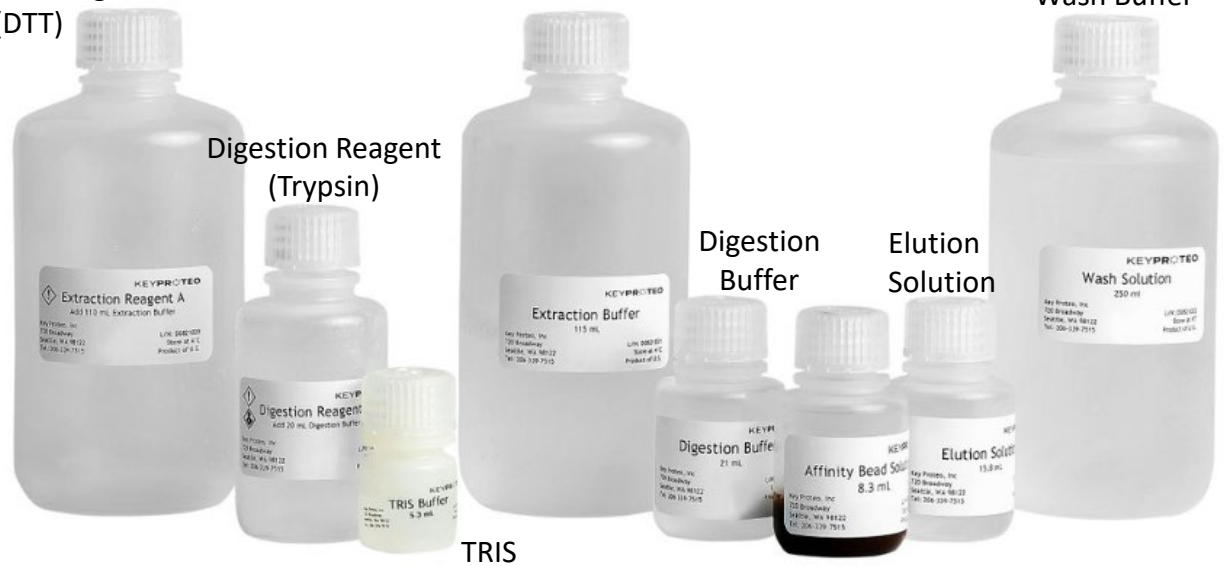
Intended Use:

- The Key Proteo Newborn Screening test is a quantitative LC-MS/MS assay, for **combined and simultaneous detection** of biomarker peptides representing (1) ATP7B, (2) BTK, (3) WASP, and (4) ADA proteins from dried blood spots. The deficiency of these proteins are diagnostic for *Wilson disease*, *X-linked agammaglobulinemia, Wiskott-Aldrich syndrome, and ADA deficiency*, respectively.

- Key Proteo Newborn Screening assay is indicated for use in a population screening of newborns in public health labs as an initial screen for the listed conditions. Those indicated as potential patients would be guided to follow up with specialists in the indicated diseases. This will allow for additional follow up testing as deemed appropriate by healthcare providers to provide or rule out a definitive diagnosis and initiate appropriate pre-emptive treatment.

Public Health Transition: Prototype 4-plex Screening Reagents

- Prototype reagents for assay performance have been manufactured at GMO facility
- Affinity beads mixed into a single solution
- Internal Standard Peptides lyophilized into 96-well plates
- DBS Control Cards
  - 3 levels of DBS spots
  - Negative, Low, High



Internal Standard Plates

Control Cards

Prototype reagents are not FDA approved and currently intended for investigational use only

Analytical Validation Study for FDA

- *Kit was classified as De novo, Class II by FDA: First-of-its kind proteomic-based NBS IVD kit*

Reproducibility/Precision:

The precision studies were performed following the recommendations in the CLSI EP05-A3 guideline at three different laboratories. Repeatability (within-run precision), between-day precision, and reproducibility (between operators) were conducted utilizing a six level multi-analyte reproducibility panel (0,20,40,60,80, and100% human blood).

Study sites 1 and 2 were conducted over 5 days, with 2 plates per day and 3 replicate measurements of DBS samples with identical peptide concentrations by two operators for a total of 360 measurements. Each study site used 1 lot of reagents and 1 instrument.

Study site 3 was conducted over 20 days, with 2 plates per day and 3 replicates per plate by two operators for a total of 720 measurements.

Site to Site Reproducibility Study

- The repeatability and within-laboratory precision of the assay for each multiplexed peptide were determined by replicate measurement of DBS samples with 6 different peptide concentrations created by serial dilution of NHC with fish blood.
- Assessment of within-laboratory precision was done using studies based on the CLSI EP5-A3 document and the modified 20 x 2 x 2 study for internal site and 5 x 2 x 2 study for two external sites.
- The study was conducted using a total of 720 measurements for each multiplexed peptide at the internal site and 180 measurements at each external site. Each kit on the three sites was from three different lots. A two-way nested ANOVA model was used to calculate the site-specific precision estimates.
- Overall, the repeatability, between-day and reproducibility on 100% human sample showed CVs of less than 30% for ATP7B peptides in all three sites.

Peptide	Panel	Mean (pmol/L)	Intra-day (%)	Between Day (%)	Between operator (%)
ATP7B 1056	Panel A	NA	NA	NA	NA
	Panel B	56.0	6.7	6.3	6.2
	Panel C	99.7	11.3	12.1	12.0
	Panel D	168.9	9.9	13.8	13.4
	Panel E	230.9	9.7	12.1	11.8
	Panel F	312.2	12.3	13.6	12.9
ATP7B 887	Panel A	NA	NA	NA	NA
	Panel B	56.8	9.7	11.2	10.6
	Panel C	101.0	13.9	13.8	14.1
	Panel D	154.6	8.3	10.9	10.6
	Panel E	200.5	8.8	11.1	10.6
	Panel F	246.7	11.1	11.0	10.9

Analytical Validation Study for FDA

Analytical Sensitivity:

The limit of blank (LOB), limit of detection (LOD) and limit of quantitation (LOQ) for the test kit were determined following the recommendations in the CLSI EP17-A2 guideline with consultation of CLSI document C64 “Quantitative Measurement of Proteins and Peptides by Mass Spectrometry”, involving replicate analysis of a set of blank and low-level samples using multiple lots and a single instrument

Detection Capability (pmol/L)	N	ATP7B 887	ATP7B 1056
Limit of Blank	120	15.5	13.7
Limit of Detection	120	24.5	22.7
Limit of Quantification	180	40.2	49.6

Analytical Validation Study for FDA

Carry-Over:

Carryover was determined by replicate measurements of blank samples with and without carryover for each multiplexed peptide. The blank samples were run followed by a DBS sample with high analyte concentrations for each multiplexed peptide and repeated to evaluate change in concentration due to carryover. The carryover effects of all peptides except ADA 93 were negligible. Based on the results, a specimen with ADA concentration within or slightly higher than the borderline cutoff should be retested if a specimen with ADA concentration within the normal range is present in the same column

Target Peptide	Carryover (%)
ATP7B 887	0.2-4.1
ATP7B 1056	0.9-4.2

Analytical Validation Study for FDA

Interference:

Analytical specificity was determined in accordance with the CLSI EP7-A2 guideline. The interferents tested for the kit are as follows: Hematocrit, Unconjugated Bilirubin, Conjugated Bilirubin, Galactose, Glucose, EDTA, Heparin, Total Protein, Hemoglobin, and Triglyceride. Blood pools were made in both 100% fish blood and a 30% mixture of NHC and fish blood. Test pools were spiked with interfering substances. None of the 11 interferents tested in this experiment had a significant effect on the concentrations of all six peptides at the recommended initial interferent concentration levels (below 20%)

Interference	Recommended Concentration	Unit
Unconjugated Bilirubin	0.4	mg/mL
Conjugated Bilirubin	0.4	mg/mL
Galactose	0.6	mg/mL
Glucose	10	mg/mL
EDTA	3.4	µM
Heparin	3.3	µg/mL
Total Protein	50	mg/mL
Hemoglobin	10	mg/mL
Cholesterol	400	mg/dL
Triglycerides	1500	mg/dL
Gamma-globulin	2.5	g/dL

Analytical Validation Study for FDA

Stability:

Stressed stability test was conducted in collaboration with Nelson Laboratories (Salt Lake City, UT). The study was conducted to determine the stability of screening kit using a classical and real-time approach as described in the CLSI EP17-A2 guideline. On-board stability testing was conducted by replicate panel of eluted samples placed into the instrument sample manager and measuring across time (0, 4, 8, 24, 32 and 48 hours). The kit was stable up to 6 month or even longer at -20'C. DBS stability was determined up to 5 days at three different temperatures. ATP7B peptides were stable at all conditions with no statistically significant differences.

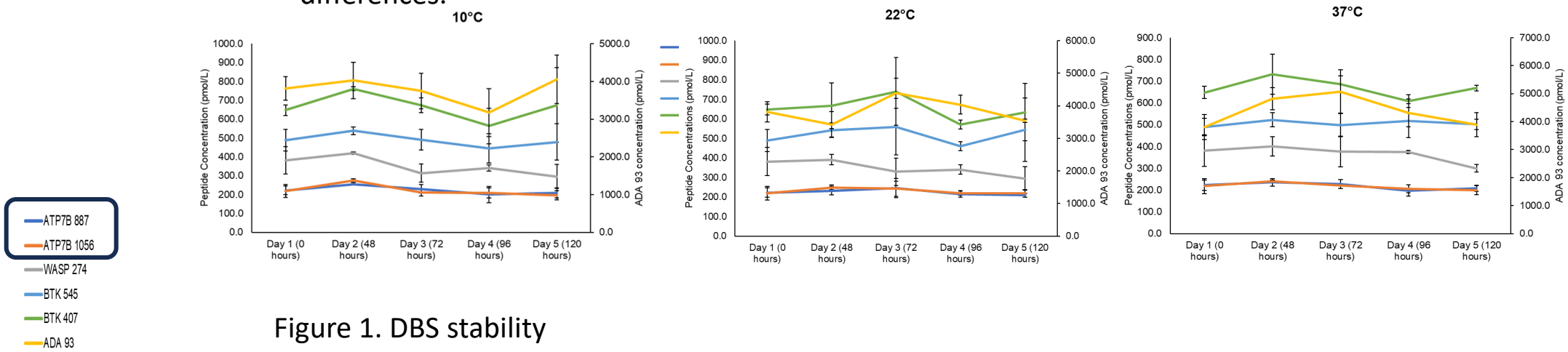


Figure 1. DBS stability

Clinical Validation Study for FDA

- A total of 3,294 newborns and 32 WD patient samples (including retrieved original NBS samples) were blindly tested at three sites (SCH, APL, KP).
  - No presumptive positive cases were detected in presumably normal newborn samples
  - All confirmed 32 positive Wilson disease cases were screened positive, and repeats were concordant with initial results
- To estimate the potential for false negative results of the test, the samples presumed to be normal (total 33 cases above the cutoff out of 3,294 newborns) were sequenced for *ATP7B* gene in a clinical molecular laboratory
  - 10 Carriers: Six (6) VUS and four (4) pathogenic variants. No second variants.
  - One uncertain case: Two variants of VUS were detected in which the peptide result was normal. The clinical significance is yet uncertain.
  - 22 cases with no variants

Additional FLEX studies

- **Upon requests, following FLEX study results can be provided:**
  - DBS punch test
  - Freeze-Thaw cycle test
  - Kit inversion test
  - Temperature/Time/Volume test
  - Hematocrit test
  - Inter-injection variation test

Summary

- FDA study demonstrated that the performance and precision of the manufactured kit was reliable, highly sensitive, and specific for targeted peptides as surrogate markers for ATP7B protein

- These studies highlighted the use of a novel IVD assay demonstrating the feasibility of LC-MS/MS proteomics for NBS of Wilson disease

- LC-MS/MS has been adopted globally in clinical laboratories and newborn screening. We anticipate the assay kit can be successfully utilized for clinical/public practice

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Key Proteo Newborn Screening Kit 1 Product Insert

Newborn Screening Kit 1

Wilson Disease | X-Linked Agammaglobulinemia | Wiskott-Aldrich Syndrome | ADA Deficiency

Reagents and Consumables for 440 specimens

Instructions For Use

Important Notice
Please read these Instructions For Use fully. If you have any questions or comments, please contact Key Proteo Customer Support at support@keyproteo.com or call (+1) 206-339-7515.

IVD
For *In Vitro Diagnostic Use*

Key Proteo, Inc.
720 Broadway
Seattle, WA 98122, USA
www.keyproteo.com

SYMBOLS GUIDE

The following symbols may be found on Newborn Screening product packaging and labeling:

	Manufacturer		*In vitro* diagnostics
	Date of Manufacture		Consult Instructions For Use
	Expiration Date		Customer Support
	Reference Item Number		Temperature Limits
	Lot Number		Do Not Reuse
	Serial Number		Control
	Caution		Keep dry
	Biohazard		Number of Tests
	Keep Away From Sunlight		

Contents

1. Intended Use

The Key Proteo Newborn Screening Kit 1 – WD|XLA|WAS|ADA is intended for quantitative measurement of levels of specific peptides that identify high-risk populations for WD, XLA, WAS, and ADA deficiency, respectively, from newborn dried blood spot (DBS) specimens, using the XEVO-TQXS mass spectrometer as an aid in screening newborns for WD, XLA, WAS, and ADA deficiency. Reduced levels of the measured peptides may be indicative of Wilson's Disease and the inborn errors of immunity (IEI) such as XLA, WAS or ADAD. The peptides measured by the Key Proteo NBS Screening Kit – WA|XLA|WAS|ADA and their associated genetic disorders and IEI are listed below.

Protein (Abbreviation)	Disease (Abbreviation)
Copper-transporting ATPase2 (ATP7B)	Wilson Disease (WD)
Bruton's Tyrosine Kinase (BTK)	X-linked Agammaglobulinemia (XLA)
Wiskott-Aldrich Syndrome Protein (WASP)	Wiskott-Aldrich Syndrome (WAS)
Adenosine Deaminase (ADA)	Adenosine Deaminase Deficiency (ADAD)

2. Indications for Use

The Key Proteo Newborn Screening Kit 1is a quantitative LC-MS/MS assay, for combined detection of ATP7B, BTK, WASP, and ADA proteins from dried blood spots. The deficiency of each of these proteins indicates high risk for Wilson's Disease, X-linked Agammaglobulinemia, Wiskott-Aldrich Syndrome, and ADA deficiency, respectively. The Key Proteo Newborn Screening assay is indicated for use in a population screening of newborns as an initial screen for the listed conditions. Those indicated as potential patients would be guided to follow up with specialists in the indicated diseases. This will allow for additional follow up testing as deemed appropriate by healthcare providers to provide or rule out a definitive diagnosis and initiate pre-emptive appropriate treatment.

Warnings: For *In Vitro Diagnostic Use*

3. Summary and Explanation of the Test

Wilson Disease (WD) is an autosomal recessive copper transport disorder with an estimated incidence of 1 in 30,000 individuals and with a carrier frequency of 1 in 90 [1]. WD is caused by mutations in the *ATP7B* gene, which encodes a copper-transporting ATPase. Copper accumulation in WD is present at birth and continues until toxic levels in the brain and liver produce noticeable symptoms which eventually lead to disease diagnosis. Symptoms usually present in the first decade of life, with most cases occurring between the ages of 5 and 35 years old [2]. Hepatic symptoms predominate in younger patients in their first decade of life, and neurological symptoms occur in older patients in the second or third decade of life [3]. Patients left undiagnosed and untreated during this accumulation phase develop irreversible brain damage, often in the form of Parkinsonism, and require liver transplants due to liver cirrhosis. Early treatment, with either a chelating agent such as Trientine or with zinc salts, has been proven to be highly effective in preventing these negative sequelae and allows patients to live essentially normal lives [4]. Nevertheless, since WD is rare, there is a significant challenge in rapid screening, thus treatment is often started only after the development of severe and permanent complications. Previous pilot studies using ceruloplasmin as a potential biomarker were unsuccessful due to high false positive and negative rates [5].

There are >1300 variants in *ATP7B* reported worldwide [6, 7] (www.varsome.com). Most mutations are rare, and the five most prevalent mutant alleles are estimated to be responsible for ~70% of the disease spectrum [8]. The two most common mutations, p.H1069Q (~35% in the European population) and p.R778L (~30% in the East Asian population) [8-10], result in markedly decreased levels of ATP7B protein, presumably due to enhanced degradation [10-12]. This is in line with observations that disease-causing missense mutations [13-16], protein-truncating nonsense mutations (~13% of known point mutations) [17], and frameshift mutations [8] result in the absence or decay of mRNA [18, 19] and therefore absent or diminished levels of the protein. ***Taken together, it is expected that most patients with WD have an absence or decreased level of ATP7B protein***. Our previous studies have confirmed this protein reduction in patient samples studied thus far [20-23].

Inborn Errors of Immunity (IEI) are a group of over 416 genetic disorders that compromise the health of affected individuals due to an improperly functioning or wholly absent immune system. These conditions often arise from genetic variants that lead to deficiencies in the abundance and/or activity of various proteins critical for immune function in the crucial time between birth and diagnosis. During this time period, serious complications related to IEI can emerge, including profound and frequent infections, autoimmunity, malignancy, and death. Although individually quite rare with varying frequency, the combined prevalence of all IEI is estimated to be about 1 in 1,200 [24-26]. Fortunately, if diagnosed early and accurately, appropriate treatments can dramatically improve, and in some cases, save the lives of the patients [27, 28]. Depending on the disorder, effectively curative methods exist such as hematopoietic stem cell transplantation (HSCT) and gene therapy [28-34]. Undoubtedly, early detection of IEI is essential to manage and prevent potentially life-threatening infections and chronic negative sequelae [35, 36].

(a) X-Linked Agammaglobulinemia (XLA) is *the most common primary immunodeficiency in men*. It is caused by a single genetic defect encoding **Bruton's tyrosine kinase (BTK) protein** and occurs with an incidence of 1:200,000 [37]. Loss of function of this kinase in males leads to a block in B cell development and B cell lymphopenia. Patients are therefore unable to make productive antibody responses and are prone to severe, life-threatening infections. Most patients with XLA who receive immunoglobulin on a regular basis will be able to lead relatively normal lives [27]. They do not need to be isolated or limited in

their activities. For **XLA**, ~60% of patients lack expression of BTK, and another 20-25% demonstrate markedly decreased levels [38, 39]. We have confirmed this reduction in 97% of patients from previous studies [21, 22].

(b) Wiskott-Aldrich Syndrome (WAS) is an X-linked congenital immunodeficiency in which affected males have very small, dysfunctional platelets and consequently have moderate to severe thrombocytopenia and a tendency to have bleeding problems with a combined immunodeficiency caused by defects in **WAS Protein (WASP)** expression [40]. It has an estimated incidence between 1-10 in 1 million [41]. The only permanent cure for WAS is transplantation of stem cells from bone marrow, peripheral blood, or cord blood. Thirty years ago, WAS was a fatal disorder with a life expectancy of only two to three years. Follow-up of the earliest WAS bone marrow transplant recipients for more than 30 years has demonstrated that this therapy is curative [31, 40]. The recent success of gene therapy for WAS holds promise for being the treatment of choice for this disease in the future [42]. In **WAS** patients, ~60% have a complete absence of WASP protein expression and another ~35% demonstrate decreased levels [43, 44]. We have confirmed this reduction in 100% of patients from previous studies [21, 22].

(c) Adenosine Deaminase (ADA) is expressed in erythrocytes, lymphoid cells, and plasma, and is mutated in ADA-deficient $T^-B^-NK^-$ SCID [45]. Inherited ADA deficiency causes a severe form of SCID that may have a somewhat variable phenotypic spectrum, with 10%-15% of patients having a 'delayed' clinical onset presenting by 6 to 24 months, and a smaller percentage of patients having a 'later' onset, diagnosed from age 4 years to adulthood. This form of SCID can be missed by the current SCID NBS method [46]. We have confirmed this reduction in previous studies [21].

4. Principles of Procedure

In the reagent Kit, protein concentrations are analyzed by measuring the amounts of representative peptides that are specific to the targeted protein. An internal standard peptide nearly identical to the target sequence is included at a known concentration and analyzed along with the peptide biomarker. The peptide concentration in blood is then calculated using the ratio of the peptide in the dried blood spots (DBS) to the internal standard.

From each DBS sample, proteins in the specimen are extracted using 50 mM ammonium bicarbonate solution. Disulfide bond reduction is performed using 2 M DTT. Trypsin enzyme is added to initiate digestion of proteins to peptides. Heavy isotope-labeled synthetic peptide internal standards (IS) are incorporated at optimized concentrations for quantification by MS/MS. Target peptides are then captured and enriched using anti-peptide monoclonal antibodies immobilized on magnetic protein G beads. Samples are incubated overnight at 4˚C with shaking. After incubation, anti-peptide MAb beads containing captured peptides are collected using a magnet and washed to remove non-target peptides. Finally, peptides are eluted with 30 µL of 5% acetic acid/3% acetonitrile. Since the internal standard and target peptide are chemically identical, any loss of target peptide (incomplete MAb capture, absorption to vessel walls, etc.) is accounted for. Target peptide-containing supernatant is transferred to a new well for subsequent MS/MS analysis.

LC-MS/MS is performed using a Waters Xevo TQ-XS. LC runs are performed with an elution gradient using H_2O + 0.1 % formic acid and acetonitrile + 0.1% formic acid before column washing and re-equilibration for a total run-time of 2.4 minutes for multiplexed analysis of 12 peptides (six endogenous peptides extracted from DBS and six isotopically labeled IS) covering 4 target conditions. The mass spectrometer is operated in MS/MS mode that allows the first mass-selective filter (quadrupole) to pass the target peptide-derived parent ion. This is followed by collision-induced dissociation of the peptide in the second quadrupole followed by a third quadrupole to allow a specific peptide-derived fragment to pass to the detector (triple-quadrupole MS/MS). This is referred to as single reaction monitoring mode (SRM). A collection of SRMs, one for each target peptide, is cycled rapidly so that all target peptides are detected in the same LC-MS/MS run (multiple reaction monitoring, MRM). The Reagent Kit contains the necessary reagents and consumables for completion of the assay before LC-MS/MS analysis.

5. Kit Contents

Each reagent kit contains sufficient reagents for 440 newborn specimens and quality controls, totaling 1760 tests as each specimen is tested for up to 4 different protein deficiencies. The expiration date for the unopened kit is on the outer label. An expiration date is noted on the labels of the individual kit components. The components are provided in two boxes- one cold (2-8 °C) box containing reagents and one room temperature (15-30 °C) box containing plastics and consumables. The kit components along with their quantity and storage conditions are listed in **Table 1** and **2.**

5.1. Materials Provided:

Table 1. Refrigerated (2-8 °C)/Frozen box contents.

Item	Composition	Delivery State	Number in Kit	Initial Storage	Storage After Initial Use
DBS Calibrator Cards	QC Card: Negative	Dried Blood Spot	1 Card x 5 spots	-20°C	-20°C
	QC Card: Low	Dried Blood Spot	1 Card x 5 spots	-20°C	-20°C
	QC Card: High	Dried Blood Spot	1 Card x 5 spots	-20°C	-20°C
Internal Standard	IS Mix in Deep Well Plate	Lyophilized	5 plates	-20°C	-20°C
Extraction Reagent A	DTT	Solid	1 Bottle x 101.8 mg	2-8°C	Aliquot and store at -20°C
Extraction Buffer	0.1% Triton X-100 in Ammonium Bicarbonate	Liquid	1 Bottle x 115 mL	2-8°C	
Digestion Reagent A	Trypsin	Solid	1 Bottle x 20 mg	2-8°C	Aliquot and store at -20°C
Digestion Buffer	50 mM Acetic Acid	Liquid	1 Bottle x 21 mL	2-8°C	
Tris Solution	1M Tris pH 8.0	Liquid	1 Bottle x 5.3 mL	2-8°C	2-8°C
Bead Mix	In 1X PBS + 0.03% CHAPS	Liquid	1 Bottle x 9.2 mL	2-8°C	2-8°C
Wash Solution	0.1X PBS + 0.01% CHAPS	Liquid	1 Bottle x 250 mL	2-8°C	2-8°C
Elution Solution	H_2O + 5% acetic acid + 3% ACN	Liquid	1 Bottle x 15.8 mL	2-8°C	2-8°C

Table 2. Room temperature (15-30 °C) box contents.

Item	Number In Kit
50 mL Reservior	2
10 mL Reservior	3
10 mL Reservior PPE	1
DWP for Extraction	5
MS Plate	5
Plate Seal	20
MS Plate Seal	5

5.2 Internal Standard Concentrations

Internal standard peptides are utilized to indicate the concentrations of the target peptides in the blood specimen. These internal standards are mixed in the correct ratios and lyophilized into 96-well *Internal Standard Plates*. Each well of the *Internal Standard Plate* contains enough internal standard peptide for 1 experiment. The amounts of each peptide lyophilized into each well is listed below in **Table 3**.

Table 3. Internal Standard amounts in each well.

Peptide	Femtomole Peptide
BTK 407	25
WAS 274	25
ATP7B 1056	5
ATP7B 887	5
BTK 545	50
ADA 93	100

5.3 Calibration

Included in each concentration calculation is a constant factor that calibrates the measured peptide concentration values between lots. This ensures consistency of the measured concentrations between lots and is designed to help maintain consistent cutoffs. The constant value by which the blood peptide/internal standard ratio should be multiplied is provided in a lot-specific quality control certificate.

5.4 Quality Control DBS

The quality control dried blood spots include 3 levels of control material: QC negative, QC low, and QC high. The composition of the quality control DBSs is summarized in **Table 4** below. The peptide concentrations in the QC dried blood spots are provided on a lot-specific quality control certificate included in each assay kit. Each laboratory should establish its own mean and acceptable range.

Table 4. Quality Control Materials.

Quality Control Level	Composition
QC Negative	Fish Whole Blood drawn with Sodium Heparin anticoagulant before spotting
QC Low	A mixture of Normal Human and Fish Whole Blood drawn with Sodium Heparin anticoagulant before spotting
QC High	Human Whole Blood drawn with Sodium Heparin anticoagulant before spotting

5.5 Materials required but not provided:

- DBS puncher
- Personal protective equipment: gown and gloves.
- Adjustable single-channel pipettes: as needed.
- Single-channel pipette tips: as needed.
- Multichannel pipettes: 10 µL, 100 µL, 300 µL, 1200 µL
- Multichannel pipette tips: 10 µL, 100 µL, 200 µL, 1200 µL
- Alpaqua Magnum EX universal magnet plate, A000380 (Beverly, MA) or equivalent.
- KimWipes, Kimberly-Clark, Fisher Scientific. (Chicago, IL) or equivalent.
- Vortex Mixer, Four E's (Amazon, M/N: MI0101002) or equivalent.
- Orbi-Shaker MP, BenchMark Scientific (Genesee Scientific) or equivalent.
    - For use at 15-30 ℃ and 2-8 ℃
- Incu-Mixer MP Plate Shaker, BenchMark Scientific (Genesee Scientific) or equivalent.
    - For use at 37+/-1 ℃
- Centrifuge 5810, Eppendorf (AG, Germany) or equivalent.
    - Capable of centrifuging deep well plates/microtiter plates
- Waters Xevo TQ-XS MS with ESI source and Low-Flow probe connected to Waters M-Class Gradient and Loading pumps, Waters (Milford, MA).
- nanoEase M/Z Peptide Colum; P/N 186009257, Waters (Milford, MA).
- 2x nanoEase loading column; P/N: 186009251; Waters (Milford, MA).
- Acetonitrile (no. A955, LCMS optima grade); Thermo Fisher Scientific (Waltham, MA) or equivalent; Stored at 15-30 °C.
- Water (no. W6, LCMS optima grade); Thermo Fisher Scientific (Waltham, MA) or equivalent; Stored at 15-30 °C.
- Formic Acid (no. A117-50, LCMS optima Grade); Fisher Scientific (Waltham, MA) or equivalent; Stored at 2-8 °C.

6. Warnings and Precautions

- For in vitro diagnostic use.
- For professional use only.
- The Key Proteo Newborn Screening Kit 1 should be used by adequately trained personnel.
- Gloves should be worn whenever working with blood samples. All blood samples, containers, and materials that contact blood should be handled as if capable of transmitting infectious disease and discarded into a biohazard waste container after use.
- This kit contains quality control DBS that were produced using human blood specimens. All quality control DBS were tested for HIV, Hepatitis B, Hepatitis C, and other bloodborne pathogens by FDA-approved or equivalent methods and found to be negative for all infectious agents. However, all recommended precautions for handling blood specimens as specified in the document provided by U.S. Health and Human Services titled "Biosafety in Microbiological and Biomedical Laboratories" should be followed.
- *Internal standard plates, QC dried blood spots, and lot-specific QC reports are all lot specific and should not be mixed between lots.*
- Disposal of all waste should be in accordance with local regulations.
- Ensure that materials from the Key Proteo Newborn Screening Kit 1 are treated as biohazard waste.
- Do not use any reagent kit components after their expiration dates.
- Do not use any damaged reagent kit components.
- Store all reagent kit materials as described in section 5.1, Materials Provided.
- Keep work areas clean per standard practices.
- Wash and dry hands before and after testing. Wear gloves and appropriate PPE.

7. Limitations

- Test results are intended to be used in conjunction with clinical and diagnostic findings, consistent with professional standards of practice, including confirmation by alternative methods, and clinical evaluation as appropriate.
- Reduced or absent protein concentrations must be confirmed by standard diagnostic methods, such as gene sequencing.
- This test is a screening test and is not intended to diagnose the genetic conditions listed herein.
- As with other screening tests, negative results do not rule out the diseases being screened.
- Conditions that are known to cause anomalous results are[47]:
    - Specimen spot not uniformly saturated with blood.
    - Specimen spot punched too close to the edge of the blood spot.
    - Poorly collected and improperly dried specimens.
    - Non-eluting blood spot due to deterioration of specimen caused by exposure to heat and humidity.
    - NBS samples collected from premature infants or sick/transfused infants may require mandated repeat specimen.
- The Key Proteo Newborn Screening Kit 1 may result in:
    - False positives by detecting carriers for the targeted diseases.
    - False negatives if disease-causing mutations do not affect protein concentration.

    - Please also refer to the Section titled "Procedural Notes".
- The test is specific for the targeted peptides. Other peptides that may be present in the blood do not react in/are not identified by the test.
- Certain regions or demographics may have differences in average protein concentrations or the incidences of false positives and negatives.
- The performance of the Key Proteo Newborn Screening Kit 1will be determined using the procedures provided in these Instructions For Use. Failure to follow these procedures and precautions as directed may alter test performance.
- Improper sample handling (through collection, storage, or transport) may impact results or lead to invalid results.
- This reagent kit cannot identify peptide levels associated with diseases other than those stated in this Instructions For Use.

8. Specimen Collection, Storage, and Handling

Blood specimens should be taken using the standard procedures utilized by public health workflows. Neonatal screening programs differ from one another in the type of specimen required. In the United States, the recommendation is that a blood spot, approximately 12.7 mm (0.5 inch) in diameter, be collected by heel prick and spotted onto filter paper. Blood from a newborn heel prick is usually collected 24-48 hours after birth. However, in some screening programs, the specimen from the neonatal heel prick may be collected 2-6 days after birth. Consult local regulations for appropriate timing and screening specimen collection. The manufacturer recommends following the procedures listed in the CLSI document number NBS01 - A6- "Blood collection on filter paper for newborn screening programs; Approved standard"- Sixth edition (2013)[47] and some important points are mentioned below:

- Ensure that the expiration date of the blood collection card has not passed.
- Wipe the newborn skin with 70% isopropanol and allow the skin to dry.
- Puncture the infant's heel with a heel incision device or a sterile lancet by making a standardized incision of 1.0 mm deep. Ensure that the puncture does not exceed 2.0 mm in depth since a deep incision may cause bone damage in small infants.
- Wipe away the first drop of blood with a sterile gauze pad and allow a large drop of blood to form. Touch the filter paper against the large drop of blood and allow a sufficient quantity of blood to soak through and fill the entire pre-printed circle. Examine both sides of the paper to ensure that the blood uniformly penetrated and saturated the filter paper.
    - o Do not excessively squeeze the puncture since it may cause hemolysis of the specimen or result in a mixture of tissue fluids with the specimen and might adversely affect the assay result.
    - o Do not apply successive blood drops to the same printed circle or already partially dried spots can result in "caking".
- Allow the blood specimen to air dry on a horizontally level, non-absorbent, open surface for at least 3 hours at an ambient temperature, away from direct sunlight. Ensure that the specimens are not stacked to avoid cross-contamination.
- Ensure that the required information on the dried blood spot card is completed which includes:
    - o Last name, First name, Sex, Birth date, Birth weight and Patient identification number
    - o First and last name of mother
    - o Date of specimen collection
    - o Name and address of the submitter
    - o Name and phone number of the physician
    - o Name of the newborn screening program and address
    - o Each card should have a unique serial number
- Follow the basic triple packaging system i.e., blood absorbed into paper, a fold-over flap or inner envelope, and an outer envelope of high-quality paper. Ensure that the local regulation and institutional policies are followed when shipping dried blood spot specimens.
- Transport the dried blood spot specimens within 24 hours of collection unless otherwise directed by the newborn screening laboratory.
- Humidity and moisture are detrimental to the quality of the dried blood spot. Special attention must be paid to the storage and transportation conditions of the dried blood spot specimens. Storage of specimens in an environment with elevated temperatures and humidity may increase the risk of false positive results.

8.1. Target Peptide Stability in Dried Blood Spots

This plan governs the determination of DBS stability for the Key Proteo Newborn Screening Kit 1 before analysis. As this is a multiplexed test, stability was determined for each of the six peptide targets. Assay design involves replicate analysis of a panel of eluted samples and measured across time up to 5 days at 10°C, 25°C, and 37°C. Through this protocol, it was determined how long the DBS samples can be expected to measure consistent concentrations over time at different temperature conditions. Quality control and 100% normal human control blood(NHC) , samples were processed per the IFU. Three (3) replicates of each sample were injected at each subsequent day and change in concentrations over time were monitored.

All six target peptides were stable for up to 5 days at all three different temperatures (10°C, 22°C and 37°C) with no statistically significant differences.

9. Assay Procedure

The Key Proteo Newborn Screening Kit 1 assay procedure is detailed below.
**NOTE*: All frozen and refrigerated reagents, components, and samples must be brought to room temperature (15-30°C) before use.*

9.1. Plate and Specimen Preparation

- Wear nitrile gloves and clean work bench with 70% Ethanol.
- Initialize automated DBS puncher or equivalent for use.
- If using a standard hole puncher, clean standard hole puncher and tweezer by spraying and wiping with 70% EtOH.
- Pre-heat Incu-Mixer MP Plate Shaker or equivalent to 37+/-1°C.
- Punch out controls and samples into wells (1/8-inch punches from filter paper disks).
    - To minimize variation in controls it is advisable to avoid punching solely from the perimeter of the dried blood spot.
    - A recommended punching layout is represented below where the three punches needed for each well can be taken from each of three DBS sections below (**Figure 1**).

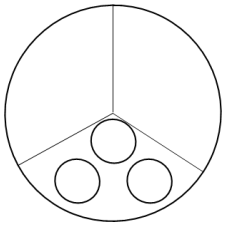

Figure 1. Diagram of recommended DBS punching locations. Three punches can be taken from each of three DBS sections.

Table 5. Recommended Quality Control DBS punch location

A12	IS
B12	IS
C12	QC Negative
D12	QC Low
E12	QC High
F12	QC Negative
G12	QC Low
H12	QC High

- A sample plate layout is shown below in **Figure 2**.

	1	2	3	4	5	6	7	8	9	10	11	12
A	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	IS
B	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	IS
C	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	QC Neg
D	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	QC Low
E	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	QC High
F	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	QC Neg
G	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	QC Low
H	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	NBS	QC High

Figure 2: Recommended plate layout

- Thaw Extraction Solution aliquots or make Extraction Solution per DBS Protein Extraction protocol below.
- For each DBS sample, punch 3 x 3.2-mm punches (equivalent to ~10.5 µl blood per punch). DBS spots are punched (using Panthera or equivalent) or placed (using tweezers) into 96-well Masterblock Extraction Plate wells.
- Generate Plate Layout document (each sample has a designated well location, i.e. A1, A2…).

8.2. DBS Protein Extraction

- Add 110 mL of Extraction Buffer into bottle containing Extraction Reagent A.
- Invert 10 times to dissolve and mix.
- Transfer resulting Extraction Solution to 50 mL Reagent Reservoir as needed.
- Add 200 µL of this Extraction Solution into each well.
- Seal plate with a new foil plate seal.
- Place the plate into Incu-Mixer MP Plate Shaker or equivalent and incubate at 37+/-1°C for 25-35 min at 1000 RPM.
- NOTE: Store remaining Extraction Solution in 21-ml aliquots in -20°C freezer.
    - To thaw:
        - remove aliquot from -20°C freezer.
        - Thaw to 15-30°C using one of the following options: let sit at 15-30°C, or incubate at 37+/-1°C.

8.3.Trypsin Digestion

- Transfer 20 mL of Digestion Buffer into bottle containing Digestion Reagent A. ***Do Not Vortex!***
- Pipette to dissolve and mix.
- Transfer resulting Digestion Solution to 10 mL Reagent Reservoir.
- Remove plastic plate seal, then add 37.5 µL of Digestion Solution to each well.
- Seal plate with new foil plate seal.
- Incubate mixture on Incu-Mixer MP Plate Shaker or equivalent at 37+/-1°C for 2 hours +/- 10 min to digest.
    - NOTE: Store remaining Digestion Solution in 4-ml aliquots in -20°C freezer.
    - To thaw:
        - remove aliquot from -20°C freezer.
        - Thaw by letting sit at 15-30°C.

8.4. Peptide Enrichment

- Remove plate seal from the Internal Standard Plate.
- Remove foil plate seal from the Extraction Plate, then transfer 200 µL of the resulting DBS digest to its corresponding well on the Internal Standard Plate.
- Remove TRIS Buffer from 2-8°C refrigerator and mix gently by swirling or pipetting up/down.
    - Do Not Vortex.
- Transfer TRIS Buffer into 10 mL reservoir for ease of addition to plate.
- To each sample, add 10 µL of TRIS Buffer.
- Mix for 1-3 minutes on Orbi-Shaker MP at 1000 RPM at 15-30°C.

- Remove Affinity Bead Solution from 2-8°C refrigerator and mix gently by pipetting.
- Transfer Affinity Bead Solution into 10 mL Affinity Bead Reservoir for ease of addition to plate.
- To each sample, add 18 µL of Affinity Bead Solution.

- Seal plate with a new foil plate seal (foil if leftover lyo spots).
- Incubate samples overnight on Orbi-Shaker MP at 1000 RPM at 2-8°C to allow for peptide capture.

8.5. Washing Beads

- The next day, remove foil plate seal from Internal Standard Plate, then place plate on Alpaqua Magnum EX magnetic plate rack and leave for 1-3 min to collect beads.
- Remove Wash Solution from 2-8°C refrigerator and invert 10-15 times to mix.
- Transfer Wash Solution to 50 mL reagent reservoir to aid in transfer to plate.
- Transfer supernatant from beads to corresponding wells in new DWP. Store plate containing transferred digest in -20°C freezer.
- Remove plate from Alpaqua Magnum EX magnetic plate rack.
- Add 220 µL of Wash Solution to each well.
- Pipette up/down 5-10 times to mix.
- Place plate on Alpaqua Magnum EX magnetic plate rack and leave for 1-3 min to collect beads.
- Remove supernatant from beads and discard.
- Remove plate from Alpaqua Magnum EX magnetic plate rack.
- Repeat wash by adding 220 µL of Wash Solution to each well.
- Pipette up/down 5-10 times to mix.
- Place plate on Alpaqua Magnum EX magnetic plate rack and leave for 1-3 min to collect beads.
- Remove supernatant from beads and discard.
- Remove plate from Alpaqua Magnum EX magnetic plate rack.

8.6. Peptide Elution

- Add Elution Solution to 10ml reservoir.
- After discarding the final supernatant, add 30 µL of Elution Solution to each well.
  - NOTE: Pipet up/down 10-20 times (until fully homogenized).
- Seal plate with a new foil plate seal.
- Shake for 4-6 minutes on Orbi-Shaker MP or equivalent at 1000 RPM at 15-30°C to elute peptides.
- Centrifuge in Eppendorf 5810 or equivalent at 600 RPM for 15-25 seconds.
- Remove plate seal, then place plate on Alpaqua Magnum EX magnetic plate rack. Leave for 1-3 min to collect beads.
- Transfer 14 µl of eluted peptide solution into corresponding 96-well MS Plate.
  - Take care not to disturb or pipette the beads collected on the bottom of the well.
- NOTE: If beads are disturbed or aspirated, return beads to well, wait 20-30 seconds to allow to settle, ensure pipet is placed in center of well, then re-attempt aspiration.
- Seal plate with new MW Waters plate seal.
- Centrifuge in Eppendorf 5430 centrifuge or equivalent at 3000 rpm for 25-35 seconds.
- Transfer plate to MS for analysis.

8.7. Liquid Chromatography Conditions and Peptide Transition List

[Liquid Chromatography (LC) Inlet Method]

- The provided Key Proteo Newborn Screening Kit 1 standard LC conditions are below.
- Run time, load time, and LC mode are listed in **Table 6**.

Table 6. Inlet method run time, loading time, and mode.

Run Time (Min)	2.4
Loading Time (Min)	1
Mode	Trapping

- Trapping Conditions for both the gradient and loading pumps are listed in **Table 7**.

Table 7. LC Inlet method trapping conditions.

Trapping - Gradient Pump			
Time (min)	Flow (µL/min)	% A	% B
Initial	8	95	5

Trapping - Loading Pump			
Time (min)	Flow (µL/min)	% A	% B
Initial	40	98	2

- Analytical conditions for both the gradient and loading pumps are listed in **Table 8**.

Table 8. LC Inlet method gradient conditions

Analytical - Gradient Pump			
Time (min)	Flow (µL/min)	% A	% B
Initial	8	95	5
0.1	8	95	5
0.3	8	80	20
0.55	8	80	20
0.95	8	70	30
1.3	8	5	95
1.75	8	5	95
1.9	8	95	5
2.4	8	95	5

Analytical - Loading Pump			
Time (min)	Flow (µL/min)	% A	% B
Initial	40	98	2

- Trap valve manager settings should be set to “Toggle” as shown in **Figure 3**.

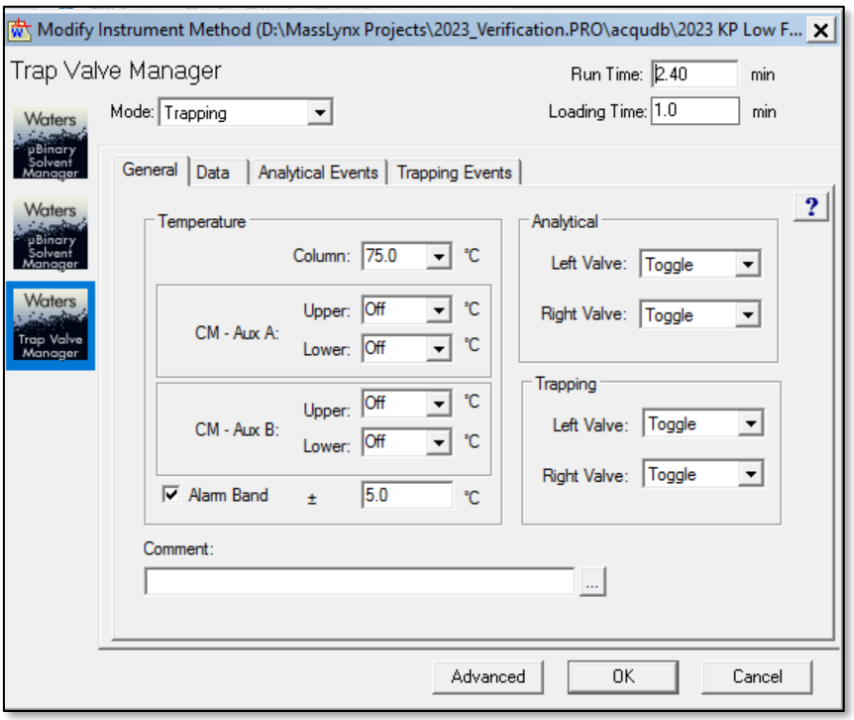

Figure 3: Trap Valve Manager Settings

- µSample Manager settings should be set as in **Figure 4**.

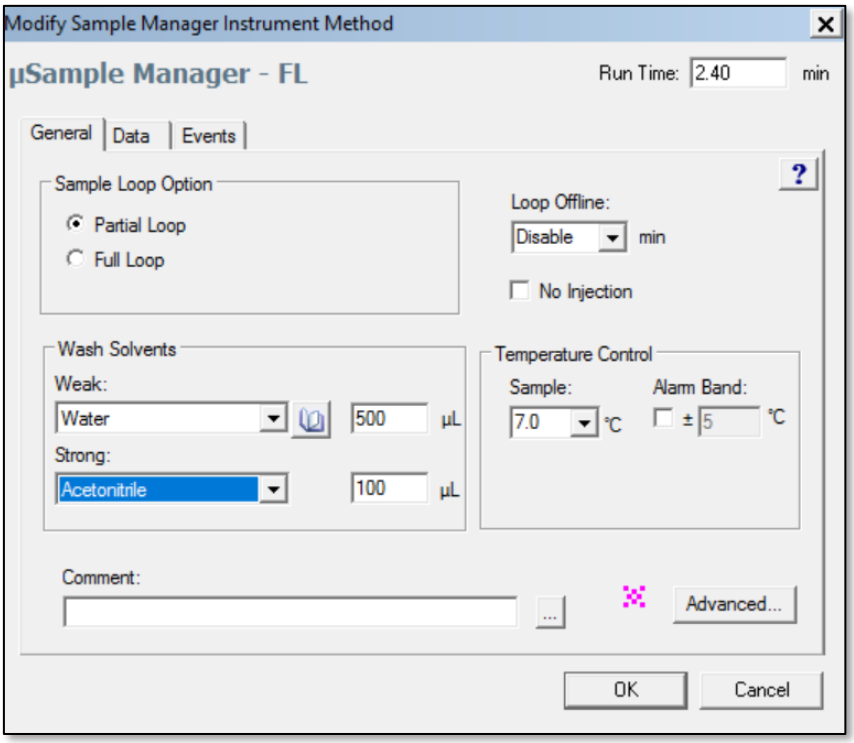

Figure 4: µSample Manager Settings

- Trapping mode advanced settings should be selected as shown in **Figure 5**.

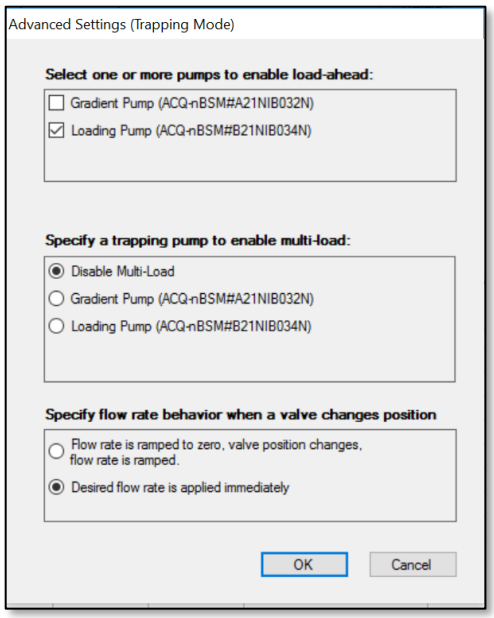

Figure 5. Advanced Trapping Mode Settings

[Mass Spectrometry (MS) Method]

- The provided Key Proteo Newborn Screening Kit 1 standard MS method conditions are below.
- **Table 9** lists the parent ions, fragment ions, cone voltages, and collision energies for each target peptide.

Table 9. Transition information for the Key Proteo Newborn Screening Kit 1 Standard MS Method.

		Parent Mass (m/z)	Fragment Mass (m/z)	Cone Voltage (kV)	Collision Energy (kV)	Fragment
Endogenous (Light)	ATP7B 887 L	523.6	558.2	0	14	y5+
		523.6	671.4	6	14	y6+
		523.6	695.3	0	14	b7+
		523.6	772.6	2	12	y7+
Internal Standard (Heavy)	ATP7B 887 IS	526.3	566.2	0	14	y5+
		526.3	679.4	6	14	y6+
		526.3	695.3	0	14	b7+
		526.3	780.6	2	12	y7+
Endogenous (Light)	ATP7B 1056 L	712.6	827.4	0	18	y17++
		712.6	877.0	0	18	y18++
		712.6	926.5	0	18	y19++
Internal Standard (Heavy)	ATP7B 1056 IS	715.2	831.4	0	18	y17++
		715.2	881.0	0	18	y18++
		715.2	930.5	0	18	y19++
Endogenous (Light)	WASP 274 L	761.0	650.7	8	23	y6
		761.0	751.5	0	22	y7
		761.0	864.6	8	24	y8
		761.0	1063.5	0	24	y10+
Internal Standard (Heavy)	WASP 274 IS	764.9	658.7	8	23	y6
		764.9	759.4	0	22	y7
		764.9	872.4	8	24	y8
		764.9	1071.5	0	24	y10+
Endogenous (Light)	ADA 93- L	525.4	403.6	5	12	y3+
		525.4	502.3	5	14	y4
		525.4	548.3	10	14	b5
Internal Standard (Heavy)	ADA 93 IS	530.5	413.6	5	12	y3+
		530.5	512.3	5	14	y4
		530.5	548.3	10	14	b5
Endogenous (Light)	BTK 545 L	781.8	828.4	0	28	y8+
		781.8	1187.5	0	20	y11+
		781.8	1300.6	0	28	y12+
Internal Standard (Heavy)	BTK 545 IS	785.8	836.4	0	28	y8+
		785.8	1195.5	0	20	y11+
		785.8	1308.6	0	28	y12+
Endogenous (Light)	BTK 407 L	568.1	402.3	2	16	y4+
		568.1	446.8	8	13	y9++
		568.1	892.5	2	16	y9+
Internal Standard (Heavy)	BTK 407 IS	571.9	410.3	2	16	y4+
		571.9	450.8	8	13	y9++
		571.9	900.5	2	16	y9+

[MS Analysis Procedure]

1. Open the MassLynx environment.

2. Open or generate the necessary sample list.
    a. Navigate to File -> Open -> *Select the relevant sample list.*
    b. Alternatively, generate the necessary sample list by entering the information for all analyte and QC samples.

3. Ensure the correct columns are present in the MassLynx Window.
    a. File Name
    b. Bottle/Vial
    c. Injection Volume
    d. Inlet Method
    e. MS File
    f. MS Tune File
    g. Sample Type
    h. ATP7B 887 (CONC A)
        i. If this column is headed with "CONC A", right click the column and use the "Properties" selection to rename the column to the peptide name.
    i. ATP7B 1056 (CONC B)
        i. If this column is headed with "CONC B", right click the column and use the "Properties" selection to rename the column to the peptide name.
    j. WASP 274 (CONC C)
        i. If this column is headed with "CONC C", right click the column and use the "Properties" selection to rename the column to the peptide name.
    k. ADA 93 (CONC D)
        i. If this column is headed with "CONC D", right click the column and use the "Properties" selection to rename the column to the peptide name.
    l. BTK 545 (CONC E)
        i. If this column is headed with "CONC E", right click the column and use the "Properties" selection to rename the column to the peptide name.
    m. BTK 407 (CONC F)
        i. If this column is headed with "CONC F", right click the column and use the "Properties" selection to rename the column to the peptide name.
    n. Quan Meth
    o. File Text
    p. If these fields are not present Right Click on the column heading bar and select "Customize Display". Then select the fields above.

4. Enter Standard Methods for control of MS, LC, and Tune page (**Figure 6**).
    a. Inlet File -> *Standard LC Method*
    b. MS File -> *Standard MS Method*
    c. MS Tune File -> *Standard Tune File*
        i. This file should be generated by Waters upon installation or calibration.

5. Enter Sample Specific Information.
    a. Under Sample Type
        i. Designate QC samples as "QC" using the dropdown menu.
        ii. Designate all other samples as "Analyte"
            1. Use Right Click -> Fill down to rapidly fill menus.

6. Enter Lot Specific QC Information.

a. Concentration values for each peptide will be provided on a lot-specific QC certificate.
b. Values will be provided for all QC DBS cards including Negative, Low, and High controls.
c. Enter these values into rows containing the QC DBS Cards
    i. Be sure to enter the values in the columns corresponding to the correct peptides.

7. Highlight all samples to be run and hit the play button in the toolbar to begin the analysis.
8. In the pop-up window, under operations select the following:
    a. Integrate Samples.
    b. Calibrate Standards
    c. Quantify Samples
9. Under the method dropdown menu select the correct TargetLynx Method.
10. Press OK.

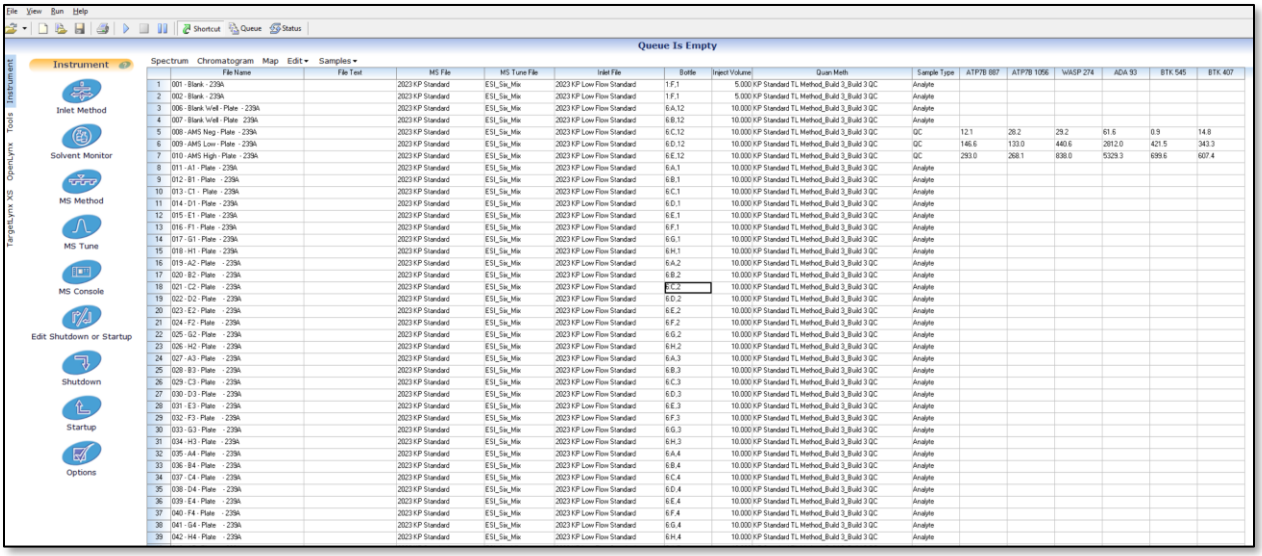

Figure 6. Example MassLynx Sample Layout

8.8. TargetLynx Results Procedure

1. On the left side of the MassLynx window, select “TargetLynx XS”.
    a. Select “Edit Method”.
    b. The Key Proteo Standard TargetLynx Method Editor should open.
    c. If it does not, navigate to File -> Open -> MethDB folder
        i. Select the Key Proteo Standard TargetLynx Method.

2. Set the lot specific Concentration Units for each peptide internal standard (**Figure 7**).
    a. There will be 12 compounds in the compound list.
        i. There will be an internal standard (IS) and an endogenous (light, L) compound for each of the six target peptides.
    b. Above the window on the right, select the “Calibration Properties” tab.
        i. For each IS compound in the compound list, there will be a <u>lot-specific</u> constant to enter under “Concentration of Standard”.
        ii. For each IS peptide, enter the constant value from the QC certificate next to “concentration of standard”.

1. This value incorporates blood volume, IS concentration, dilution, and lot specific calibration to generate a peptide concentration as an output.

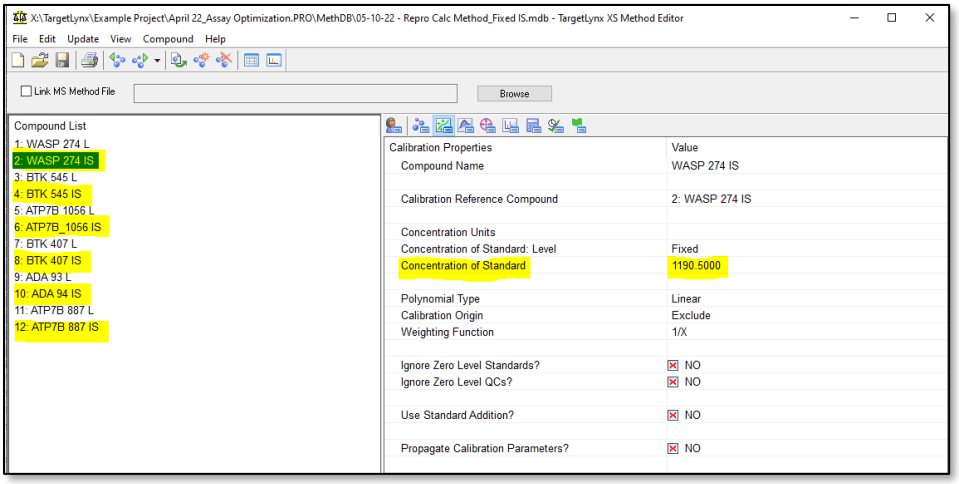

Figure 7. TargetLynx Concentration of Standard

3. Set the lot specific QC settings for each endogenous (Light) peptide (**Figure 8**).
   a. Above the window on the right of the TargetLynx XS method editor, navigate to the "QCMonitor" tab.
      i. For each L compound in the compound list, there will be a lot-specific QC and percent deviation values to enter under "QC Settings".
      ii. For each L peptide, enter the values from the QC certificate.
         1. These values set the allowable deviations from the pre-determined QC card concentrations and enable QC flagging.

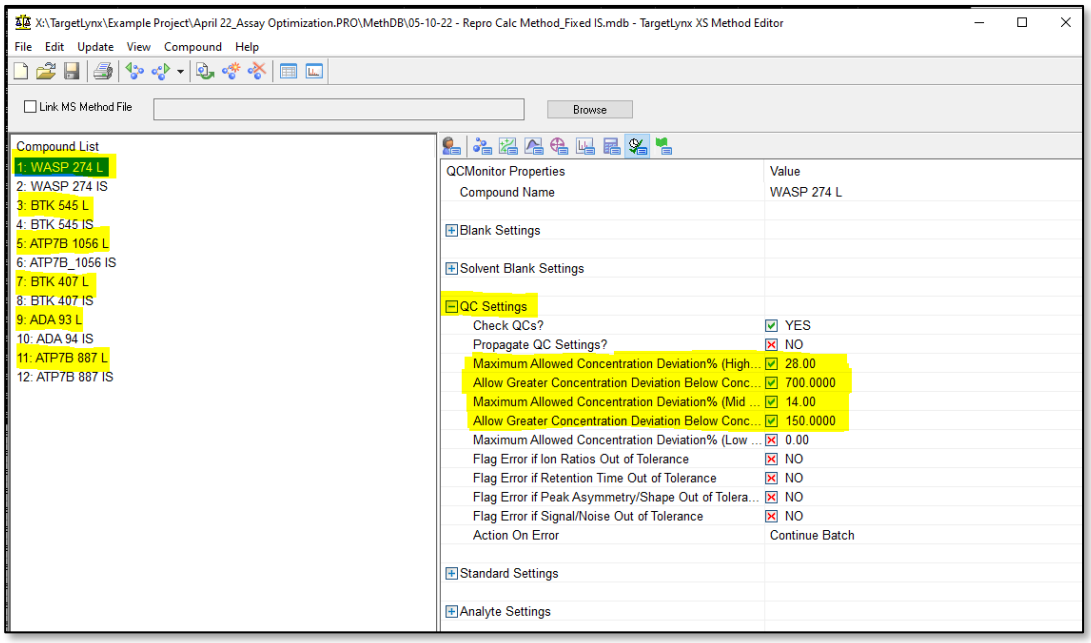

Figure 8. TargetLynx QC Monitor

4. TargetLynx method should now be prepared for sample processing.

8.9. Process Samples After Data Collection

1. Sample processing can be done after data collection.
    a. Open MassLynx software.
    b. Open the sample list that requires sample processing.
    c. Select all sample rows that require processing by clicking and dragging along the leftmost column.
    d. Highlight the “TargetLynx XS” tab along the leftmost side of the MassLynx window.
        i. Select “Process Samples” under the TargetLynx XS menu.
    e. A “Create TargetLynx XS Dataset” window will appear.
        i. Under operations select the following
            1. Integrate Samples
            2. Calibrate Standards
            3. Quantify Samples
        ii. Under the method dropdown menu select the “Key Proteo Processing Method”.
    f. Select OK.

8.10. TargetLynx Reports, Layouts, and Cutoffs

1. After processing, a TargetLynx Quantify MFC application window will open.

2. Two layouts will be provided that will format this TargetLynx window and allow for application of peptide cutoffs.
    a. Each layout will contain cutoffs for 3 peptides.
        i. KP_Layout 1_ATP7B_WASP contains cutoffs for ATP7B 887, ATP7B 1056, and WASP 274.
        ii. KP_Layout 2_ADA_BTK contains cutoffs for BTK 545, BTK 407, ADA 93.

3. Apply a Key Proteo layout.
    a. Select File -> Apply Layout
        i. A MethDB folder window will open.
        ii. Select one of the KP Peptide Layouts.

4. The Key Proteo Newborn Screening Reagent Kit Peptide Layouts will report Areas, Concentrations, Standard Concentrations, Deviations, Concentration Flags, and Cutoffs in columns.
    a. Chromatograms can be reviewed sample by sample.
    b. Deviations from QC concentrations can be monitored.

5. Peptide concentration cutoffs can be changed as needed (Figure 9).
    a. Load the TargetLynx layout that contains the peptide cutoff that needs to be changed.
        i. Right click the column containing the peptide of interest.
        ii. Select “Edit Column Properties”
        iii. Select “Formula”
        iv. A formula will be displayed of the format:
            1. Peak Response<=*[CUTOFF]*?1:0
        v. A formula will be preloaded with a recommended cutoff value e.g.

1. Peak Response<=100?1:0

vi. Changing the number between "=" and "?" will apply a new cutoff.
   1. Change this number
   2. Select "OK".

vii. The column will now flag any peptide with a concentration below the new cutoff with a "1".

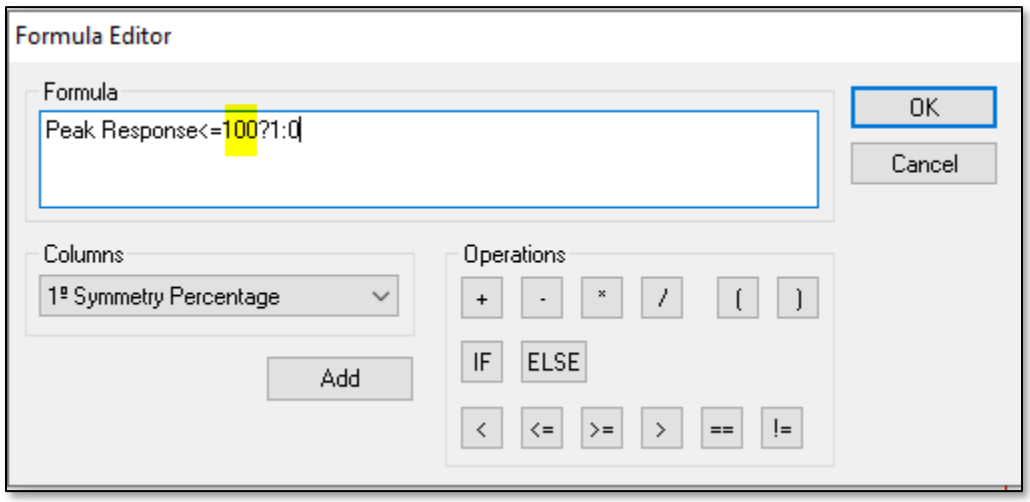

Figure 9: Setting TargetLynx Cutoffs

6. Each Layout can flag 3 cutoffs. Both layouts will be needed to monitor the six target peptides of interest.
7. To Add Concentrations that are generated.

8. Reports will need to be generated from both layouts.

8.11 . Procedural Notes:

- ***Internal standard plates, QC dried blood spots, and lot-specific QC reports are all lot specific and should not be mixed between lots.***

- Store and handle IS plates right-side up as lyophilized internal standard peptide pellets can become dislodged and lyophilized pellet dust can get stuck to adhesive of plate seal.
- The pellet itself can move around and potentially stick to adhesive.
  - If a pellet is loose in the well or stuck to the adhesive, clean forceps can be used to gently move the pellet to base of well to ensure accurate reading of sample.
- Ensure that all Wash Solution is removed from wells before Elution Solution is added.

10. Results and Reporting

10.1 Result Calculation: Target peptide concentrations are calculated using a ratio. The specimen peptide is determined as a specific concentration in the blood. Internal standard peptide is incorporated into the assay at a specific and known concentration. The ratio of the specimen peptide concentration to the internal standard peptide concentration gives the concentration of each peptide in the injected samples. This ratio is then multiplied by a constant value incorporating the assumed blood volume and between-lot calibration factors. Results are reported out in pmol/L of each peptide.

10.1. Run Failure:

10.1.1. Internal standard signals are monitored in wells A1 and A2 where no DBS samples are located. Lack of signal for any internal standard analyte in these wells constitutes a failure of the assay for that specific analyte. The sample run is recommended to be stopped and/or the samples are recommended to be repeated. The results are considered INVALID.

10.1.2. It is possible that an individual sample may experience a loss of chromatographic performance during a run. Any peptide signal presenting as a smear of signal across the gradient time or at a retention time different from the retention time of all other samples in the plate should be considered a failure and reported as INVALID and that specific sample should be re-tested. It is recommended that a third test is run to clarify the result.

10.1.3. Human blood is positive for all target peptides in the multiplex. The fish blood mixture is negative for all target peptides. DBS samples containing different levels of peptide targets will be created by serial dilution of NHC with negative fish blood. QC DBS cards will be processed twice during each run. These values for each individual peptide analyte at each of the low and high concentration levels should fall within the established ± 3 SD limits for the run to be accepted outright. Individual peptide values for the negative QC DBS cards should fall below pre-defined clinical cutoff concentrations to be accepted. Values outside of the established range across all QC samples on a plate will result in failure of a run and need to be retested.

10.1.4. Quality control DBS cards are supplied and must be processed alongside the assay on every plate. These standard deviation ranges are reported on lot specific quality control reports. QC DBS cards will be processed twice during each run. It is recommended that each lab establishes mean and SD values for each peptide and determine their own acceptable range of peptide values for QC samples. Control DBS results should be interpreted based on the laboratory's established criteria for acceptability and any state or federal requirements.

10.1.5. Presumptive positive results should only be reported from plates with passing DBS QC values as established by laboratory's internal guidance.

10.2. Values below LOQ: Values reported below the LOQ of the assay are positive for the targeted disease(s). Samples should be repeated to confirm peptide deficiency and reported as positive for the specific condition.

10.3. Values above Linear Range: Values reported above the linear range of the assay are likely negative for the targeted diseases and should be reported as negative (normal)

11. Quality Control

11.1. Materials: The quality control dried blood spots include 3 levels of control material: QC negative, QC low, and QC high. These spots contain mixtures of human and fish blood with varying concentrations of target peptides.

11.2. Use: It is recommended that QC samples are utilized to monitor the day-to-day validity of testing results. Control DBS at three levels are included in the kit. These controls should be run on each plate in accordance with the assay protocol. If more than one plate is run, these controls should be included on each plate. Peptide concentrations for all 6 target peptides are determined along with the newborn specimens. Lot-specific mean peptide concentration values and standard deviations (SD) will be provided with each Newborn Screening reagent kit. It is recommended that each lab establishes mean and SD values for each peptide and determines their own acceptable range of peptide values for QC samples. Patient results should be reported only if the control results meet the laboratory's established criteria for acceptability.

11.3. External Quality Control: QC testing should be performed in accordance to local, state and federal regulations. It is recommended to participate in external quality assurance programs such as CDC's NSQAP (Centers for Disease Control Newborn Screening Quality Assurance Program). It is also recommended that laboratories monitor trends in peptide concentrations for the QC samples on a weekly basis and compare them with the established limits.

12. Analytical Performance Characteristics:

12.1. Reproducibility/Precision: The precision studies were performed following the recommendations in the CLSI EP05-A3 guideline at three different laboratories. Repeatability (within-run precision), between-day precision, and reproducibility (between operators) were conducted utilizing a six level multi-analyte reproducibility panel (0,20,40,60,80, and100% human blood).

Study sites 1 and 2 were conducted over 5 days, with 2 plates per day and 3 replicate measurements of DBS samples with identical peptide concentrations by two operators for a total of 360 measurements. Each study site used 1 lot of reagents and 1 instrument.

Study site 3 was conducted over 20 days, with 2 plates per day and 3 replicates per plate by two operators for a total of 720 measurements.

Table 10. Site 1 Precision study (N=180)

Peptide	Panel	Mean (pmol/L)	Intra-day (%)	Between Day (%)	Between operator (%)
ADA 93	Panel A	379.6	26.8	24.2	35.2
	Panel B	900.8	6.1	11.2	10.4
	Panel C	1390.1	7.6	11.8	11.2
	Panel D	2263.5	20.8	24.4	24.4
	Panel E	2587.5	10.4	16.1	15.0
	Panel F	3324.0	8.2	14.8	14.0
ATP7B 1056	Panel A	NA	NA	NA	NA
	Panel B	59.1	5.1	8.5	7.9
	Panel C	79.2	11.8	12.9	12.3
	Panel D	117.5	9.9	11.3	10.9
	Panel E	156.0	6.0	9.7	9.5
	Panel F	223.0	9.3	14.6	13.5
ATP7B 887	Panel A	94.8	48.0	63.5	68.8
	Panel B	66.7	14.9	23.6	22.1
	Panel C	109.9	17.0	20.4	20.8
	Panel D	160.8	10.5	11.2	12.8
	Panel E	184.8	7.7	9.5	10.2
	Panel F	248.0	13.5	12.5	12.8
BTK 407	Panel A	53.9	6.8	10.3	10.1
	Panel B	107.3	21.7	23.5	25.5
	Panel C	192.3	34.5	35.2	35.9
	Panel D	268.2	12.5	17.5	18.9
	Panel E	335.3	21.6	21.8	21.8
	Panel F	405.0	11.6	10.9	11.0
BTK 545	Panel A	NA	NA	NA	NA
	Panel B	152.9	3.3	31.7	32.9
	Panel C	238.4	46.8	41.9	42.3
	Panel D	384.8	13.2	16.3	15.4
	Panel E	533.0	24.3	24.8	25.0
	Panel F	707.4	14.5	13.1	13.4
WASP 274	Panel A	190.1	19.3	21.8	20.8
	Panel B	188.7	7.4	34.9	31.4
	Panel C	226.1	9.8	35.3	31.8
	Panel D	279.9	11.0	26.0	24.0
	Panel E	324.5	10.8	26.5	24.1
	Panel F	380.9	10.7	17.1	15.9

Table 11. Site 2 precision study (N=180)

Peptide	Panel	Mean (pmol/L)	Intra-day (%)	Between Day (%)	Between operator (%)
ADA 93	Panel A	337.9	13.6	57.6	55.4
	Panel B	828.6	7.4	11.5	10.8
	Panel C	1387.8	8.2	9.7	9.2
	Panel D	2180.1	5.7	9.0	10.0
	Panel E	2854.8	6.5	9.3	9.5
	Panel F	3780.2	9.9	11.0	10.5
ATP7B 1056	Panel A	NA	NA	NA	NA
	Panel B	56.0	6.7	6.3	6.2
	Panel C	99.7	11.3	12.1	12.0
	Panel D	168.9	9.9	13.8	13.4
	Panel E	230.9	9.7	12.1	11.8
	Panel F	312.2	12.3	13.6	12.9
ATP7B 887	Panel A	NA	NA	NA	NA
	Panel B	56.8	9.7	11.2	10.6
	Panel C	101.0	13.9	13.8	14.1
	Panel D	154.6	8.3	10.9	10.6
	Panel E	200.5	8.8	11.1	10.6
	Panel F	246.7	11.1	11.0	10.9
BTK 407	Panel A	NA	NA	NA	NA
	Panel B	141.8	26.2	27.1	26.5
	Panel C	282.3	15.8	16.2	15.7
	Panel D	441.9	11.2	12.4	12.7
	Panel E	616.0	27.3	24.7	30.0
	Panel F	716.3	24.3	22.6	22.2
BTK 545	Panel A	NA	NA	NA	NA
	Panel B	145.5	21.1	21.0	21.6
	Panel C	299.8	20.0	20.3	19.7
	Panel D	497.1	15.7	16.9	16.7
	Panel E	728.9	28.7	26.9	30.5
	Panel F	865.2	22.7	21.4	20.9
WASP 274	Panel A	NA	NA	NA	NA
	Panel B	126.1	23.9	21.9	21.6
	Panel C	217.3	20.3	21.0	20.5
	Panel D	297.7	14.0	15.8	15.0
	Panel E	397.2	15.6	15.2	14.8
	Panel F	559.7	17.2	16.6	16.7

Table 10. Site 3 Precision study (N=720)

Peptide	Panel	Mean (pmol/L)	Intra-day (%)	Between Day (%)	Between operator (%)
ADA 93	Panel A	391.3	31.7	37.5	47.6
	Panel B	1066.2	22.0	36.0	33.7
	Panel C	1501.3	21.7	25.2	24.0
	Panel D	2224.6	18.7	24.0	25.1
	Panel E	2920.6	15.9	20.9	23.8
	Panel F	4178.2	20.2	31.0	34.6
ATP7B 1056	Panel A	68.0	NA	NA	NA
	Panel B	63.1	18.9	19.2	18.5
	Panel C	80.9	28.0	28.4	27.7
	Panel D	128.4	23.1	25.2	25.9
	Panel E	178.6	21.2	24.4	23.4
	Panel F	259.3	24.7	26.7	25.5
ATP7B 887	Panel A	71.3	36.8	37.8	38.1
	Panel B	92.4	33.0	35.6	37.3
	Panel C	123.7	24.9	26.9	28.1
	Panel D	156.9	20.8	25.5	25.9
	Panel E	197.0	21.5	22.5	21.8
	Panel F	260.6	20.6	23.3	22.3
BTK 407	Panel A	85.9	38.1	49.2	55.0
	Panel B	101.7	38.1	51.7	49.4
	Panel C	191.5	39.9	41.2	40.6
	Panel D	276.8	21.6	23.8	23.6
	Panel E	371.4	21.9	24.7	23.6
	Panel F	482.2	21.3	22.9	23.0
BTK 545	Panel A	166.0	NA	NA	NA
	Panel B	153.3	19.5	22.6	21.5
	Panel C	230.5	38.5	42.4	47.1
	Panel D	360.0	30.6	32.2	31.4
	Panel E	528.1	23.0	24.9	25.8
	Panel F	670.5	24.7	24.3	23.5
WASP 274	Panel A	83.5	50.4	48.3	50.3
	Panel B	114.8	30.1	40.7	38.0
	Panel C	181.8	32.0	33.6	32.2
	Panel D	244.4	21.8	22.6	23.5
	Panel E	321.2	22.6	26.9	26.1
	Panel F	402.7	24.8	26.2	25.7

12.2 Analytical Sensitivity: The limit of blank (LOB), limit of detection (LOD) and limit of quantitation (LOQ) for the test kit were determined following the recommendations in the CLSI EP17-A2 guideline with consultation of CLSI document C64 "Quantitative Measurement of Proteins and Peptides by Mass Spectrometry", involving replicate analysis of a set of blank and low-level samples using multiple lots and a single instrument.

The LOB, LOD and LOQ are summarized in **Table 13**.

Table 13. Analytical sensitivity

Detection Capability (pmol/L)	N	ATP7B 887	ATP7B 1056	WASP 274	ADA 93	BTK 407	BTK 545
Limit of Blank	120	15.5	13.7	6.2	77.5	7.5	2.6
Limit of Detection	120	24.5	22.7	23.9	172.7	21.4	13.3
Limit of Quantification	180	40.2	49.6	51.4	197.8	41.2	108.8

5 outliers were identified and removed them before determining the LOD and LOQ, as detailed below:

ATP7B 887: outlier A (z-score 2.4), C (z-score 2.5) and D (z-score 2.2)
ATP7B 1056: outlier A (z-score 2.0) and B (z-score -1.9)
WASP 274: outlier C (z-score 3.0)
BTK545: outlier E (z-score 3.5)
BTK407: outlier D (z-score 3.0) and E (z-score 3.2)

12.3 Linearity: The linearity study was performed following the recommendations in the CLSI EP06 guideline using two lots of reagents. The linearity of the assay was determined by replicate measurements of DBS samples with a range of endogenous peptide concentrations. These specimens consisted of fish blood and normal human control (NHC) blood in percentages from 0-100% NHC. For each peptide, the assay shows linearity for the intervals reported below, with deviations from linearity within 30%.

Table 14. Linearity range

Target Peptide	Linearity Range (pmol/L)
ATP7B 887	269.8-430.8
ATP7B 1056	70.3-451.9
WASP 274	51.4-547.0
ADA 93	197.8-3753.5
BTK 545	108.8-311.4
BTK 407	55.3-1290.7

12.4 Carry-Over: Carryover was determined by replicate measurements of blank samples with and without carryover for each multiplexed peptide**.** The blank samples were run followed by a DBS sample with high analyte concentrations for each multiplexed peptide and repeated to evaluate change in concentration due to carryover. The carryover effects of all peptides except ADA 93 were negligible. Based on the results, a specimen with ADA concentration within or slightly higher than the borderline cutoff should be retested if a specimen with ADA concentration within the normal range is present in the same column.

Table 15. Carry-over

Target Peptide	Carryover (%)
ATP7B 887	0.2-4.1
ATP7B 1056	0.9-4.2
WASP 274	0.1-0.6
ADA 93	5.0-21.2
BTK 545	0.4-2.0
BTK 407	0.0-0.8

12.5 Interference: Analytical specificity was determined in accordance with the CLSI EP7-A2 guideline. The interferents tested for the kit are as follows: Hematocrit, Unconjugated Bilirubin, Conjugated Bilirubin, Galactose, Glucose, EDTA, Heparin, Total Protein, Hemoglobin, and Triglyceride. Blood pools were made in both 100% fish blood and a 30% mixture of NHC and fish blood. Test pools were spiked with interfering substances. None of the 11 interferents tested in this experiment had a significant effect on the concentrations of all six peptides at the recommended initial interferent concentration levels (below 20%).

Table 16. Interference

Interference	Recommended Concentration	Unit
Unconjugated Bilirubin	0.4	mg/mL
Conjugated Bilirubin	0.4	mg/mL
Galactose	0.6	mg/mL
Glucose	10	mg/mL
EDTA	3.4	µM
Heparin	3.3	µg/mL
Total Protein	50	mg/mL
Hemoglobin	10	mg/mL
Cholesterol	400	mg/dL
Triglycerides	1500	mg/dL
Gamma-globulin	2.5	g/dL

The effect of hematocrit was tested by adjusting the amount of red blood cells to 40%, 50%, and 60% following the CLSI EP7-A2 guideline. The samples were constructed from mixtures of human and fish blood product. Human plasma and packed RBCs were mixed as described by CLSI guidelines NBS-04, Appendix F. 0%, 30%, and 100% human blood at 40/50/60% hematocrit was tested. The assay was not subject to interference for samples with low (40%) or high (60%) hematocrit with 30% human blood concentrations. However, the assay was subject to interference for five peptides (ATP7B 1056, ATP7B 887, ADA 93, BTK 407, and BTK 545) in samples in low hematocrit (40%) with 100% human blood concentration.

12.6 Outlier Rate: As detailed in section 11.4, a total of 5 outliers were observed in all the analytical studies from three sites (LOB/LOD/LOQ). Specifically, outliers were observed at a rate of 0.71% for ATP7B 887, 0.48% for ATP7B 1056 and BTK 407, and 0.24% for WASP 274 and BTK 545.

12.7 Stability: Stressed stability test was conducted in collaboration with Nelson Laboratories (Salt Lake City, UT). 3 kits were randomly selected from the manufactured lot and placed into controlled chambers. These kits were taken through a specified stress sequence (40 ˚C, -20˚C, 30˚C, and 2-8˚C) while the remaining the kits in the lot were stored at the recommended conditions of 2-8˚C. Temperature extremes affected ATP7B 887, ATP7B 1056, and WAS 274 concentrations.

The study was conducted to determine the stability of screening kit using a classical and real-time approach as described in the CLSI EP17-A2 guideline. In-use 30 days after open kit testing showed stable for up to 1 month, however the difference bias has increased at 6 months. Real time stability at 4˚C at 3 months and 6 months showed high bias likely due to degradation of IS plate. Frozen stability at -20˚C showed stable peptide concentration through 6 months with low CV and bias. We recommend storing the kit at -20˚C to prevent the internal standard from degrading over time and the properly stored kits are stable up to 6 months.

On-board stability testing was conducted by replicate panel of eluted samples placed into the instrument sample manager and measuring across time (0, 4, 8, 24, 32 and 48 hours). Differences in the six peptide concentrations across all time points were below 25%.

13 Key Proteo Newborn Screening Clinical Validation Study and Cutoff Determinations

12.1. Clinical Sensitivity
Not applicable

12.2. Clinical Specificity
Not applicable

12.3. Other Clinical Supportive Data

12.3.1. To establish the initial cutoff values, Key Proteo performed a preliminary study prior to the clinical study by analyzing 1,000 presumed normal de-identified samples and 12 positive samples. The cutoffs were selected to ensure that all known affected specimens would be detected and to minimize the false positive rate. The cut-off for ATP7B 887, 1056 was set for 25% of median values in pmol/L, 20% of median values in pmol/L for ADA 93 and the rest, BTK 407, 545 and WASP274 were set for 10% of median values in pmol/L. If the target peptide concentration was lower than cutoff, retest on remaining sample was performed. Only male newborns were reported for WAS and XLA test results. All positive samples were sequenced.

The initial cutoff used at the start of the study is shown in **Table 17**. The average peptide concentrations were not affected by the age of sample collection up to 7 days or gender for WASP274, BTK545 and BTK407.

Table 17. Initial proposed cutoff

	ATP7B 887	ATP7B 1056	WASP 274	ADA 93	BTK 545	BTK 407
Initial	66.6	67.5	226.4	1145.6	156.4	112.2

12.3.2. The screening performance of the kit was determined in a prospective clinical study of routine newborn screening samples and previously confirmed positive patient samples which have been saved in the biorepository at -20C for WD, XLA, WAS and ADAD. Six of them were original newborn blood spots from affected patients. A total of 3,294 newborns and 49 genetically confirmed positive samples were tested at three sites. No presumptive positive cases were detected at the three sites.

Table 18. Clinical study cohort

Site	NBS samples	Screen Negative	False Positive rate
1	601	601	0
2	601	601	0
3	2092	2092	0

Table 19. Blinded patient study cohort

Site	Patient samples	Screen Positive	False Negative rate
1	15	15	0
2	14	14	0
3	20	20	0

All confirmed 49 positive cases were screen positive, and repeats were concordant with initial results. Four confirmed Wilson disease cases had ATP7B 887 level above the cutoff while ATP7B 1056 level were below the cutoff. Two Wilson disease patients had ATP7B 1056 level above the cutoff but ATP7B 887 level were below the cutoff. In one confirmed case of Wilson disease, WAS 274 level was below cutoff. Sequencing showed the variant of uncertain significance.

Table 20. Confirmed patient cohort

Site	WD	XLA	WAS	ADAD	Total
1	10	2	2	1	15
2	9	3	1	1	14
3	14	3	2	1	20

12.3.3. To estimate the potential for false negative results of the test, the samples presumed to be normal were evaluated using next generation sequencing in a clinical molecular laboratory. Specimens included 20% or higher of the median values in pmol/L and were pulled from three different sites. (A total of 100 newborn samples).

ATP7B gene: Heterozygous genetic variants of uncertain significance (VUS) were detected in six routine samples and heterozygous pathogenic variants were detected in two routine samples that were screened negative. The second variants were not detected in those samples. In one routine case, two variants of uncertain significance were detected in which the screen was negative. The clinical significance is yet uncertain.

ADA gene: Three heterozygous variants of uncertain significance and one heterozygous pathogenic variant was detected that were screen negative. The second variants were not detected in all these cases.

BTK gene: One variant of uncertain significance was detected in a case that was screened negative. The clinical significance is yet uncertain.

12.4. Interpretation of Results

12.4.1. Applying cutoff and retesting

- Specimens with initial test results above the cutoff for all six peptides were presumed negative (or normal). No additional test was taken.
- Specimens below initial cutoff was retested and reported presumed positive when the target concentration was still below the cutoff.
- For XLA and WAS, the specimens from females were not reported regardless of the test results.

- The specimen in which two or more target peptides were below cutoff was considered potentially poor-quality sample.

13. **Expected Values and Interpretation of Results**

Please note that the values described in this insert should be used as a guideline only and each laboratory should establish their own cutoffs for their own populations.

- The measurement of target peptide concentrations for WD, XLA, WAS, and ADAD from the dried blood spots specimens is performed by using a cutoff concentration that distinguishes between presumed affected and presumed normal newborns. Each laboratory should follow its own procedures for establishing cutoffs. For the diseases targeted by the Newborn Screening kit, specimens with peptide concentrations below the cutoff are highly likely to have the specific diseases and should be referred for diagnostic follow-up.

- It is recommended that each laboratory establish cutoffs per laboratory guidelines and any applicable local, state, and/or federal requirements. It is recommended to include as many true positive samples as possible to estimate the cutoff values with higher confidence levels.

- The cutoffs should be selected to ensure that all known affected samples will be detected and to minimize the false positive rates, keeping in mind the expected incidence of the diseases.

- As larger numbers of samples are screened and presumed positive results are obtained, cutoffs should be reviewed in consultation with specialists who can provide additional guidance based on incidence rates, disease severity, and typical profiles of known positive patients. It is also recommended that the performance of the test be monitored for seasonal variability (for example by monitoring daily or weekly medians). If a laboratory chooses to apply a two-tiered cutoff, it is recommended that a borderline cutoff value for each analyte be set above the cutoff.

- Samples with results above the cutoff for all target peptides are considered low risk and should be presumed negative (normal). Local regulations and guidelines should be followed for the handling and reporting of presumed normal results. Samples that fall below the cutoff should be retested for confirmation. If the repeated sample results are different from the first result, repeat test for a third time for clarification.

14. **Appendices**

15. References

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Advancing Newborn Screening in Washington State: A Novel Multiplexed LC-MS/MS Proteomic Assay for Wilson Disease and Inborn Errors of Immunity

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Newborn Screening (NBS) is considered one of the most successful public health programs in identifying infants with treatable disorders for early intervention with favorable outcomes. Unfortunately, for many genetic disorders there are no specific metabolic biomarkers nor any analytical methods suitable for population screening even where highly effective pre-emptive treatments are available. Most causative mutations in genetic disorders result in reduction or absence of their proteins, therefore, direct measurements of the peptide as a surrogate marker for these proteins using multiplexed proteomic methods from dried blood spots (DBS) can be highly diagnostic and utilized in population screening.

Direct measurement of signature peptides in DBS has been shown to be a sensitive and specific proteomic screening method for the multiplex detection of patients with Wilson Disease and three life-threatening inborn errors of immunity, X-linked agammaglobulinemia, Wiskott-Aldrich syndrome, and Adenosine Deaminase deficiency. Each of these disorders results in severe negative sequelae if undetected but are treatable if diagnosed early in life. Analysis of signature peptides found statistically significant reduction or absence of peptide levels in affected patients compared to control groups in each case.

A novel proteomic-based IVD (*Invitro* diagnostics) kit for NBS has been manufactured to identify these four conditions in a single-run multiplex assay from DBS with inject-to-inject time at < 3 minutes using LC-MS/MS. For the FDA De Novo Class II application, the screening performance of the kit was determined in a prospective clinical study of routine newborn samples and previously confirmed positive patient samples. In this validation study, a total of 3,294 newborns and 49 genetically confirmed positive samples were tested at three sites. No presumptive positive cases were detected, and all confirmed 49 positive cases were screen positive. Analytical performance including stability, day to day reproducibility, and inter-operator variation was all acceptable at three sites. A pilot study is also underway in conjunction with the WA State public health NBS laboratory. To date, more than 22,000 newborn samples have been screened, and no true presumptive positive cases have been found with only a few false positive cases detected. These ongoing studies support both the feasibility of newborn screening for these conditions and the use of multiplexed proteomic analysis as an effective methodology for pediatric population screening.

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Population screening for Wilson's disease

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Wilson's disease is an autosomal recessive disorder of copper transport caused by mutations in the gene encoding an ATPase, *ATP7B*. Early detection of Wilson's disease is critical because effective medical treatments such as chelating agents and zinc salts are available, which can prevent lifelong neurological disabilities and/or cirrhosis. It is unfortunate that most patients are brought to our attention after they have developed serious complications such as brain damage or cirrhosis, despite the availability of effective treatments. The diagnosis is usually made through copper measurement in the liver tissue, followed by confirmation with genetic testing of the *ATP7B* gene. Currently, there are no effective biomarkers or methods suitable for newborn screening for Wilson's disease. Ceruloplasmin has been tested for pediatric and newborn screening with limited outcome. Recently, liquid chromatography–multiple reaction monitoring–mass spectrometry (LC-MRM-MS) has emerged as a robust technology that may enable multiplex quantification of signature proteotypic peptides with low abundance. The application of this technology may help facilitate the research on Wilson's disease for protein expression, biomarker study, diagnosis, and, hopefully, screening.

Keywords: Wilson's disease; newborn screening; population screening; ceruloplasmin; ATP7B; LC-MS/MS; LC-MRM-MS

Wilson's disease has evolved from a diagnosable to a treatable disease over the 100 years since the first cases were reported in 1912 by S.A.K. Wilson.[1] The disease is progressive and ultimately fatal if untreated and is probably one of the most frequent causes of chronic liver disease in children. Although the prevalence varies across the population, it was estimated to be 1 in 30,000 with a carrier frequency of 1 in 90.[2] Over 500 mutations have been reported, along with >100 polymorphisms, in the *ATP7B* gene in the Human Genome Mutation Database (http://www.hgmd.cf.ac.uk/ac/gene.php?gene=ATP7B). The most common mutation in Europe and North America is p.H1069Q, while p.Arg778L is the most common in Far Eastern Asian populations.

Clinical and laboratory ascertainments for Wilson's disease are often very challenging, as clinical presentations of Wilson's disease show considerable variations and may be even much broader than we know. Guidelines for the diagnosis of Wilson's disease have been approved;[3] however, a small number of patients still cannot be diagnosed with current tests. Despite recent improved awareness and advanced DNA testing, a substantial number of patients still present with delayed diagnosis, which is unfortunate when we have effective treatments available that include copper chelators (trientine, penicillamine) to promote copper excretion from the body and zinc salts to inhibit the copper absorption from the gastrointestinal epithelium. Early diagnosis is critical for better prognosis. Patients with neurological symptoms may experience worsening with the treatment, and half of those patients may develop permanent neurological damage and will never return to the baseline of function.[4] Hepatic presentations range from acute and chronic hepatitis to cirrhosis and acute liver failure. Hepatocellular carcinoma has become an important recent issue for patients with Wilson's disease, as current treatment has improved life expectancy.

Considering all these factors, population screening for Wilson's disease has been extensively discussed, with strong needs; however, no screenable,

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Table 1. Results of population screening trials for Wilson's disease

Year	Study	No. of children	Age range	Positive cases	Method/sample
1993–1995	Yamaguchi *et al.*[7]	126,810	Newborns	0	ELISA, DBS
1977–1996	Yamaguchi *et al.*[7]	24,165	Late infancy to elementary school	3	ELISA, DBS
1999	Ohura *et al.*[8]	2789	1–6 years	2	ELISA, DBS
2001	Hahn *et al.*[11]	3667	3 months to 15 years	1	ELISA, DBS
2002	Owada *et al.*[9]	48,819	Primary school children	2	ELISA, urine
2006	Kroll *et al.*[12]	1380	Newborn to 18 years	0	ELISA, DBS
2008	Nakayama *et al.*[10]	11,362	3 years	1	ELISA, urine
2008	Zappu *et al.*[14]	5290	Newborn (Sardinia)	1:2707	PCR-based, DBS
		397	Newborn (Kalymnos)	27:20,000	

DBS, dried blood spots; ELISA, enzyme-linked immunosorbent assay.

cost-effective biomarkers or methods have yet been developed for Wilson's disease. To date, only a few studies have been conducted using ceruloplasmin as a marker, with limited outcomes (Table 1).

Newborn screening seeks to identify infants with treatable congenital disorders. Recent tandem mass spectrometry (MS/MS) applications have provided the ability to screen for >50 metabolic diseases from a single dried blood spot. The feature that makes metabolic disorders particularly amenable to screening is the presence of abundant small-molecule metabolites in plasma. However, many treatable disorders are characterized by absent or diminished large proteins in plasma or within circulating blood cells, for which there are currently no cost-effective screening methods. The research in our laboratory has focused on developing mass screening methods for these conditions, including Wilson's disease.

Previous experiences from pilot studies

Serum copper and ceruloplasmin are low in the majority of patients with highly elevated copper concentrations in the liver (>250 μg/g of tissue) and in the urine (>100 μg/day).[3] In some patients with Wilson's disease, serum ceruloplasmin is not low, and serum copper could be high in patients suffering from acute liver failure, owing to the release of copper from hepatocytes. Ceruloplasmin, a major copper-carrying protein in the blood, is a secretory enzyme that plays a role in iron metabolism as a ferroxidase. Ceruloplasmin is synthesized mainly by hepatocytes and incorporated with copper by the ATP7B protein before secretion into the blood. Earlier studies indicated that holoceruloplasmin (ceruloplasmin bound with copper) levels in plasma from patients with Wilson's disease were reduced, but the amount of apoprotein (ceruloplasmin without copper) was similar to that observed in normal individuals.[5] Based on this observation, a sandwich enzyme-linked immunosorbent assay (ELISA) kit was developed in Japan using a monoclonal antibody specific to holoceruloplasmin to measure the ceruloplasmin in dried blood spots.[6] Nevertheless, no patients with Wilson's disease were detected after screening over 126,810 newborns.[7] However, three patients were identified from 24,165 children from infancy to elementary school age in Japan. In another subsequent study using the same sandwich ELISA kit, two presymptomatic patients were identified among 2789 children aged from 1 to 6 years.[8] As a result of these studies, 3 years was considered to be the optimal age for Wilson's disease screening.

A mass screening measuring urinary holoceruloplasmin was also conducted in Japan.[9] The investigators found that the quantity of holoceruloplasmin protein in the urine of patients with Wilson's disease was significantly lower than in healthy subjects. However, in 2 of 41 Wilson's disease control samples (5%), urinary ceruloplasmin was not low, supporting previous observations that some patients with Wilson's disease could have normal ceruloplasmin levels. Two cases of Wilson's disease were found by testing urine samples from 48,819 primary school children. The rate of second request was about 0.9%. Another pilot study was conducted in Hokkaido Prefecture in Japan for 3-year-old children to screen for Wilson's disease using an automated assay of ceruloplasmin measurement in urine specimens.[10] A total of 11,362 children were screened, and one

true positive case was identified and confirmed by genetic testing. The urinary ceruloplasmin level in Wilson's disease controls was 13 ± 9.25 ng/mg Cr, while the normal control was 190 ± 154 ng/mg Cr. When the cut-off value was 45.0 ng/mg Cr, the rate of second request was about 1% of total participants.

We also developed a sandwich ELISA method for ceruloplasmin measurement in dried blood spots using specific monoclonal antibodies against ceruloplasmin. In a small pilot study on 3667 children aged 3 months to 15 years, we identified one patient, a 32-month-old boy with a ceruloplasmin concentration of 2.3 mg/dL.[11] The rate of second request of this study was 0.3%. In a subsequent validation study, we were able to retrieve the original newborn blood spots from two affected patients with Wilson's disease along with age-matched controls.[12] The original newborn blood spots were kept in a –20 °C freezer. Ceruloplasmin concentrations in the original newborn blood spots from two patients were indeed very low: 2.6 and 2.8 mg/dL (normal control 47.2 ± 15.6 mg/dL with range from 6.5 to >60). It is conceivable that the ceruloplasmin concentration in Wilson's disease could be much lower during the newborn period compared to normal newborns, but further study will be required to determine the baseline level of ceruloplasmin in newborns with Wilson's disease. Although these findings supported the view that presymptomatic screening for Wilson's disease using dried blood spots could be feasible in the newborn period, subsequent screening over 100,000 newborn blood spots in Minnesota was unsuccessful in identifying a patient (unpublished data), similar to a previous experience in Japan.

We later developed an assay using liquid chromatography–triple quadrupole mass spectrometry (LC-MS/MS) to quantify ceruloplasmin-specific peptides from dried blood spot samples digested by trypsin.[13] Most state laboratories in the United States currently use LC-MS/MS for newborn screening. A method-comparison study on previously tested patient samples for ceruloplasmin gave comparable results, suggesting that it may be feasible to use LC-MS/MS for screening. Lower-limit quantification for ceruloplasmin was around 0.7 mg/dL. Inter- and intra-assay imprecision was acceptable for clinical use; however, this approach has not been studied in a large-scale population.

DNA analysis can be utilized for a genetically homogeneous group with highly prevalent mutations. In Sardinia, a 15-nucleotide deletion in the promoter region of *ATP7B* accounts for 61.7% of alleles. Six common mutations together account for approximately 85% of Wilson's disease in that population. By screening 5290 newborns in Sardinia, Wilson's disease incidence in this population was estimated to be approximately 1 in 2707 live births.[14]

Challenges

Ceruloplasmin is an acute-phase reactant, so the concentration can vary depending on the health status at the time of sample collections. Ceruloplasmin can be in the normal range in some symptomatic patients with Wilson's disease. Low ceruloplasmin can be observed in other conditions such as copper deficiency, Menkes disease, hereditary aceruloplasminemia, severe protein-losing enteropathy, or liver failure. Very little information is available for the baseline concentration of ceruloplasmin in Wilson's disease before patients develop symptoms.

The application of ELISA or immunoassay for ceruloplasmin quantification to newborns, regardless of different antibodies against ceruloplasmin epitopes, may not be sensitive enough to identify newborn patients with Wilson's disease. A substantial number of newborns present with physiologically low ceruloplasmin, which makes this approach difficult. The method using LC-MS/MS developed in our laboratory for ceruloplasmin measurement may also encounter similar problems with sensitivity and detection limit.

However, ceruloplasmin measurement by immunoassay, either on dried blood spots or urine specimens, could be a feasible approach for infants or older children. Indeed, pediatric or infant screening by ceruloplasmin assay seems the only amenable approach for screening Wilson's disease at present. The limited studies in Japan yielded some promising results. The cost is only $2.50 per test, but high false-positive rates, particularly for the urine test, necessitate considerable improvement of the assay. A disadvantage of this screening is that it would be effective only when used in conjunction with a mandatory health care program available at the age of 3 years. This system is not universally available worldwide and may be difficult to establish in the United States with limited federal resources.

Molecular genetic testing is useful in confirming the diagnosis in affected patients, but is also very useful in identifying affected siblings, including

those without definite symptoms.[15,16] Although no single test can permit *de novo* diagnosis of Wilson's disease, the genetic test has been increasingly used in the clinical field and often underscores the important medical decision. Genetic tests play critical roles in the diagnosis of Wilson's disease. A stepwise approach with DNA-based screening in certain ethnic backgrounds or regions, starting with common mutations accounting for the majority of Wilson's disease patients in that region, may be an appropriate strategy.[17] Although the continuous decrease in the cost of DNA testing will render the screening more approachable, the current cost is still prohibitive for mass screening for Wilson's disease.

Recent investigation on 181 patients from the United Kingdom with clinically and biochemically confirmed Wilson's disease showed that overall mutation detection frequency was 98%.[18] The likelihood of mutation in genes other than *ATP7B* causing Wilson's disease is very low. Their study indicates that the frequency of heterozygosity is considerably higher than the previously reported occurrence of 1:90. The calculated frequency of individuals predicted to carry two pathogenic mutations was 1:7026, considerably higher than the reported prevalence of Wilson's disease of 1:30,000. This significant discrepancy between the genetic prevalence and the number of clinically diagnosed cases of Wilson's disease was explained by both a reduced penetrance of *ATP7B* mutations and a failure to diagnose patients. This underlines the need for more reliable biomarkers for screening Wilson's disease to prevent the development of serious complications.

As serum copper is reduced in Wilson's disease, measuring copper in dried blood spots was considered as a potential screening method. Unfortunately, copper is an environmentally abundant heavy metal; we detect very high copper content randomly in dried filter papers, which makes screening for Wilson's disease using dried blood spots unfeasible.

Future development

Many treatable congenital disorders are caused by mutations that result in absent or diminished levels of proteins; thus, protein biomarkers have enormous potential in the diagnosis/screening of congenital disorders. LC-MRM-MS has emerged as a robust technology that enables highly precise, specific, multiplex quantification of signature proteotypic peptides as stoichiometric surrogates of biomarker proteins. As proof of concept, we have developed a novel proteomic screening approach using LC-MRM-MS to simultaneously identify specific signature peptides derived from the transmembrane protein CD3ε (a general marker for T cell number) and the intracellular proteins WASP and BTK (expressed in B and myeloid cells) as markers of three life-threatening primary immunodeficient diseases (PIDD): severe combined immune deficiency (SCID), Wiskott–Aldrich syndrome (WAS), and X-linked agammaglobulinemia (XLA).[19] Blinded patients' peripheral blood mononuclear cell (PBMC) samples with sufficient actin recovery were successfully analyzed for BTK, WASP, and CD3ε signature peptides, using an actin peptide for normalization, to accurately identify patients with PIDD.

With depletion of high-abundance proteins, we were also able to identify signature peptides for BTK and WASP in the dried blood spot extract, along with their isotopically labeled standards. This is compelling evidence that detection of low-abundance proteins in dried blood spots will be feasible by employing specific enrichment techniques using antipeptide antibodies. Our lab is currently exploring the use of peptide immunoaffinity enrichment to overcome a number of caveats, including limited sensitivity and reproducibility, especially for low-abundance proteins or small sample volumes.[20,21] Our preliminary analysis indicates that candidate signature peptides for ATP7B in HepG2 cells can be identified, along with their isotopically labeled internal standards (unpublished data). A substantial proportion of Wilson's disease–associated missense mutations, including p.H1069Q and p.R778L, result in markedly decreased levels of the ATP7B protein caused by enhanced degradation.[22–24] Other prevalent mutations, such as protein-truncating nonsense mutations (~13% of known point mutations)[25] and frameshift mutations,[26] are predicted to result in the absence or decay of mRNA[27,28] or a severely truncated protein, resulting in absent or diminished protein levels. Taken together, it is expected that most patients with Wilson's disease would have absent or significantly reduced levels of ATP7B. Nevertheless, it is still not known whether the MRM-based technique could be sensitive enough or feasible for screening patients or newborns using blood-based specimens. The expression of ATP7B protein

in blood-based specimens may not be high enough to be detected, even with this technology.

In summary, early recognition, diagnosis, and preemptive treatment are critical for improving outcomes in Wilson's disease. Wilson's disease meets the universal criteria for mass population screening, except that there is no cost-effective method yet available. Although the optimum time to screen is disputable, the newborn period has the advantage of an infrastructure that is currently in place for blood spots and screening worldwide. The current ceruloplasmin assay may be suitable for infant or pediatric screening around the age of 3 years, but there is a lack of outpatient-based mandatory programs, which is a significant barrier to implementation. A study using LC-MRM-MS technology is in progress, but at this point, no scientific data is available. New biomarkers and the development of new methods should be continuously explored to provide early and effective preemptive treatment that can drastically improve outcomes in patients with Wilson's disease.

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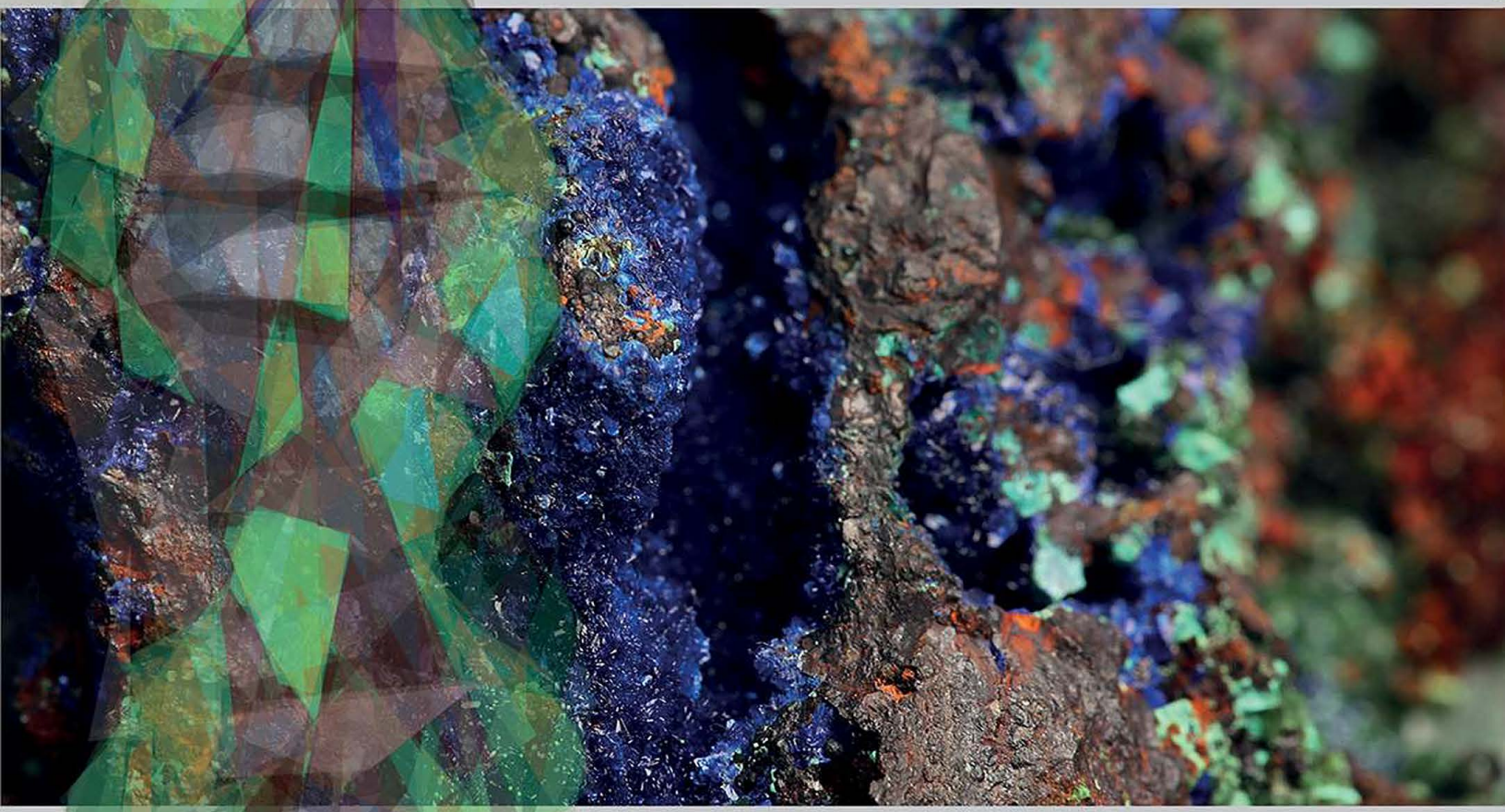

Chapter 26

Population Screening for Wilson Disease

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BACKGROUND

Wilson disease (WD, OMIM 277900) is an autosomal recessive disorder of copper metabolism with an estimated prevalence of one in 30,000 individuals and carrier frequency of one in 90 individuals in most populations [1]. The only gene known to be associated with WD is *ATP7B*, which encodes a copper-transporting ATPase ATP7B (Wilson ATPase) [2–5]. Over 500 different pathogenic variants and >100 polymorphisms have been identified in *ATP7B* in the Human Genome Mutation Database (http://www.hgmd.cf.ac.uk/ac/gene.php?gene = ATP7B), and most affected patients are compound heterozygotes, with one each of two different mutations [6]. There is wide geographic variation in *ATP7B* mutations [7–9]. These common mutations cause enhanced degradation and decreased levels of ATP7B, the ATPase abnormal in WD [10–13].

Effective treatment for WD is widely available and affordable, highlighting the importance of early detection and intervention. Copper chelators such as penicillamine and trientine bind excess copper and promote excretion, and zinc salts inhibit enteral copper absorption [14–18]. These treatments are highly effective in preventing the downstream morbidity and early mortality associated with WD [15–19]. Zinc monotherapy has also been demonstrated to be an effective long-term maintenance treatment for WD [20–24]. Left untreated, half of affected patients with neurologic symptoms may develop permanent neurologic damage [25]. Patients with chronic liver disease may progress to hepatocellular carcinoma [26,27]. Therefore, the ideal time to detect WD and begin treatment is prior to onset of symptoms for the best long-term clinical outcome.

NEWBORN SCREENING

Newborn screening is implemented by well-established public health programs in most developed countries. Each year, over 4 million newborn babies are screened for congenital disorders in the United States, and over 4000 infants are diagnosed as having a condition [28]. Given the incidence of WD, over 130 infants are likely born each year with WD.

Guidelines for deciding whether a particular condition is a suitable candidate for newborn screening was first formulated by Wilson and Jungner in 1968 and revised by the World Health Organization in 2008 [29,30]. Unfortunately, many aspects of these guidelines are subjective, and there is not always agreement about which disorders to include in the newborn screening panel [31]. Therefore, despite existing guidelines, there are now varying newborn screening panels worldwide. Nonetheless, given the aforementioned factors of high prevalence, severe disease burden, and available treatment, WD has always been an attractive target for newborn screening. However, no cost-effective, specific biomarkers or screening methods have been developed for WD. Previous studies have evaluated the predictive value of low ceruloplasmin, urinary copper, and holoceruloplasmin, with limited outcomes. The last decade has remained largely devoid of new studies on biochemical screening for WD.

The application of tandem mass spectrometry (MS/MS) has recently expanded the ability to screen for >50 metabolic diseases from a single dried blood spot collected on filter paper [32–36]. Inborn errors of metabolism are particularly amenable to screening due to the accumulation of small metabolites, which are detectable by mass spectrometry

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[37]. Many treatable disorders such as WD remain difficult to detect as they are characterized by absent or diminished levels of proteins or metabolites present only within leukocytes.

Recently, the emergence of liquid chromatography–selected reaction monitoring–mass spectrometry (LC–SRM–MS) has enabled multiplexed quantification of low-abundance signature proteotypic peptides [38,39]. The application of this technology may help facilitate research on WD for measuring protein abundance, identifying biomarkers, and enabling early diagnosis. Our laboratory has demonstrated a promising method of screening for WD by peptide immuno-SRM assay, raising the possibility of population screening for WD using peptide biomarkers for the ATP7B protein [40,41].

COPPER SCREENING

Most patients with WD have elevated serum non-ceruloplasmin-bound copper (>25 μg/dL), making serum copper quantification a logical potential target for screening in dried blood spots. Relative exchangeable copper (REC = exchangeable copper/total copper) has been proposed as a reliable, sensitive, and specific biomarker for screening and diagnosis of WD in adults (see Chapter 22: Direct Determination of Non-Ceruloplasmin-Bound Copper in Plasma). However, reference ranges have not been established for newborns or children [42]. Copper is also a naturally occurring abundant heavy metal in the environment. In a random sample of commercial filter papers, we detected very high copper concentrations, eliminating the possibility of screening for WD using any copper measurements in dried spots for both blood and urine.

CERULOPLASMIN SCREENING

Ceruloplasmin is an α_2-glycoprotein that contains 90% of circulating copper in the body [43]. Ceruloplasmin is initially synthesized in hepatocytes as apoceruloplasmin. Copper is then incorporated into apoceruloplasmin by the Wilson ATPase to form holoceruloplasmin before secretion into circulation. In WD, the absence of copper binding causes ceruloplasmin to be rapidly degraded by plasma proteases, leading to low plasma ceruloplasmin in most affected patients. Ceruloplasmin is an acute phase reactant, and the presence of low ceruloplasmin levels should be cautiously interpreted as other medical conditions may also lead to low ceruloplasmin (see Chapter 9: Ceruloplasmin). The majority of patients with WD have low plasma ceruloplasmin levels (<20 mg/dL or < 200 mg/L); however, some affected patients have been shown to have borderline or normal ceruloplasmin levels. Up to 20% of heterozygous carriers for WD may also have low-normal range serum ceruloplasmin levels [44]. Previous studies have demonstrated low diagnostic accuracy of serum ceruloplasmin for WD screening [45]. Using the conventional cutoff of 20 mg/dL, the estimated positive predictive value for WD was only 48.3% in adults with WD compared to carriers and controls [46]. A retrospective review of pediatric patients with hepatitis revealed accuracy of a ceruloplasmin level of <20 mg/mL of 84.8% [47].

Previous studies have demonstrated that while holoceruloplasmin levels were reduced in patients with WD, apoceruloplasmin levels were comparable to those observed in unaffected individuals [48]. Based on these findings, Endo and colleagues developed a sandwich enzyme-linked immunosorbent assay (ELISA) using a monoclonal antibody specific to holoceruloplasmin to measure ceruloplasmin levels in dried blood spots [49]. Their initial screening of 126,810 newborns did not detect any patients with WD, but their subsequent study of 24,165 infancy to elementary school-aged children identified three affected patients [50]. In another Japanese study using the same sandwich ELISA-based assay, two presymptomatic patients with WD were identified among 2789 children between 1 and 6 years old [51].

A pilot study of mass screening in 3667 children for WD in Korea using the sandwich ELISA assay identified one presymptomatic individual [52]. In a subsequent validation study, retrospective analysis of 353 newborn dried blood spots showed an average ceruloplasmin concentration of 40.0 ± 14.4 mg/dL [53]. Original newborn dried blood spots from two known affected patients were retrieved and ceruloplasmin concentrations were 2.6 and 2.8 mg/dL. A separate ceruloplasmin screening study on dried blood spots of ~100,000 newborns in Minnesota failed to detect any patients with WD (unpublished data). Ceruloplasmin levels in some newborns were unstable and physiologically lower than in older children and adults, and they continue to rise to adult levels 12 months after birth before stabilizing around 3 years of age [50]. Therefore, the optimal age of ceruloplasmin-based screening was thought to be ~3 years.

Other groups have studied population screening for WD by measuring urine holoceruloplasmin levels using sandwich ELISA assay. Owada and colleagues conducted mass screening of 48,819 primary school-aged children in Japan using urinary holoceruloplasmin and identified two presymptomatic individuals with WD [54]. Urinary holoceruloplasmin levels in the two affected patients were low at 1.9 and 9.3 ng/mg creatinine compared to 99.3 ± 77.7 ng/mg creatinine in unaffected 3–5-year-old children. However, two of the 41 WD control samples had higher levels of urinary

TABLE 26.1 Previous Population Screening Studies Using Ceruloplasmin Assays

Year	Study	Number of Subjects	Age Range	Positive Cases	CP in Positive Patients (mg/dL)	Mean CP (mg/dL)	PPV (%)/FP rate (%)	CP Cutoff Value	Methods/ Samples
1993–1995	Yamaguchi et al. [50]	126,810	Newborns	0	N/A	N/A	N/A	−2 to −2.5 SD	ELISA/DBS
1977–1996	Yamaguchi et al. [50]	24,165	Late infancy to elementary school	3	N/A	N/A (98.83% screened had CP > 20)	N/A	−2 to −2.5 SD	ELISA/DBS
1997	Cauza et al. [45]	2867	Adults	1 (then her sister)	N/A	±	PPV 5.9%	20 mg/dL	Radial immunodiffusion/ serum
1999	Ohura et al. [51]	2789	1–6 years	2	Pt1: 4.0,3.5 Pt2: 1.7,1.5	12.4 ± 3.95	FP rate 0.6%	3rd percentile	ELISA/DBS
2001	Hahn et al. [52]	3667	3 months to 15 years	1	2.3	30.5 ± 9.5	PPV 9.1%	15 mg/dL	ELISA/DBS
2002	Owada et al. [54]	48,819	Primary school children	2	Urinary HCP < 1.9, 9.3 ng/mg Cr, serum HCP 2.7, 7.0 mg/dL	99.3 ± 77.7 ng/ mg Cr Serum 21–37	N/A	3rd percentile	ELISA/urine
2006	Kroll et al. [53]	1398	Newborn to 18 years, retrospective	0	N/A, retrospective positive controls 2.6, 2.8	Newborn: 47.2 ± 15.5 Pediatric: 40.0 ± 14.4	N/A	< 15 mg/dL	ELISA/DBS
2008	Nakayama et al. [77]	11,362	3 years	1	13.3, 1.3, 24.9 ng/mg Cr	WD ctrl: 13.2 ± 9.25 ng/ mg Cr Normal ctrl: 190 ± 154ng/ mg Cr	N/A	1% of participants (<45.0 ng/mg Cr), 3 screenings	ELISA/urine
2008	Zappu et al. [70]	5290 397	Newborn (Sardinia) Newborn (Kalymnos)	1:2707 27:20,000	N/A	N/A	N/A	N/A	PCR-based/DBS

DBS, dried blood spots; *ELISA*, enzyme-linked immunosorbent assay; *CP*, ceruloplasmin; *HCP*, holoceruloplasmin; *Cr*, creatinine; *WD*, Wilson disease.; *N/A*, not available; *PPV*, positive predictive value; *FP*, false positive.

holoceruloplasmin within the range of normal controls, consistent with previous reports of patients with WD and normal ceruloplasmin levels. In addition, urinary holoceruloplasmin levels decreased after 3 days of storage in chlorhexidine gluconate at room temperature, and after 7 days at 4 °C. The accuracy and true predictive value were not reported. Furthermore, the study did not include phenotypic data or elaborate on the methodology behind which patients were selected for further testing. A follow-up study of urinary holoceruloplasmin levels in 11,362 primary school children in the island of Hokkaido identified one patient with WD, which was later confirmed molecularly. The wide range of normal values, uncertain accuracy, and instability for storage and transport make urinary holoceruloplasmin less plausible for newborn screening. Previous population screening studies using ceruloplasmin assays and polymerase chain reaction (PCR)-based mutation analysis are summarized in Table 26.1.

At present, assaying ceruloplasmin in late infancy or childhood appears to be the only cost-effective approach to population-based screening for WD. The studies in Japan had an average cost of USD $2.50 per assay, not adjusted for inflation. However, the childhood-based screening model relied on the mandatory healthcare program infrastructure in Japan, which is not universally available in other countries. In the United States, this program would be difficult to implement given limited federal resources, significant population influx and efflux, and lack of compulsory routine pediatric healthcare follow-ups.

From a technical standpoint, ELISA-based immunoassays for ceruloplasmin quantification in plasma and urine, regardless of type of antibody used against different ceruloplasmin epitopes, may not be sensitive enough to detect newborn patients with WD. Taken together, while most patients with WD appear to have low ceruloplasmin levels at baseline, the low positive predictive value, false positive rates in heterozygotes and normal newborns, and variable antibody binding makes ELISA-based assays less than ideal for population screening of WD.

MOLECULAR-BASED SCREENING

Previous studies have failed to detect biallelic pathogenic variants in *ATP7B* in up to 20% of patients with clinically diagnosed WD, raising suspicion for genetic heterogeneity [58]. However, it is more likely that novel variants, reduced penetrance, and unusual mechanisms of inheritance such as uniparental disomy or the presence of three concurrent mutations contribute to this discrepancy. Sequencing of the entire coding region and adjacent splice sites of *ATP7B* in 181 patients with WD in the United Kingdom showed an overall mutation detection frequency of 98%, strongly supporting monogenic disease [59]. The data also suggest a much higher prevalence of biallelic pathogenic variants in *ATP7B* than conventionally reported (that is 1:7026 instead of 1:30,000). Sequencing of all 21 exons of *ATP7B* is a time-consuming and cost-prohibitive approach to population screening for WD. However, targeted DNA analysis can be utilized in genetically homogenous populations with high prevalence of disease and allelic frequency for specific pathogenic variants. For instance, the incidence of WD is particularly high in Sardinia, where a molecular-based screening study of 5290 newborns estimated an incidence of one in 2707 live births [60]. In Sardinia, a 15-nucleotide deletion in the promoter region of *ATP7B* is implicated in 60.5% of patients with WD [61]. In aggregate, six common mutations constitute up to 85% of disease-causing alleles in that isolated population. In the Canary Islands, the L708P mutation is seen in up to 64% of patients [62].

In large, genetically heterogeneous populations such as China, the United States, and India, the most prevalent pathogenic variants have lower combined allelic frequencies; many affected individuals are compound heterozygotes for rare or novel mutations. In China, the two most prevalent mutations (that is, R778L and P992L) were present at a combined allelic frequency of approximately 40%–50% [57,63,64]. In Korea, the two most prevalent mutations (R778L and N1270S) account for 50% of cases [65]. In the United States and Europe, the allelic frequency of H1069Q accounts for approximately 30% of cases alone [8,66,67]. The allelic frequencies of common mutations in India vary largely by geographic region [55,56,68,69].

As the cost of molecular testing decreases and accessibility increases, a stepwise approach starting with DNA-based screening of common mutations accounting for the majority of cases in certain regions may become a feasible and rapid method for diagnosis. Indeed, Zappu and colleagues applied TaqMan technology to initially screen for the six most common mutations in Sardinia, followed by testing for the remaining 16 mutations in heterozygotes. They report a sensitivity of 94.6% with this stepwise screening approach [70]. Mak and colleagues demonstrated detection of 28 common mutations in WD patients in Hong Kong within 3 hours using a gene panel, real-time amplification refractory mutation system PCR, and green fluorescent dye for analysis [71]. In Taiwan, Lin and colleagues employed a three-tiered screening approach in 14 patients with WD and 50 normal controls. First, they screened for

the two most common mutations in exons 8 and 13 using High Resolution Melting analysis, followed by mutation analysis in exons 2, 5, 11, 12, 16, and 18 in negative cases, and finally examining the 5′UTR region and all remaining exons in twice-negative cases. Abnormal melting curves at each step were confirmed with direct DNA sequencing. They report that approximately 67.86% of *ATP7B* mutation alleles could be detected at the first step and 96.43% at the second step [72].

High-throughput technology, such as microarray chips, has also been examined for rapid, cost-effective screening of selective WD mutations [73,74]. However, the detection rate must be improved with follow-up direct sequencing. In China, a large-scale, two-step screening study based on array technology was recently conducted on 1222 patients with WD and 110 healthy controls [57]. Rapid multiplex PCR-MassArray was used to first screen for 110 mutational hotspots common in East Asian populations. Patients who were negative were analyzed with PCR-Sanger sequencing of all exons, flanking regions, and 5′UTR and 3′UTR regions. They identified 88 pathogenic variants and 9 novel mutations, with the six most common mutations accounting for 57.46% of all cases. The authors also report some genotype−phenotype correlation, the strongest being younger age of onset and lower levels of plasma ceruloplasmin and serum copper levels in patients with the R778L mutation. Tiered testing starting with array-based technology customized for common regional mutations offers a promising rapid and sensitive method for WD screening, but it cannot be implemented as a large-scale, universal screening method for WD due to the limited detection rate.

MASS SPECTROMETRY-BASED SCREENING

Liquid chromatography−MS/MS (LC−MS/MS) techniques enable antibody-independent, highly precise, and multiplexed quantification of signature proteolytic peptides as stoichiometric surrogates of biomarker proteins. There are multiple advantages to mass spectrometry-based screening methods on dried blood spots. First, the analysis can be performed on newborn dried blood spots, which are stable without any additional processing, modifications, refrigeration, or special transport. Second, the technology employs existing newborn screening infrastructure, which relies on mass spectrometers already present in most newborn state laboratories. Third, the method offers rapid turnaround. Fourth, the assay directly analyzes affected proteins with high specificity instead of screening for downstream accumulated metabolites. Most importantly, surrogate protein biomarker identification by mass spectrometry offers a plethora of opportunities for application to other treatable congenital disorders characterized by absent or low-abundance proteins such as lysosomal storage diseases and primary immunodeficiency syndromes.

More recently, our laboratory developed a novel proteomic screening assay using peptide immunoaffinity enrichment coupled to selected reaction monitoring (SRM) to improve the detection sensitivity of LC−MS/MS. SRM is a targeted mass spectrometry technique with increased sensitivity compared to profiling modes of analysis while maintaining high specificity for the target analyte and the possibility of multiplexing.

Our laboratory initially harnessed this technology to quantify ceruloplasmin by detecting its tryptic-digest signature peptides. After determining signature peptides, establishing internal standards, and conducting validation studies, our results showed patients with WD had lower ceruloplasmin levels by surrogate biomarker peptides compared to carriers and healthy controls. These findings were consistent with those obtained by ELISA assay, with average ceruloplasmin levels of 0−20 mg/dL in affected individuals, compared to >20 mg/dL in WD carriers. The lower limit quantification for ceruloplasmin was determined to be around 0.7 mg/dL, and inter- and intra-assay precision suggests feasibility for clinical use. However, large-scale studies using this technology have not been performed due to the relatively time-consuming sample preparation and long chromatographic run time [40].

As proof-of-concept, we used LC−SRM−MS to simultaneously identify signature peptides as surrogate biomarkers for three life-threatening primary immunodeficiency diseases—severe combined immune deficiency (SCID), Wiskott−Aldrich syndrome (WAS), and X-linked agammaglobulinemia (XLA). The signature peptides were derived from transmembrane protein CD3ε (a general marker for T-cell number) and intracellular proteins Wiskott−Aldrich syndrome protein (WASP) and Bruton tyrosine kinase (BTK) (expressed in B cells and myeloid cells), respectively [75].

Concentrations for each representative protein (that is, WAS, CD3ε, and BTK) were established in 45 control samples and signature isotopically labeled peptides were customized for each protein. In a blinded fashion, 16 de-identified peripheral blood monocyte samples with sufficient actin recovery from molecularly confirmed affected patients (specifically, 5 with WAS, 5 with SCID, and 5 with XLA) were analyzed for these signature peptides. All three signature peptide biomarkers were quantifiably detectable in unaffected leukocyte samples and absent in

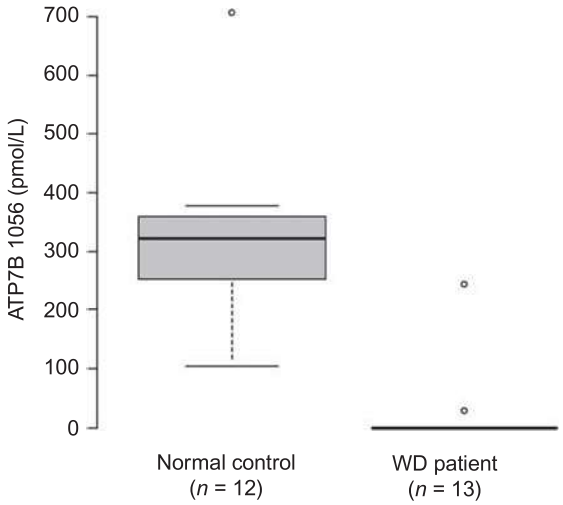

FIGURE 26.1 Distribution of the levels of ATP7B (Wilson ATPase) in dried blood spots from 13 WD patients and 12 normal controls. The bold black line indicates the median, the inner quantiles are represented by boxes, and the whiskers show 95% of the data. *WD*, Wilson disease. *Adapted from Jung S, Whiteaker JR, Zhao L, Yoo HW, Paulovich AG, Hahn SH. Quantification of ATP7B protein in dried blood spots by peptide immuno-SRM as a potential screen for Wilson's disease. J Proteome Res 2017;16:862–71.*

affected leukocyte samples using an actin peptide for normalization. The results were also replicated in T-cell depleted cell lines, demonstrating the ability of LC–SRM–MS to accurately identify patients with primary immunodeficiency disorders. However, the inter- and intra-assay coefficient of variation needed to be lowered for clinical application [75].

To improve the sensitivity and reproducibility of our results for low-abundance proteins and small sample volumes, we enhanced our assay with immunoaffinity enrichment using antipeptide antibodies coupled to SRM (immuno-SRM). Our most recent proof-of-concept study extended this technology to the detection of signature peptides to ATP7B protein in HepG2 cell lines and their isotopically labeled internal standards. This is based on the premise that many missense pathogenic variants associated with WD, including H1069Q and R778L, result in markedly decreased levels of ATP7B protein due to enhanced degradation. Other common WD mutations result in mRNA decay or severely truncated proteins, theoretically resulting in absent or diminished levels of ATP7B in leukocytes. Relative mRNA expression of ATP7B in various tissues, including peripheral leukocytes, has been shown in previous studies (http://biogps.org/#goto = genereport&id = 540) [76]. Therefore, we hypothesized that individuals affected with WD will have lower or absent signature peptides of ATP7B (Wilson ATPase) in their leukocytes and dried blood spots by immuno-SRM compared to carriers or negative controls.

Indeed, our study demonstrated reliable quantification of endogenous ATP7B biomarkers in dried blood spot samples by immuno-SRM. First, we used in silico trypsin digestion to select several signature peptides for ATP7B that are predicted to be well-detected by LC–MS/MS, using criteria previously described [75]. Next, affinity-purified rabbit polyclonal antibodies were generated against four signature peptides. The ATP7B 1056 peptide was selected as a target peptide for quantification in human samples based on good peptide recovery after antibody-enrichment, minimal background signal noise, and the fact that the most common WD mutation in Europeans, H1069Q, occurs within this peptide. Quantification of this ATP7B 1056 peptide was carried out by immuno-SRM on trypsinized dried blood spot samples of patients with WD and unaffected individuals. In 12 out of 13 samples from patients with WD, signature peptides to ATP7B were nearly absent compared to healthy controls, confirming our hypothesis (Fig. 26.1). The genotype and ATP7B 1056 levels in dried blood spots of the 13 WD patients compared to normal controls is depicted in Table 26.2. The one patient with ambiguous results by screening is a compound heterozygote with a variant of uncertain significance in *ATP7B*. Our findings provide compelling evidence that surrogate biomarkers of ATP7B protein may be used as a novel platform to screen for WD in dried blood spots [41]. However, whether the immuno-SRM-based technique is sensitive enough or feasible to screen newborn patients remains to be elucidated. Large-scale studies in newborns with proven carriers and controls are required in the future.

TABLE 26.2 Genotype and ATP7B Peptide 1056 Levels in Dried Blood Spots from 13 Wilson Disease Patients and 12 Normal Controls

Sample	ATP7B 1056 (pmol/L)	Mutation
WD1	29.5	p.R778W and p.T977M
WD2	ND[a]	p.H1069Q and p.R1319*
WD3	ND	p.H1069Q and p.R1319*
WD4	ND	p.G943D and p.T1178A
WD5	ND	p.R778L homozygote
WD6	ND	p.C2304_2305insc and p.L1083F
WD7	NA[b]	p.R778L and p.A874V
WD8	244.8	p.T974M and p.S391L
WD9	ND	p.R778G and p.K175S_fs/p.Q260P_fs
WD10	ND	p.R778G and p.K175S_fs/p.Q260P_fs
WD11	ND	p.R778L and p.E1064A
WD12	ND	p.R778L and p.E1064A
WD13	ND	p.H1069Q and p.Y1331Tfs*61
Controls ($N = 12$)	327.6 ± 147.4	

[a] *ND,* not detected
[b] *NA,* not applicable due to signal to noise ratio (S/N) < 10
Jung S, Whiteaker JR, Zhao L, Yoo HW, Paulovich AG, Hahn SH. Quantification of ATP7B protein in dried blood spots by peptide immuno-SRM as a potential screen for Wilson's disease. J Proteome Res 2017;16:862–71.

SUMMARY

Early detection and treatment of WD is imperative for optimizing long-term clinical outcomes. Diagnosis remains complicated by the nonspecific, broad phenotype of WD and occasionally ambiguous laboratory and molecular studies. WD meets consensus criteria for newborn screening, except that no reliable biomarker or cost-effective screening methods are currently available. Controversy exists on the optimal time to screen, as studies suggest ceruloplasmin assays are most sensitive and specific at around 3 years of age. However, the lack of mandatory outpatient-based pediatric health initiatives presents a challenge for widespread implementation in many countries such as the United States. Screening during the newborn period takes advantage of existing infrastructure for newborn dried blood spots and screening worldwide. Moreover, newborn dried blood spots are sufficiently stable, and they do not require any additional processing, modifications, refrigeration, or special transport. Initial studies utilizing LC–SRM–MS and immuno-SRM technology to quantify signature peptides to target proteins of various metabolic diseases show promising results, but larger studies in newborns compared to carriers and controls are needed. Research into novel biomarkers and honing of DNA- and mass spectrometry-based technology continue to push the frontiers of screening for patients with WD and may result in highly effective strategies for newborn screening.

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Clinical and Translational Perspectives on WILSON DISEASE

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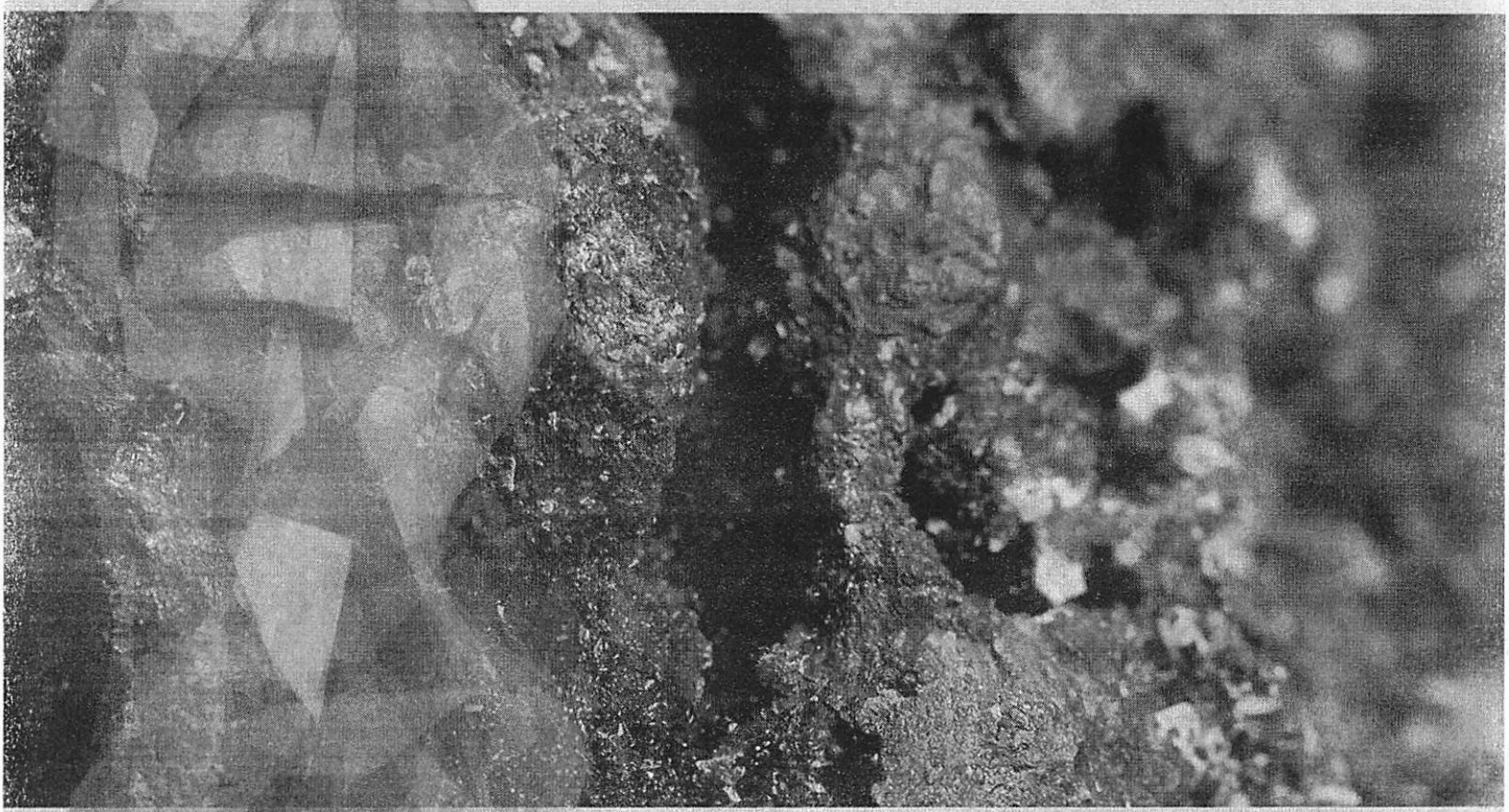

Chapter 17

Wilson Disease in Infancy through Adolescence

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Wilson disease (WD), an inherited disorder mainly of hepatocellular copper disposition, due to dysfunction of the Wilson ATPase, a P_{1B}-ATPase encoded by the gene *ATP7B*, has been regarded as affecting mainly children and young adults. In children, as in older adult patients, WD has protean manifestations. It can involve multiple systems: notably hepatic, neurologic, and psychiatric but also ocular, hematological, renal, skeletal, and cardiac. An important general feature of WD in the pediatric age-bracket is that it often presents with nonspecific features which can be difficult to distinguish from other hepatic or neuropsychiatric disorders. A high level of clinical suspicion, along with effective methodical diagnostic testing, is required. Early diagnosis, preferably before development of clinical symptoms, is associated with normal, healthy life span in patients who are adherent to an effective medical regimen.

WILSON DISEASE IN VERY YOUNG CHILDREN

Classically, in the pediatric age-bracket, WD was most commonly diagnosed in adolescents. It is now well established that WD can present with clinical disease, mainly hepatic, in very young children. Definitions of "very young" children vary slightly among reports. We define "very young" as <5 years-old. Since the first detailed well-documented report of a 3 year-old with cirrhosis [1], numerous further reports have appeared [2–16], as shown in Table 17.1. Some children are diagnosed with WD when they present with an intercurrent health problem such as diarrhea and are found to have elevated serum aminotransferases; others are found through family or general infant screening. An early instance of molecular diagnosis in infancy was an 8-month-old Japanese boy picked up on mass screening [17]. A newborn infant was identified because of established genetic risk of having WD [18]. Among reports predating the identification of *ATP7B*, one appears to be actual WD, not simple copper toxicosis [19]. Many large case series include at least one child <5 years-old. Overall, there appear to be 50–60 such very young children currently reported in the literature. The majority have basal 24-hour urinary copper excretion >0.6 μmol/24 hours (>40 μg/24 hours); however, difficulties in collecting a 24-hour collection in this age-bracket can limit the test's utility. Finding elevated serum aminotransferases in a preschool-aged child is an indication to look for WD.

CLINICAL FEATURES IN CHILDREN AND ADOLESCENTS

The major clinical presentations of WD in children/adolescents are hepatic, neurologic, and psychiatric—and "silent" (no symptoms at all: apparently well). Occurrence of silent WD is extremely important in the pediatric age-bracket. Silent WD includes those patients conventionally described as being asymptomatic or having presymptomatic WD. These terms are often applied interchangeably, although they do not mean the same thing. (Calling a disease state "presymptomatic" makes the assumption that the disease will become clinically manifest.) Silent WD is generally identified

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TABLE 17.1 Features of Wilson Disease in the Very Young Child, Based on Available Published Data

Age, years	Number	Sex	Reason for Ascertainment	Serum Ceruloplasmin[a]	Basal 24-hour Urinary Cu[b]
0–1	7	5 M, 2 F	Incidental (diarrhea)→abnl LFTs: 2	No info: 2	No info: 3
			Family screening: 2; mass screening: 1; projected risk: 1; no info: 1	<100: 3	<0.6: 3
				>140: 2	>0.6: 1
1–2	6	1 M, 5 F	Family screening: 3; no info: 3	No info: 1	No info: 3
				<100: 4	<0.6: 3
				100–140: 1	
2–3	13	6 M, 7 F	Clinical liver disease: 1; "hepatic": 6	No info: 1	No info: 5
			Incidental→abnl LFTs: 3; no info: 3	<100: 4	<0.6: 2
				>140: 6	>0.6: 6
3–4	14	8 M, 6 F	Clinical liver disease: 1; "hepatic": 2	No info: 0	No info: 1
			Incidental→abnl LFTs: 3; pre-kindergarten exam→abnl LFTs: 3	<100: 7	<0.6: 3
			Family screening: 3; no info: 2	>140: 5	>0.6: 10
4–5	12	6 M, 6 F	Incidental→abnl LFTs: 2; family screening: 3; pre-kindergarten exam→abnl LFTs: 1; no info: 6	No info: 1	No info: 1
				<100: 8	<0.6: 1
				>140: 3	>0.6: 10
	52	26 M, 26 F			

abnl, abnormal; *LFTs*, liver function tests; *info*, information; *Cu*, copper.
[a] *< 100 mg/L suggests severe mutation; >140 mg/L is above suggested cutoff for ceruloplasmin in WD determined by immunological methodology.*
[b] *< 0.6 μmol/24 hours (<40 μg/24 hours) is below the informative threshold value for diagnosing WD in children.*

by screening or by chance. Individuals with silent WD have a definite diagnosis of WD, either genetically or biochemically (subnormal serum ceruloplasmin, basal 24-hour urinary copper excretion >0.6 μmol/24 hours; elevated hepatic copper concentration, if sought). While free of clinical symptoms, many have biochemical abnormalities indicating organ damage, typically, elevated serum aminotransferases.

Hepatic-WD tends to be evident at an earlier age than WD presenting with neurologic symptoms (called here "neuro-WD"); however, onset of neuro-WD or psychiatric manifestations of WD in childhood is not uncommon [20]. Psychiatric presentation may be very subtle. Based on data from numerous pediatric clinical series, gender distribution shows slight predominance of males over females (M:F = 5:4), whereas in adults, there may be a female predominance; average age at diagnosis is ~10 years-old.

LIVER DISEASE

The symptoms of hepatic-WD in children are frequently nonspecific, ranging from fatigue, anorexia, nausea, vomiting, weight loss to abdominal pain and jaundice. Jaundice is typical of more severe liver involvement but can be due to intravascular hemolysis. Jaundice may indicate liver decompensation in a chronic case or may be the presenting feature in classic Wilsonian acute liver failure (ALF-WD). If the rate of increase in serum bilirubin is high, the prognosis may be poor, as jaundice is then usually accompanied by other signs of liver failure including coagulopathy, ascites, and/or encephalopathy. Perhaps surprisingly, epistaxis is frequently mentioned in pediatric series as a presenting complaint. Hepatic-WD should be considered as a possible diagnosis in any child/adolescent who has hepatomegaly, elevated serum aminotransferases, or evidence of fatty liver.

WD is an important genetic cause of childhood cirrhosis worldwide. Actually determining the prevalence of cirrhosis in pediatric WD is difficult. A scan of the worldwide literature suggests that approximately one-third of children with clinically evident WD present with cirrhosis. The potential for rapid progression to cirrhosis emphasizes the

TABLE 17.2 Reported Clinical Features of Wilson Disease Resembling Autoimmune Hepatitis

		Age, Sex	Onset	IgG > ULN	Antibodies	Ceruloplasmin (mg/dL)	24-hour Urinary Copper (μg)	KF Ring/ DCT	Cirrhosis/ Interface Hepatitis	Liver Copper (μg/g)	Rx	Outcome
1	Milkiewicz [36]	15 years, F	Chronic	−	ANA 1:100, GPC +	1.1	213	−/−	+/+	385	Pred, D-Pen	Stable
2	Milkiewicz [36]	24 years, F	Chronic	+	SMA 1:60, ANA 1:40	1.8	326	−/−	+/+	965	Pred, LTx	Post-LTx
3	Yener [37]	22 years, F	ALF-WD	NA	ANA 1:400, SMA +	11.2	187.5	+/+	Necrosis	−	Pred	Death
4	Deutsch [38]	32 years, M	ALF-WD	+	ANA 1:1280	20	855	−/−	−/+	NA	Pred/AZA	Stable
5	Santos [39]	17 years, F	ALF-WD	−	↓C3, C4	14	10322	−/+	+/+	961	Pred, LTx	Post-LTx
6	Hunt [40]	29 years, M	Chronic	+	ANA 1:40	NA	NA	NA	+/−	1369	Trientine	Stable
7	Dara [41]	10 years, M	Acute Father: IDDM	−	ANA 1:160, SMA 1:80, LKM 1:20	2.8	1600 μg/dL	−/−	−/+	X20ULN	Pred/AZA and D-Pen	Stable
8	Loudianos [42]	15 years, F	ALF-WD	+	ANA 1:320, SMA 1:160	13.7	NA	+/−	+/+	388	Pred, D-Pen, LTx	Post-LTx

IgG, immunogloblin G; *DCT*, direct Coombs test; *KF*, Kayser–Fleischer ring; Rx treatment; *ANA*, anti-nuclear antibody; *GPC*, gastric parietal cell antibody; *SMA*, smooth muscle antibody; *ALF-WD*, classic Wilsonian acute liver failure; *Pred*, prednisolone or prednisone; *D-Pen*, D-penicillamine; *AZA*, azathioprine; *IDDM*, insulin-dependent diabetes mellitus; *LKM*, liver kidney microsomal antibody; *LTx*, liver transplant; *NA*, not applicable; +, positive/present; −, negative/absent.

importance of investigating persistent, even trivial, elevation of serum aminotransferases. In some countries, WD is an important cause of childhood cirrhosis [21,22]. Cirrhosis is uncommon in pediatric patients identified through screening of first-degree relatives [20].

WD can present as acute liver failure. When it is the initial presentation of WD, it may have highly characteristic clinical features. This classic Wilsonian acute liver failure (ALF-WD) typically consists of moderate-to-severe Coombs-negative acute intravascular hemolysis, coagulopathy unresponsive to vitamin K supplementation, some degree of encephalopathy including lethargy, relatively low serum aminotransferases running $\leq$2000 U/L throughout the clinical illness and very low, occasionally undetectable, serum alkaline phosphatase. Why alkaline phosphatase activity is severely depressed is uncertain but may be related to oxidative damage to the active site [23]. Renal failure attributed to renal tubular damage from copper may develop early in the course of illness. Jaundice may be severe, due to bilirubin load released from erythrocytes plus the renal insufficiency. Serum levels of copper not incorporated in ceruloplasmin are high, and consequently, urinary copper excretion is greatly increased. Classic Wilsonian acute liver failure occurs more often in females than in males and can occur in young children. It is worth noting that it is not unusual to see cirrhosis in the hepatic histology of a patient with ALF-WD either when liver biopsy is performed or in the explanted liver at the time of liver transplantation. Much less commonly, a less distinctive acute liver failure may occur with WD.

As obesity has emerged as a worldwide epidemic, nonalcoholic fatty liver disease (NAFLD) has become the most common cause of liver disease in the pediatric population in the United States. The characteristic histology in NAFLD ranges from bland steatosis to steatohepatitis to cirrhosis [24]. Given that steatosis is also a characteristic histologic feature of WD, there is the possibility of missing a diagnosis of WD. A 4.5-year-old girl with fatty liver and nonspecific hepatitis on liver biopsy was diagnosed with WD based on copper studies. Her timely diagnosis was due to early liver biopsy whose histology led to appropriate WD screening [25]. While diffuse fatty infiltration is a known feature of WD [3,6,26], nodular fatty infiltration has also been reported in childhood WD [27]. Concurrence of metabolic syndrome and WD has been reported in adults [28,29]. While NAFLD is much more common than WD, failure to diagnose WD is serious. It is extremely important to rule out WD before labeling a child or adult with NAFLD as a diagnosis. The best test for distinguishing these two conditions is the basal 24-hour urinary copper excretion, which is much lower in NAFLD than in WD [30]. Checking serum ceruloplasmin is informative only if it reveals a distinctly subnormal serum ceruloplasmin, but the test is not very sensitive.

An important clinical presentation mainly in children, but also in young adults, closely resembles autoimmune hepatitis (AIH). This is a chronic inflammatory condition of the liver characterized by elevated serum aminotransferases, hypergammaglobulinemia, presence of nonspecific autoantibodies (anti-nuclear antibody, ANA; smooth muscle antibody, SMA; liver kidney microsomal antibody, LKM), interface hepatitis on liver histology, and response to immunosuppression. Diagnosis of AIH is facilitated by a scoring system, which has been revised and updated, since there is no single pathognomonic feature [31,32]. Classic AIH may have a chronic or an acute presentation. Importantly, the apparent simple acute hepatitis of WD may prove to be identical to an acute autoimmune hepatitis.

Since the early cases [33–35] of WD clinically resembling autoimmune hepatitis, numerous reports document a clinical presentation of pediatric WD resembling that of AIH (Table 17.2) [36–39,41,42]. In some, WD was misdiagnosed as AIH and treated, albeit inadequately, as AIH. For example, an adolescent girl with an 8-month history of jaundice, ascites, hyperglobulinemia, positive ANA, with interface hepatitis and established cirrhosis on liver biopsy, showed improvement with corticosteroid therapy, but a month later, WD screening results returned positive with low serum ceruloplasmin, increased urinary copper and liver copper of 385 μg/g dry weight. She was started on D-penicillamine and tapered off corticosteroids and became clinically stable [36]. ANA-positivity (type 1 AIH) is more frequent than LKM-positivity (type 2 AIH) in AIH-like WD but the latter pattern can occur.

Acute liver failure poses significant diagnostic problems since acute liver failure due to AIH can be difficult to diagnose. Pediatric patients with WD may present a similarly nondescript clinical picture of acute liver failure, not the classic picture of ALF-WD. A 17-year-old girl with acute liver failure was initially started on a trial of corticosteroids based on clinical and histological features of AIH, despite normal IgG and negative autoantibodies. Clinical deterioration prompted urgent liver transplantation. Histopathology of the explant was consistent with WD; her diagnostic basal 24-hour urinary copper excretion was 10,322 μg/24 hours (162.6 μmol/24 hours), results available only after the transplant [39]. A 15-year-old girl with acute liver failure had ANA 1:320, SMA 1:160, hypergammaglobulinemia, and plasma cell infiltrate on liver biopsy suggesting AIH, but she also had low serum ceruloplasmin, Kayser–Fleischer rings, and elevated liver copper concentration. She was started on both corticosteroids and penicillamine, but she underwent liver transplantation as her condition worsened. She had genetically proven WD [42].

A further problem is that possibly some individuals with WD may get autoimmune hepatitis as such. The disease mechanism in such cases might be similar to the situation where an individual with presymptomatic WD gets acute

liver failure from an intercurrent viral infection [43]. In this uncommon circumstance, the affected person develops AIH, possibly but not necessarily due to copper-mediated damage to the hepatocellular plasma membrane. Ulcerative colitis may occur in children who have WD with prominent autoimmune features [7,44]; however, it has also been reported as an adverse event from trientine [45]. Some children have a genetic makeup predisposing them to autoimmune disease: it might coincide with *ATP7B* mutations. In any case, occurrence of each disease needs to be established, or refuted, fully. Diagnostic problems may occur with primary sclerosing cholangitis [46] (Roberts EA, unpublished observations), or notably with autoimmune sclerosing cholangitis (ASC), because stainable copper may be found on liver biopsy. The pattern of distribution of copper in the liver tends to be different between these disorders and WD. In cholestasis, copper accumulation is typically limited to periportal hepatocytes. Early reports confounding WD and AIH may have been mistaking ASC for AIH, since ASC was not conceptualized until the 1990s.

NEUROLOGIC DISEASE

Neurologic disease is less common in pediatric patients with WD than in the adult age-bracket. It has been reported in children as young as 6 years-old [47] and rarely in preschool-aged children [48]. Walshe reported nine children with neuro-WD aged 12–19 years old [49]. A recent series included patients as young as 7 and 9 years old [50]. Neurologic features can develop in a child with hepatic-WD who is non-adherent to treatment. The neurologic changes of WD are predominantly extrapyramidal. Three distinct neurologic patterns have been described including dystonic, ataxic, and Parkinson-like syndromes, each with characteristic changes in magnetic resonance imaging (MRI) of the brain in each group [51]. The presence of "wing beating" or flapping tremor in association with dysarthria is characteristic of WD. "Risus sardonicus" or the involuntary grimace with an open mouth and contracted upper lip has also been described. These are classic and somewhat late findings. More commonly, in the pediatric population, subtle problems such behavioral changes, deterioration in handwriting, and inability to perform activities requiring good hand-eye coordination are noted. Pseudobulbar palsy can occur with either dystonia or ataxia: features include drooling, dysarthria, and problems with swallowing. Recent pediatric series feature dystonia, dysarthria, cognitive decline, tremor, gait abnormalities, and a Parkinson-like movement disorder, dysphagia, and drooling [50,52]. The majority of these pediatric patients with neuro-WD have Kayser–Fleischer rings. Seizures are atypical but may occur in some adolescents.

Diagnostic problems arise because of the subtlety of clinical presentation. Initial symptoms may be extremely nonspecific. One child presented initially with "learning disabilities" whose cause was discovered only in the course of investigating abdominal pain [53]. A teenager developed tremor, cramps in hands and feet, indecisiveness, and poor school performance [54]. Tremors, gait disorders, clumsiness, and school aversion may be too subtle to be noticed or may be attributed to teenage awkwardness.

PSYCHIATRIC DISORDERS

In Wilson's 1912 monograph, 8 of the 12 patients had psychiatric symptoms. Psychiatric symptoms can occur in both untreated and treated WD patients. Behavioral and psychiatric symptoms are more common in patients with neurologic involvement than in patients with hepatic involvement. Psychiatric symptoms may precede the recognition of hepatic or neurologic WD by a significant period of time, and there may be a delay in diagnosis of WD for several years [55,56]. This is likely because behavioral and psychiatric symptoms due to WD are often misdiagnosed. For example, they may be erroneously attributed to emotional stresses associated with puberty. The mechanism of development of psychiatric manifestations is not clear. Since many patients have the symptoms prior to diagnosis, it cannot be merely due to the psychological impact of the diagnosis. Other factors that have been explored are dopamine dysregulation from basal ganglia involvement, and the role of copper and trace elements in schizophrenia and bipolar illness [57,58]. Changes in circadian rhythm may be relevant [59].

The most common psychiatric features are abnormal behavior (typically increased irritability or disinhibition), personality changes, anxiety, and depression. Psychosis and schizophrenia have also been reported in WD [60,61]. Psychiatric presentation of WD may be more common in the pediatric age-bracket than is generally appreciated. Worsening performance at school or at work can be regarded as a psychiatric feature. A child whose academic performance drops off for no apparent reason requires consideration with respect to WD. Psychiatric aspects of WD in children presenting with hepatic-WD may not be prominent; moreover, development of psychiatric issues after the diagnosis of WD has received relatively little attention. It may have an impact on adherence (see Chapter 19: Wilson Disease: Psychiatric Aspects).

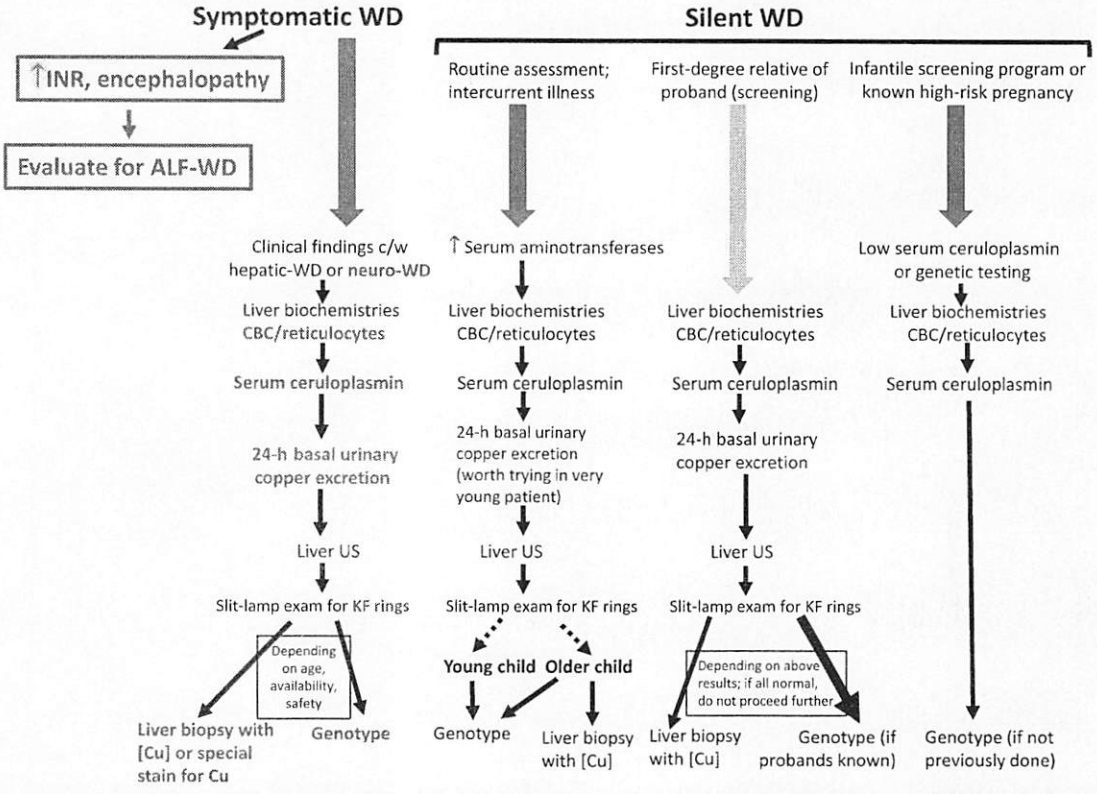

FIGURE 17.1 **A general approach to diagnosis of Wilson disease in the pediatric age-bracket, following comprehensive review of patient's medical history and a full physical examination.** Symptomatic WD comprises a spectrum of hepatic and neurologic disease: red lettering denotes inclusion in the Leipzig score for diagnosing WD. Silent WD includes children found incidentally to have nonspecific liver abnormalities (typically, elevated serum aminotransferases, measured as part of routine screening, or with an intercurrent illness) as well as infants/children identified through general population screening, based on ceruloplasmin or genetic testing. Silent WD also includes first-degree relatives of a WD patient for whom no preliminary data are available. "Liver biochemistries" signifies full liver panel including (at minimum) liver enzymes, total and conjugated bilirubin, albumin, and a coagulation test such as INR. Arrows indicate general progression of investigations, which can be grouped; dotted arrows indicate age dependence; heavy arrow indicates preferred investigation. *c/w*, consistent with; *24-h*, 24-hour; *US*, ultrasonography; *KF*, Kayser–Fleischer; *CBC*, complete blood count; *[Cu]*, parenchymal copper concentration; *ALF-WD*, classic Wilsonian acute liver failure.

OTHER CLINICAL FEATURES

Eye signs include the Kayser–Fleischer ring and sunflower cataracts. The latter are quite rare in children but were reported in a neurologically normal 8-year-old girl with WD [62]. Kayser–Fleischer rings are present in only ~40% of affected children. They are highly informative of the diagnosis when present. Slit-lamp examination by a skilled pediatric ophthalmologist is required for identifying them, especially in very young children. Neither Kayser–Fleischer rings nor sunflower cataracts affect vision. Both resolve with successful treatment.

Transient episodes of intravascular hemolysis are frequently reported in children, mainly as brief self-limited episodes of jaundice. Hemolysis may be an initial presentation of WD. In some cases presenting with hepatic or neurologic features, medical history reveals one or more previously undiagnosed hemolytic episodes. Hemolysis in WD is sometimes apparently precipitated by infection or drugs. A recent retrospective analysis of 321 WD patients revealed presentation with acute hemolysis in 22 (7%). The patients' age ranged from 7 to 20 years with an average onset of 12.6 years. Delayed diagnosis resulted in progression to severe hepatic disease and neurologic deterioration; 4 of these 22 patients may have had classic Wilsonian acute liver failure. [63]. Chronic hemolytic anemia may also occur in pediatric WD [64]. The mechanism is direct damage to the erythrocyte membrane by copper: hence, it is Coombs-negative. As previously noted, severe acute Coombs-negative intravascular hemolysis is prominent in classic Wilsonian acute liver failure.

Renal Fanconi syndrome is typical of WD in children. It may lead to nephrolithiasis. Renal tubular acidosis may also be present. In a Chinese series, five children aged 4–11 years old presented with a renal disorder causing pedal

edema [65]. Evident cardiac involvement is uncommon. Subtle asymptomatic myocardial dysfunction [66] or frank cardiomyopathy may be found [65].

Pediatric patients may have "osseomuscular" WD: arthralgia or less commonly arthritis, proximal muscle weakness, bony deformities, and pathological fractures [67]. These patients are mainly from the Indian subcontinent. Osseomuscular WD seems to be less common elsewhere. Individual musculoskeletal involvement including arthritis, myopathy [68,69], noteworthy muscle cramps [54,70], and rhabdomyolysis [71] has been reported. Osteochondritis dissecans have also been reported [72]. Diagnosis can be delayed if these complaints are misattributed: for example, confused with benign "growing pains" [73].

Other relatively rare organ involvement includes hypoparathyroidism and pancreatitis [74]. Skin signs in children are nonspecific but include extremely dry skin [75]. A clinical picture resembling Henoch–Schönlein purpura with palpable purpura was reported in pediatric WD [65].

DIAGNOSIS

Diagnosis of WD in children can be difficult (Fig. 17.1). The problems of diagnosing WD in a child with acute liver failure may be mitigated by finding Coombs-negative acute intravascular hemolysis, cholestasis in combination with a strikingly low serum alkaline phosphatase, and comparatively low serum aminotransferases from the onset of clinical illness. Very low serum alkaline phosphatase may also be found in WD with severe liver disease not quite qualifying as acute liver failure. For the child with less severe liver disease, such as seronegative acute hepatitis or unexplained elevation of serum aminotransferases or unexplained hemolysis producing jaundice, the most important biochemical criteria are serum ceruloplasmin and basal 24-hour urinary copper excretion, which is an indirect quantification of non-ceruloplasmin-bound copper in the plasma compartment. Measurement of hepatic parenchymal copper concentration provides critical data but involves an invasive procedure. For most patients, genotype determination demonstrating homozygosity for one disease-causing *ATP7B* alteration, or compound heterozygosity for two, is conclusive of the diagnosis. Genotype interpretation can be complex (see Chapter 14: The *ATP7B* Gene).

Among biochemical tests, serum ceruloplasmin remains an important parameter, despite being insufficient as a single test to rule in or exclude WD. Basically, the immunologically based analytical methods for measuring serum ceruloplasmin now in general use for automated measurement of serum ceruloplasmin have changed how that parameter supports the diagnosis of WD. Immunological measurement of serum ceruloplasmin measures apo- as well as holo- (copper-containing) ceruloplasmin. Fully functional holoceruloplasmin is produced as a function of the Wilson ATPase itself; its half-life is ~5.5 days. In contrast, apoceruloplasmin has a relatively short half-life of about 5 hours [76]: serum levels tend to end up lower than normal. Levels may be exceedingly low if the mutation is truncating. Thus, very low serum ceruloplasmin (<50 mg/L, or <5 mg/dL, measured immunologically) may be taken as strong evidence in favor of the diagnosis of WD. One clinical approach is to regard 140 mg/L (14 mg/dL) as the informative cutoff [77]. A more pragmatic approach is to investigate anyone with a subnormal serum ceruloplasmin. As many as one-third of pediatric WD patients may have a normal serum ceruloplasmin. In children with congenital nephrotic syndrome, serum ceruloplasmin may be subnormal due to excessive urinary loss, but anemia is present, as well as features of renal dysfunction [78]. Serum ceruloplasmin level is elevated by inflammation or the oral contraceptive pill since ceruloplasmin is an acute phase reactant.

Measurement of the basal 24-hour urinary copper excretion is valuable. Urine must be collected in copper-free containers; completeness of a full 24-hour collection should be confirmed by measuring urinary creatinine excretion (normal: 10–20 mg in 24 hours). The reference value of 100 μg/24 hours (1.6 μmol/24 hours) is insensitive for diagnosing WD in any age group [79]. The value >0.6 μmol/24 hours (>40 μg/24 hours) is preferable for children. This recommendation is based on a series of 29 pediatric patients of whom 27 had basal 24-hour urinary copper excretion exceeding 0.6 μmol/24 hours (>40 μg/24 hours); in that series, 8 of 29 (28%) had values <1.6 μmol/24 hours (<100 μg/24 hours) [26]. Systematic assessment by ROC analysis showed that 0.6 μmol/24 hours (40 μg/24 hours) is the best cutoff (sensitivity = 79%; specificity = 88%), at least in children [8]. In this latter study, patients who did not reach this level of cupruria were girls under the age of 4 years-old. Clearly, this test is limited by potential difficulties in getting an accurate 24-hour urine collection in very young children. Otherwise, it has physiological rationale and works well. For the purposes of screening, erring toward false positives is acceptable. Chronic cholestatic disorders such as MDR3 deficiency causing chronic cholestasis [80] can produce cupruria >0.6 μmol/24 hours (>40 μg/24 hours): genetic diagnosis is required.

The penicillamine challenge test, in which 500 mg D-penicillamine is given by mouth as a 24-hour urine collection is commenced and then again 12 hours later at the halfway point of the collection, was recommended for diagnosing

WD in children [81]. The diagnosis was established if urinary copper excretion equaled/exceeded 25 μmol/24-hours (>1600 μg/24-hours). Over time, this test has proved less reliable. In various series, 40%–50% or more of patients failed to meet that diagnostic criterion and yet were shown to have WD. It appears that asymptomatic patients may not be detected [82], possibly because the test was developed based on findings in WD children with established disease. The practical problems of testing very young children apply, and the best dose of D-penicillamine for testing very young children is not determined.

Liver biopsy can be highly informative for the diagnosis of WD in children, but it immediately entails the risk and cost of an invasive procedure. The time-honored criterion of hepatic parenchymal copper concentration >250 μg/g dry weight as diagnostic for WD is actually based on an early small study. Thus, the criterion has been subject to reassessment [30,83]. For children with WD, the proposed threshold of 75 μg/g dry weight requires further evaluation: thus, the cutoff of >250 μg/g dry weight remains preferred. Further diagnostic testing is required for children whose hepatic parenchymal copper is between 75 and 250 μg/g dry weight. Some risk of misdiagnosis of WD exists when liver copper content is used as the only test [84]. While normal hepatic parenchymal copper concentration (<50 μg/g dry weight) argues against the diagnosis of WD in the symptomatic patient, erroneously low concentrations can be found if the sample is too small. Presence of cirrhosis may also compromise this test. Sometimes hepatic copper content is not dramatically increased in toddlers with WD. Likewise, in ALF-WD, hepatic parenchymal copper shown by special stains may be misleadingly low.

Identifying an individual as homozygous for a disease-causing *ATP7B* mutation establishes the diagnosis of WD. Many individuals with WD have one each of two different disease-causing mutations. More complex patterns of inheritance may occur (see Chapter 14: The *ATP7B* Gene). In the context of first-degree-relative screening, if the genotype of the proband is known, genetic testing is the most efficient method for identifying siblings with WD. (Genetic screening of a pediatric proband's offspring may be more complicated—at least, logistically.) Generally in children, genetic testing is invaluable for diagnosing cases with atypical clinical findings because it provides the definitive diagnosis.

Scoring systems fall broadly into two categories: for diagnosis and for estimating prognosis (see Chapter 25: Role of Scoring Systems in Wilson Disease). The Leipzig criteria developed for adult patients [85,86] have been modified for use in the pediatric age-bracket [8]. A problem in the pediatric age-bracket is that often genotype is the best, or only, route to a Leipzig score diagnostic of WD. A methodical approach to diagnosis of WD in children may be the best strategy, as opposed to depending on a scoring system (see Fig. 17.1). Among scoring systems for predicting prognosis, the modified King's College Hospital score (new Wilson Index) is in wide use [87] (see Chapter 25: Role of Scoring Systems in Wilson Disease).

WILSON DISEASE-MIMICS

At the present time, mutations in the gene *ATP7B* appear to be the only genetic etiopathogenesis of WD. However, a few disorders have some claim to being a WD-mimic: namely, a different genetic defect produces a highly similar disorder. These include MEDNIK syndrome (mental retardation–enteropathy–deafness–neuropathy–ichthyosis–keratodermia), certain congenital disorders of glycosylation, and a hepatic disorder characterized by manganese retention in hepatocytes (see Chapter 41: Disorders that Mimic Wilson Disease). Indian Childhood Cirrhosis and similar infantile copper toxicoses (see Chapter 44: Indian Childhood Cirrhosis and Other Disorders of Copper Handling) feature hepatic copper accumulation but can be distinguished clinically.

SCREENING NEWBORNS AND PEDIATRIC FIRST-DEGREE RELATIVES

WD fits the criteria of a disease suitable for general newborn and infant screening; however, finding reliable and cost-effective methodology for such screening has proven challenging (see Chapter 26: Population Screening for Wilson Disease). A sensitive immunoaffinity enrichment mass spectrometry test to quantify Wilson ATPase in dried blood spots of newborns may serve effectively to screen for WD [88]

Retrospective reviews have shown that screening brothers and sisters of a child diagnosed with WD and initiating treatment in those found to have WD before they manifest symptomatic disease prevent clinical WD [89,90]. Screening involves physical examination, liver biochemistries, serum ceruloplasmin, and basal 24-hour urinary copper excretion. If positive, liver biopsy with measurement of parenchymal copper should be performed. If the genotype of the proband is known, genetic testing of first-degree relatives is an efficient option, when available and affordable (cost covered by insurance for those who cannot pay). A problem for managing these patients is that they may not fully understand that they have a serious disease: adherence to treatment may be poor [89,91,92] (see Chapter 37: Transition of Care and Adherence in Patients with Wilson Disease).

PROGNOSIS

Early diagnosis, preferably before a child/adolescent has symptomatic hepatic or neurologic disease, is most likely to result in near-normal longevity with generally good health so long as the patient tolerates effective medication, is adherent to the lifelong treatment regimen, and has consistent access to the medication. Families/Patients need to understand that stopping therapy completely may be fatal. An urgent contemporary social justice problem for children with WD is to assure life-long accessibility to effective drug treatment.

MANAGEMENT

Availability of oral chelators revolutionized the management of WD and changed its dire prognosis to a good outlook. Penicillamine is the amino acid cysteine to which two methyl groups have been attached. Extensive clinical experience attests to the efficacy, and relative safety, of D-penicillamine in children [87,93]. The dosage of D-penicillamine in children is 20 mg/kg/day, divided into two to three equal doses. If possible, D-penicillamine should be taken well away from mealtime. Vitamin B6 supplementation (25–50 mg daily) is customary. Adverse side effects, such as pancytopenia and proteinuria, are a major problem: the drug has to be stopped in ~30% of patients. As with adults, clinical deterioration of hepatic or neurologic disease when D-penicillamine is started has been reported in children [94,95]. Starting D-penicillamine at gradually increasing doses over the first 10–14 days of treatment may minimize the risk of the so-called hypersensitivity syndrome. In the first 2–3 months of treatment with D-penicillamine, very close monitoring is required to identify any adverse reactions immediately. Trientine is structurally very different form D-penicillamine. It is safe and effective for WD patients intolerant of D-penicillamine, including children [91,96,97]. Many in the United States advocate it as the preferred first-line oral chelator for any pediatric WD patient, not merely children predicted to be at risk for adverse effects associated with D-penicillamine. Trientine itself has few adverse side effects. Dosage in children is also 20 mg/kg/day, divided into two to three equal doses. In general, trientine should be taken away from meals as food can interfere with its already limited absorption.

Zinc influences copper homeostasis in the body by altering expression of metallothioneins in gut and possibly in liver. It interferes with copper absorption and increases fecal copper excretion. In general, hepatic copper concentrations do not change. Zinc is regarded as effective and well tolerated in children [3,98,99]. Recent data cast some doubt on this enthusiasm [100]. Lack of very long-term follow-up is problematic. Dosage in children is 25 mg elemental zinc three times daily, or twice daily in 5-year olds [101]; teenagers can take the adult dose (50 mg elemental zinc three times daily); optimal dose for toddlers and preschool-aged children remains undetermined. While the actual zinc salt is unimportant, zinc acetate and gluconate are well tolerated, and zinc sulfate seems to be the least well tolerated. It has been associated with gastric or duodenal mucosal erosion/ulceration [102]. Zinc should not be taken with food. Zinc may be more effective for neuro-WD than for hepatic-WD, a relevant consideration since hepatic-WD is more common in children. A child whose hepatic status deteriorates on zinc monotherapy requires oral chelation instead. With pediatric WD, establishing a consistent routine of taking zinc may be difficult because it must be taken three times a day and away from meals/snacks. Zinc salts can cause nausea, vomiting, and epigastric pain—further disincentives to taking it. Changing to a different salt or taking the zinc with a little food may solve this problem.

Tetrathiomolybdate (TTM) is a strong anticopper drug which works by chelation and also by interfering with copper absorption. It is an attractive option for treatment of neuro-WD. A new formulation of tetrathiomolybdate as the bis-choline salt is in development, but it is not yet available for treatment in children.

MEDICAL MANAGEMENT OF CLINICALLY EVIDENT WILSON DISEASE

Most children with WD who have clinical evidence of hepatic or neurologic damage require treatment with an oral chelator. Primary treatment with zinc is often reserved for those who have prominent neurologic involvement. Imaging of the central nervous system, preferably by MRI, should be performed in any child with neurologic WD prior to commencing treatment.

For very severe clinical presentations, mainly decompensated cirrhosis, an induction regimen consists of an oral chelator (D-penicillamine or trientine) and zinc given on a temporally dispersed regimen where some drug is given every 5–6 hours, chelator alternating with zinc. This regimen provides a full dose of chelator plus an adequate dose of zinc in each 24-hour period. It is unwieldy for long-term treatment: after 2–3 months, patients who respond should be transitioned to monotherapy. This regimen appears effective [103–105]. For the patient with decompensated cirrhosis, backup liver transplantation must be arranged because medical treatment failure may occur.

Zinc acetate was approved in the United States as maintenance therapy for WD in 1997. Pediatric patients may be suitable for conversion from oral chelator to zinc, usually after 1–5 years of oral chelation. The patient should be clinically stable with normal liver chemistries and biochemical evidence of good response to chelator therapy. The switch can be immediate (no tapering or ramping up), but close monitoring is required during conversion [83]. Alternatively, pediatric patients have been treated initially with chelation (trientine) for 4–8 months, followed by gradual conversion to zinc including a brief period of combined therapy [96].

MEDICAL MANAGEMENT OF SILENT WILSON DISEASE, INCLUDING THE VERY YOUNG CHILD WITH SILENT WILSON DISEASE

Since many children have silent WD, finding the best treatment for them is important. It is not a "one-size-fits-all" situation. Several biological considerations attain: actual mutation(s), evidence of liver damage, and age. Economic or social considerations may force a treatment decision independent of pathobiology.

Current treatment strategies focus on toxic copper overload. Oral chelators are regarded as aggressive treatment: the aim is to "de-copper" the liver. Zinc is considered gentler, but it is well established that zinc does not radically alter hepatic copper concentration. Extended experience in adults, while limited [106], suggests that zinc may not be effective for hepatic-WD long-term. Overall, pharmacological management of WD requires monitoring and flexibility in drug treatment.

Any child with silent WD who has mutation(s) resulting in failure to produce the Wilson ATPase protein altogether requires aggressive treatment and close monitoring. These individuals likely approximate to the rapidly progressive deterioration of the knockout mouse. Likewise, a child with silent WD and, additionally, silent cirrhosis probably needs oral chelation since evidence for histological improvement in children is based on chelation [107]. Silent WD with abnormal liver biochemistries may respond adequately to zinc or may be recalcitrant. Accruing pediatric experience suggests that close monitoring is mandatory [100]. The patient's age poses various problems. It cannot be assumed that teenagers necessarily have greater hepatic copper concentrations than toddlers. Dosing may be problematic in young children. Adherence may be a game-changer in adolescents.

Specifically for very young children (<5 years-old) with definite WD, best treatment remains undetermined. Although zinc is currently preferred for treatment of very young asymptomatic children, the optimal dose has not been established. The commonsense determination is 25 mg elemental zinc once (toddlers) or twice daily. Early chelation therapy to reduce the hepatic parenchymal copper concentration deserves serious consideration. Any treatment regimen in the very young child must be assessed for risks associated with copper depletion or zinc surfeit in a critical period of growth. For example, if WD is diagnosed in the neonatal period, immediate restriction of dietary copper may interfere with neurologic development. A regular baby formula may thus be appropriate. Delaying treatment to the second year of life may be reasonable if the very young child has no evidence of hepatic or neurologic disorder. Dosing becomes easy once the child weighs 10–12 kg.

In the future, there may be a second therapeutic focus, namely rehabilitation of the defective Wilson ATPase. Decoppering would then take place through the normal function of the rehabilitated protein. This strategy may apply to only selected mutations, for example, H1069Q, but it will enhance the need for genotypic diagnosis.

DIETARY CONSIDERATIONS FOR CHILDREN WITH WILSON DISEASE

WD cannot be treated successfully under any circumstances by dietary manipulation alone. Opinion is divided about the importance of excluding dietary copper in pediatric patients on pharmacological treatment [108]. Some restriction in the first year of treatment may be appropriate for children with abnormal liver tests or clinical illness. The problem is that dietary limitations reinforce the stigma of being unwell. In any case, if a child's dietary copper is to be limited, guidelines must be extremely simple. Patients should avoid liver and organ meats, shellfish, mushrooms, nuts, and chocolate. Vegetarian and vegan patients do require supervision by a dietitian, possibly an issue if an adolescent adopts such dietary practices after achieving clinical stability. If drinking water is suspected to be high in copper, it should be checked: filtered water can be substituted, if necessary. No systematic data are available in children regarding the merits of supplementation with dietary antioxidants such as vitamin E. Other complementary/alternative medicines and dietary supplements [109] may be high in copper.

FOLLOW-UP STRATEGIES FOR THE PEDIATRIC PATIENT ON TREATMENT

Follow-up is similar to that for adults (see Chapter 16: Wilson Disease in Adults: Clinical Presentations, Diagnosis, and Medical Management), with at least twice yearly clinical review. When starting therapy, patients should be advised to report any adverse events; they should have complete blood count, liver and renal biochemistries performed at each review. Pediatric patients maintained on zinc need to be followed specifically for signs of advancing hepatic disease (elevated aminotransferases, thrombocytopenia, ultrasonographic abnormalities). Measuring 24-hour urinary copper excretion is useful in monitoring therapy; measuring 24-hour urinary zinc is useful for monitoring adherence to zinc intake. On oral chelators, urinary copper <200 μg/24 hours (<3 μmol/24 hours) suggests non-adherence or overtreatment. Estimate of the non-ceruloplasmin-bound copper may distinguish between these situations, as it is subnormal (<5 μg/dL; <50 μg/L) in overtreatment and elevated (>15 μg/dL; >150 μg/L) with inadequate treatment or non-adherence [83]. Issues relating to how non-ceruloplasmin-bound copper is estimated limit the utility of this approach (see Chapter 22: Direct Determination of Non-Ceruloplasmin-Bound Copper in Plasma). If the non-ceruloplasmin-bound copper is consistently 0–5 μg/dL (0–50 μg/L), dose of chelator should be gradually reduced and patient monitored. Serial ophthalmologic examinations may be helpful in documenting that the Kayser–Fleischer rings have resolved and vanished, versus development of Kayser–Fleischer rings in those with poor adherence.

CLASSIC WILSONIAN ACUTE LIVER FAILURE AND LIVER TRANSPLANTATION

The child with acute liver failure in WD may benefit from specific interventions as a bridge to transplant, in addition to meticulous supportive care. Specifically, albumin dialysis [110] and related techniques [111,112] may stabilize the pediatric patient. In an adolescent, this type of intervention has on occasion obviated the need for transplant [113]; however, this outcome is exceptional with classic Wilsonian acute liver failure.

Prognostic scoring systems can assist in determining who needs a liver transplant [87]. Liver transplantation can be lifesaving in classic Wilsonian acute liver failure and true treatment failures. For the patient with decompensated cirrhosis, backup liver transplantation should be arranged because medical treatment failure may occur. Living-related donors who are obligate heterozygote carriers are acceptable as donors [114]. Review of the United Network for Organ Sharing database in the United States has shown that those transplanted for WD-related chronic liver disease have higher patient survival rates than those with classic Wilsonian acute liver failure and that children had better survival rates than adults undergoing transplantation for WD [115]. Overall, outcomes for liver transplantation in WD are excellent and comparable to transplantation for other indications (see Chapter 38: Liver Transplantation in Wilson Disease).

CONCLUSION

With improved methods of diagnosis, WD has become an important disease in childhood. The hepatic manifestations are varied, and the neuropsychiatric manifestations may be subtle or nonspecific. Treatment needs to be individualized; comprehensive follow-up is important for all patients. Scoring systems may assist with diagnosis or clinical decisions relating to need for liver transplantation. A recently published position paper complements the views expressed in this chapter [116].

SUMMARY

WD can affect children of all ages. In the current era of increased availability of genetic testing, WD is being diagnosed earlier in life. Indeed, there are over 50 cases of WD in children under 5 years of age documented in the literature. The need to be aware of the possibility of WD as the cause of liver dysfunction regardless of age is important. Clinical presentation may be chronic liver disease, acute liver failure with distinctive features (ALF-WD) or it may be "silent" liver disease. A broad spectrum of neurologic or psychiatric conditions can occur in childhood or adolescence. While hepatic presentation is more common than neurologic in the pediatric population, manifestations of WD are characteristically multisystemic. WD can resemble autoimmune liver disease clinically. Particularly when there is inadequate response to immunosuppression therapy in autoimmune hepatitis, every effort must be made to ensure that a diagnosis of WD is not being missed. A combination of biochemical tests including serum ceruloplasmin, 24-hour urinary copper excretion, and liver copper concentration as well as genetic tests is used to establish diagnosis. Scoring systems to assist diagnosis and to assess prognosis are available. Current management includes chelators like D-penicillamine or trientine; zinc has multiple effects exclusive of chelation. Economic and social factors often dictate drug therapy. Foods containing high

amounts of copper like shellfish and chocolates may be restricted, at least in the first year of treatment. After a diagnosis of WD, first-degree relatives of the child should be screened. Therapy is lifelong. Adherence to medical recommendations is necessary for good outcomes. Parents should be made aware that stopping therapy suddenly may prove fatal for the child. Liver transplantation is required in classic Wilsonian acute liver failure and when medical therapy fails. Newer therapies are being investigated. Newborn screening may become routine in the future.

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Chapter 3

The genetics of Wilson disease

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Abstract

Wilson disease (WD) is an autosomal-recessive disorder of hepatocellular copper deposition caused by pathogenic variants in the copper-transporting gene, *ATP7B*. Early detection and treatment are critical to prevent lifelong neuropsychiatric, hepatic, and systemic disabilities. Due to the marked heterogeneity in age of onset and clinical presentation, the diagnosis of Wilson disease remains challenging to physicians today. Direct sequencing of the *ATP7B* gene is the most sensitive and widely used confirmatory testing method, and concurrent biochemical testing improves diagnostic accuracy. More than 600 pathogenic variants in *ATP7B* have been identified, with single-nucleotide missense and nonsense mutations being the most common, followed by insertions/deletions, and, rarely, splice site mutations. The prevalence of Wilson disease varies by geographic region, with higher frequency of certain mutations occurring in specific ethnic groups. Wilson disease has poor genotype–phenotype correlation, although a few possible modifiers have been proposed. Improving molecular genetic studies continue to advance our understanding of the pathogenesis, diagnosis, and screening for Wilson disease.

INTRODUCTION

In this chapter, we will discuss the inheritance, gene frequency, variants, genotype–phenotype correlation, and modifiers of the *ATP7B* gene, and the clinical molecular diagnosis and population screening for Wilson disease.

INHERITANCE

Wilson disease is a monogenic autosomal-recessive condition and carriers do not manifest any symptoms. Autosomal-recessive conditions are not usually present in consecutive generations, but may occur in populations with particularly high carrier frequency of Wilson disease (Wu et al., 2015). Our group and others have reported the presence of Wilson disease in two or more successive generations within the same family, reflecting a "pseudo-dominant" inheritance (Dziezyc et al., 2011, 2014; Bennett et al., 2013; H. Park et al., 2015). Therefore, the diagnosis of Wilson disease should not be excluded simply due to a misleading family history consistent with an autosomal-dominant inheritance pattern. Furthermore, recent studies have also identified Wilson disease due to atypical forms of inheritance, such as the presence of three concurrent mutations in a single patient or segmental uniparental disomy (Coffey et al., 2013). Uniparental disomy occurs when both homologs of a chromosome originate from a single parent. These findings have implications for clinical practice and genetic counseling, as clinicians may need to consider genotyping asymptomatic parents or obtaining full sequencing of *ATP7B* to confirm that pathogenic variants occur in *trans*.

ATP7B GENE AND ATPASE

Wilson disease is caused by homozygous or compound heterozygous mutations in the *ATP7B* gene (OMIM# 606882), which encodes a transmembrane copper-transporting P-type ATPase of the same name. Currently,

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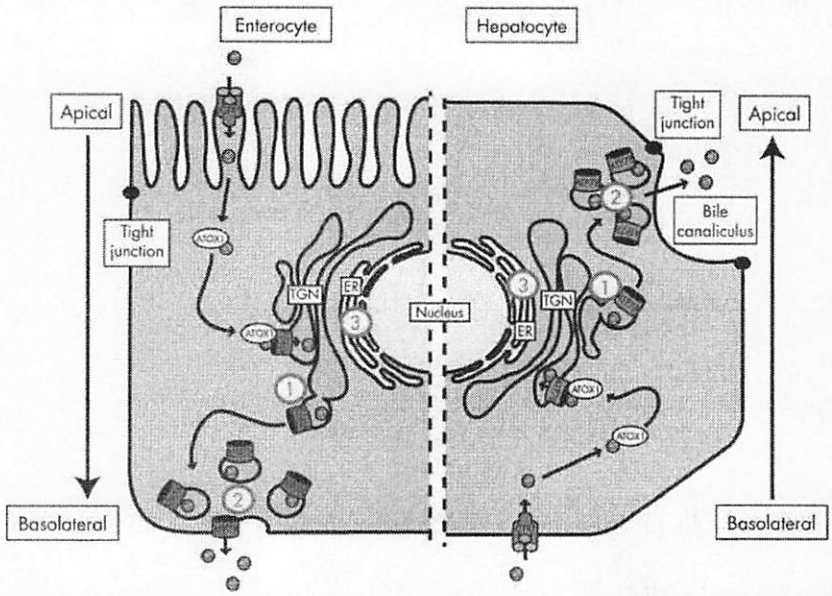

Fig. 3.1. Schematic representation of copper-induced relocalization of ATP7A and ATP7B. The left side of the diagram represents an enterocyte and the right side represents a hepatocyte. On both sides, copper enters the cell through copper transporter 1 (CTR1) and is escorted by copper chaperone antioxidant protein 1 (ATOX1) to ATP7A or ATP7B in the trans-Golgi network (TGN). When copper levels rise above a certain threshold, ATP7A and ATP7B excrete copper into the plasma on the basolateral side of the enterocyte and into the bile on the apical side of the hepatocyte. Defects in localization of ATP7B may lead to copper accumulation at the (1) TGN due to unresponsiveness, (2) cell periphery, and (3) endoplasmic reticulum (ER) due to misfolding. (Reproduced from de Bie et al., 2007.)

ATP7B is the only identified gene known to cause Wil son disease (Bull et al., 1993; Petrukhin et al., 1993; Tanzi et al., 1993). Mutations in the *ATP7B* gene have been reported in almost all exons. Previous studies have reported individuals with both biochemical and clinical diagnosis of Wilson disease in the absence of two *ATP7B* mutations, raising the possibility of a second causative gene (Lovicu et al., 2006; Kenney and Cox, 2007; S. Park et al., 2007; Mak and Lam, 2008; Nicastro et al., 2010; Coffey et al., 2013). Nonetheless, *ATP7B* remains the only known gene responsible for Wilson disease.

Human dietary intake of copper is about 1.5–2.5 mg/day, which is absorbed in the stomach and duodenum, bound to circulating albumin, and transported to the liver for regulation and excretion (Culotta and Scott, 2016). The uptake of copper occurs on the basolateral side of hepatocytes via copper transporter 1 (CTR1), as illustrated in Figure 3.1. A specific copper chaperone, antioxidant protein 1 (ATOX1), delivers copper to the Wilson disease protein, ATP7B, by copper-dependent protein–protein interactions (Walker et al., 2004). Within hepatocytes, ATP7B performs two important functions in either the trans-Golgi network (TGN) or in cytoplasmic vesicles. In the TGN, ATP7B activates ceruloplasmin by packaging six copper molecules into apoceruloplasmin, which is then secreted into the plasma. In the cytoplasm, ATP7B sequesters excess copper into vesicles and excretes it via exocytosis across the apical canalicular membrane into bile (Bull et al., 1993; Tanzi et al., 1993; Yamaguchi et al., 1999; Cater et al., 2007). Due to the binary role of the ATP7B transporter in both the synthesis and excretion of copper, defects in its function lead to copper accumulation and the progressive features of Wilson disease (Fig. 3.1).

MOLECULAR STRUCTURE OF *ATP7B*

ATP7B is located on 13q14.3 and contains 20 introns and 21 exons, for a total genomic length of 80 kb (Bull et al., 1993; Petrukhin et al., 1993; Tanzi et al., 1993). The gene is synthesized in the endoplasmic reticulum, then relocated to the TGN within hepatocytes. *ATP7B* is most highly expressed in the liver, but is also found in the kidney, placenta, mammary glands, brain, and lung.

ATP7B (P-TYPE ATPASE) PROTEIN STRUCTURE AND FUNCTION

ATP7B belongs to class 1B (PIB) of the highly conserved P-type ATPase superfamily, which is responsible for the transport of copper and other heavy metals across cellular membranes (Gourdon et al., 2011). The protein contains 1465 amino acids, a phosphatase domain (A-domain), phosphorylation domain (P-domain, amino acid residues 971–1035), nucleotide-binding domain (N-domain, amino acid residues 1240–1291), and

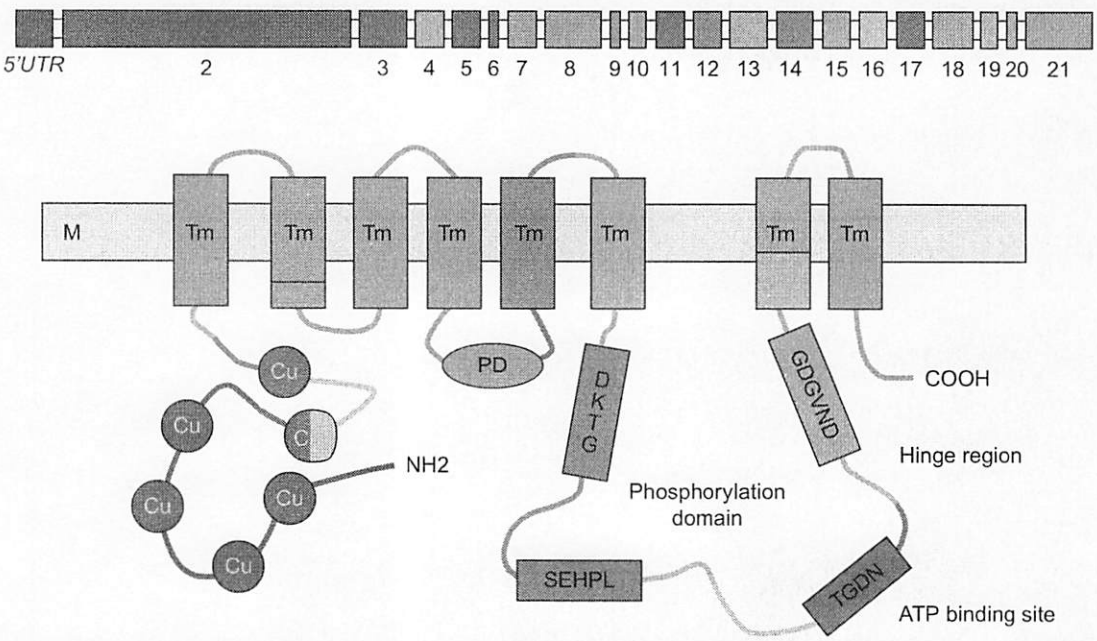

Fig. 3.2. Schematic representation of *ATP7B* gene and corresponding human ATP7B protein. Top diagram shows 5'UTR promoter region and exons separated by introns. Bottom diagram shows the domain organization of human copper ATPase. Conserved amino acid motifs are present at the core structure of each functional domain, i.e., TGDN and GDGVND at the A-domain, DKTG at the P-domain, and SEHPL in the N-domain. M, phospholipidic bilayer of the membrane; Cu, the metal-binding domains of the trasmembrane cation channel; Tm, transmembrane domains; PD, phosphatase domain. (Reproduced from Fanni et al., 2005.)

M-domain, which comprises eight transmembrane ion channels (Fig. 3.2) (Cater et al., 2004, 2007; Lenartowicz and Krzeptowski, 2010).

Unique amino acid motifs are present at the core structure of each domain, such as TGEA at the A-domain, DKTGT at the P-domain, and SEHPL in the N-domain. Specifically, the N-terminal metal-binding domain (MBD) is composed of six copper-binding sites, each with the conserved sequence motif GMXCXXC (Fatemi and Sarkar, 2002; Sazinsky et al., 2006). These MBDs play a central role in accepting copper from copper chaperone ATOX1 through protein–protein interactions. Previous studies have demonstrated unequal impact of MBDs on ATP7B activity, with MBD 5 and 6 having stronger effects on the catalytic activation of ATP7B than MBDs 1–4 (Lutsenko et al.,1997).

The active transport of copper across membranes is a complex process that begins with ATP7B binding copper at the N-terminal domain and transporting it across cellular membranes, using ATP as an energy source (Fig. 3.2). Next, free copper binds intracellularly to GG motifs in the MBDs, followed by transport on to the Cys-Pro-Cys (CPC) sequence motifs in MBD 6. Finally, dephosphorylation of acyl-phosphate at the A-domain discharges copper across the cellular membrane. Mutations causing copper accumulation may occur at any of these steps (Huster et al., 2006; Schushan et al., 2012).

Although the mechanism by which the histadine-containing SEHPL motif affects copper transport remains to be elucidated, it is clear that histidine-to-glutamate substitution at amino acid 1069 (p.H1069Q) in this motif is the most common cause of Wilson disease in northern Europeans. In the hepatocytes of patients homozygous for p.H1069Q, ATP7B was found in the endoplasmic reticulum instead of its usual TGN location, suggesting abnormal protein trafficking (Huster et al., 2003). Insect models with the p.H1069Q mutation in SF9 cells showed decreased ATP-mediated catalytic phosphorylation but no major protein misfolding, suggesting a role for p.H1069Q in the orientation of the ATP7B catalytic site for ATP binding prior to hydrolysis (Tsivkovskii et al., 2003).

VARIANTS IN THE *ATP7B* GENE

More than 600 pathogenic variants in *ATP7B* have been identified, with single-nucleotide missense and nonsense mutations being the most common, followed by insertions/deletions and splice site mutations (Human Gene Mutation Database, accessed 29 April 2016; Stenson et al., 2014). Other rare genetic mechanisms that have been reported in the literature include whole-exon deletions, promoter region mutations, three concurrent pathogenic variants, and monogenic disomy (Coffey et al., 2013; Bandmann et al., 2015). Mutation "hotspots" in

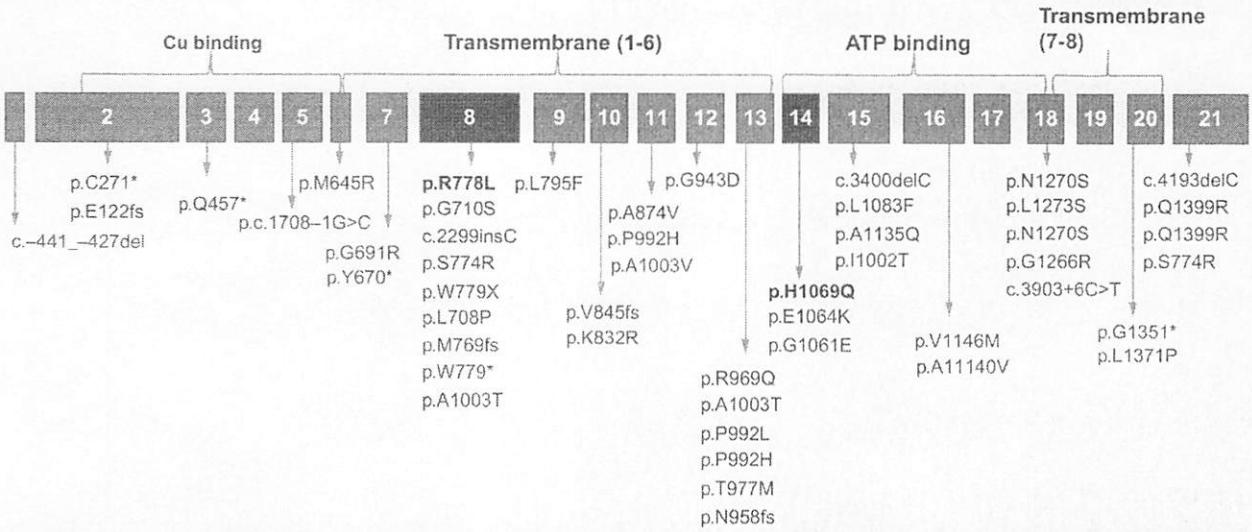

Fig. 3.3. Schematic of the *ATP7B* gene with common mutation sites, including p.H1069Q (rs76151636), p.R778L (rs28942074), p.E1064K (rs376910645), c.3400delC, and p.Ala1135fs (rs137853281). Please refer to Table 3.1 for more details.

ATP7B have also been reported to vary by geographic region (see regional gene frequency section, below). The majority of pathogenic mutations are located in the M- and N-domains in presymptomatic patients or in those with hepatic symptoms (S. Park et al., 2007). The common mutations in *ATP7B* seen in various populations are listed in Figure 3.3.

The p.H1069Q mutation is one of the most common mutations, with a population allelic frequency of 10–40% (30–70% among Caucasians). Most patients are compound heterozygotes, carrying different mutations on each copy of the chromosome (Usta et al., 2014). The p.H1069Q mutation occurs when histidine of the conserved SEHPL motif in the N-domain of *ATP7B* is replaced by glutamic acid, resulting in N-domain protein misfolding, abnormal phosphorylation in the P-domain, and decreased ATP binding affinity (Rodriguez-Granillo et al., 2008). This mutation also leads to decreased heat stability and abnormal localization of the protein to the TGN (Ralle et al., 2010).

Other common mutations in *ATP7B* include p.E1064A, p.R778L, p.G943S, and p.M769V. Mutations in p.E1064A, also found in the SEHPL motif, completely disable ATP binding affinity but do not result in protein misfolding, transport abnormalities, or thermal instability. The p.R778L mutation affects transmembrane transport of copper (Dmitriev et al., 2011). The p.G943S and p.M769V mutations result in defective copper metabolism but preserved ceruloplasmin levels (Okada et al., 2010).

A substantial proportion of Wilson disease-associated missense mutations, including p.H1069Q and p.R778L, result in markedly decreased level of the protein caused by enhanced degradation (Payne et al., 1998; de Bie et al., 2007; van den Berghe et al., 2009). Other prevalent mutations, such as protein-truncating nonsense mutations (~13% of known point mutations) (Merle et al., 2010) and frameshift mutations (Vrabelova et al., 2005), are predicted to cause decay of mRNA (Mendell et al., 2004; Chang et al., 2007) or a severely truncated protein, resulting in absent or diminished levels of protein. It is therefore expected that most patients with Wilson disease have absent or significantly reduced levels of ATP7B.

REGIONAL GENE FREQUENCY

The prevalence of Wilson disease varies by geographic region, with higher prevalence of specific mutations reported in certain populations (Ferenci, 2006) (see Chapter 2 for more details). A list of the common regional variants of ATP7B mutations and geographic clustering of mutations are shown in Table 3.1 and Figure 3.4, respectively.

GENOTYPE–PHENOTYPE CORRELATION

Direct genotype–phenotype relationships in Wilson disease have been difficult to establish, despite several studies examining correlation (Panagiotakaki et al., 2004; Vrabelova et al., 2005; Nicastro et al., 2010; Cocoş et al., 2014; Usta et al., 2014). The numerous low-frequency and compound heterozygous nature of Wilson disease obfuscate the process of characterizing its numerous genetic variants and their clinical consequences. Descriptions of phenotypes are limited to age of onset and presenting symptoms, both of which may be affected by inaccurate diagnostic criteria, delayed diagnosis, and practitioner selection bias. Therefore,

Table 3.1

Regional distribution of common Wilson disease mutations by geographic location

Region	AF (%)	Prevalent mutations					
		Protein	Nucleotide	RS	Exon	Type	Domain
Europe							
Austria (**Ferenci**, 2006)	34.1	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
	6.4	p.Gly710Ser	c.2128G>A		8	Missense	TM2
	3.6	p.Met769fs	c.2298_2299insC	rs137853287	8	Premature stop	TM4
Benelux (**Ferenci**, 2006)	53	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
Bulgaria (**Todorov** et al., 2005)	58.8	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
Canary Islands (**García Villarreal** et al., 2000)	64	p.Leu708Pro	c.2123 T>C		8	Missense	TM2
Czech Republic (**Vrabelova** et al., 2005)	57	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
Denmark (**Møller** et al., 2011)	18	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
	16	p.Trp779*	c.2336G>A	rs137853283	8	Nonsense	TM4
France (**Bost** et al., 2012)	15	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
Germany (**Ferenci**, 2006)	47.9	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
Germany (East, former) (**Caca** et al., 2000)	63	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
Greece (**Panagiotakaki** et al., 2004; **Dedoussis** et al., 2005; **Gomes** and **Dedoussis**, 2016)	35	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
	12	p.Arg969Gln	c.2906G>A	rs774028495	13	Missense	TM6
Hungary (**Firneisz** et al., 2002; **Folhoffer** et al., 2007)	42.9	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
Iceland (**Thomas** et al., 1995a; **Hofer** et al., 2012)	100	p.Tyr670*	c.2007_2013del		7	Nonsense	TM1
Italy (**Loudianos** et al., 1999)	17.5	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
	9	p.Val845fs	c.2530delA	rs755709270	10	Premature stop	Td
	6	p.Met769fs	c.2298_2299insC	rs137853287	8	Premature stop	TM4
Netherlands (**Stapelbroek** et al., 2004)	33	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop

Continued

Table 3.1

Continued

Region	AF (%)	Prevalent mutations		RS	Exon	Type	Domain
		Protein	Nucleotide				
Poland (**Gromadzka** et al., 2005)	72	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
	7.3	p.Ala1135fs	c.3400delC	rs137853281	15	Premature stop	ATP loop
	3.7	p.Gln1351*	c.4051C>T		20	Nonsense	
Romania (**Iacob** et al., 2012)	38.1	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
Russia (**Ivanova-Smolenskaya** et al., 1997)	49	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
Sardinia (**Figus** et al., 1995)	60.5		c.-441_-427del		5prime	Unknown	Promoter
	8.5	p.Met822fs	c.2463delC		10	Deletion	TM4/Td
	7.9	p.Val1146Met	c.3436G>A		16	Missense	ATP loop
Serbia (**Tomić** et al., 2013)	38.4	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
	11.6	p.Met769fs	c.2304dupC		8	Missense	TM4
	9.3	p.Ala1003Thr	c.3007G>A	rs1801247	13	Missense	TM6/Ph
Spain (**Margarit** et al., 2005)	27	p.Met645Arg	c.1934 T>G	rs121907998	6	Missense	Cu6/TM1
Sweden (**Shah** et al., 1997)	38	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
Turkey (**Ferenci**, 2006; **Simsek Papur** et al., 2013)	17.4	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
	5.3	p.Gly710Ser	c.2128G>A	rs772595172	8	Missense	TM2
	4.53	p.Gln457*	c.1369C>T		3	Nonsense	Cu4/Cu5
UK (**Coffey** et al., 2013)	19	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
	8	p.Met769Val	c.2305A>G		8	Missense	TM4
Yugoslavia (former) (**Loudianos** et al., 1999)	48.9	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
	11.4	p.Met769fs	c.2298_2299insC	rs137853287	8	Premature stop	TM4
Asia							
China (**Gu** et al., 2003; **Z.-Y. Wu et al.**, 2003; **Wang** et al., 2011; **Wei** et al., 2014)	31	p.Arg778Leu	c.2332C>T	rs28942074	8	Missense	TM4
	10	p.Pro992Leu	c.2975C>T	rs201038679	13	Missense	TM6/Ph
	9.6	p.Ile1148Thr	c.3443 T>C	rs60431989	16	Missense	ATP loop
	3.3	p.Thr935Met	c.2804C>T		12	Missense	TM5
	19	p.Arg778Leu	c.2332C>T	rs28942074	8	Missense	TM4

North India (**S. Kumar** et al., 2006; **Gupta** et al., 2007)							
	12	p.Ile1102Thr	c.3305 T>C	rs560952220	15	Missense	ATP loop
	9	p.Pro992His	c.2975C>A		13	Missense	TM6/Ph
South India	11	p.Ala1003Val	c.3008C>T		13	Missense	TM6/Ph
(**Santhosh** et al., 2006; **S. S. Kumar** et al., 2012)							
	11	p.Cys271*	c.813C>A	rs572147914	2	Nonsense	Cu3
	9	p.Pro768Leu	c.2303C>T		8	Missense	TM4
	9	p.Arg969Gln	c.2906G>A	rs121907996	13	Missense	TM6
East India	16	p.Cys271*	c.813C>A	rs572147914	2	Nonsense	Cu3
(**Gupta** et al., 2005)							
	11	p.Gly1061Glu	c.3182G>A		14	Missense	ATP loop
	8.5		c.1708-1G>C	rs137853280	5	Splice	Cu6
West India	20	p.Cys271*	c.813C>A	rs572147914	2	Nonsense	Cu3
(**Aggarwal** and **Bhatt**, 2013; **Aggarwal** et al., 2013)							
	11	p.Glu122fs	c.365_366delins TTCGAAGC		2	Ins/Del	Cu1
	6	p.Thr977Met	c.2930C>T	rs72552255	13	Missense	TM6
	6	p.Leu795Phe	c.2383C>T		9	Missense	TM4/Td
Japan	17.95	p.Asn958fs	c.2871delC		13	Premature stop	TM5/TM6
(**Okada** et al., 2000; **Tatsumi** et al., 2010)							
	16.7	p.Arg778Leu	c.2332C>T	rs28942074	8	Missense	TM4
	10.5		c.1708-5 T>G		5	Splice	Cu6
Korea	37.9	p.Arg778Leu	c.2332C>T	rs28942074	8	Missense	TM4
(**E. K. Kim** et al., 1998; **Yoo**, 2002; **G.-H. Kim** et al., 2008; **Song** et al., 2012)							
	12.1	p.Asn1270Ser	c.3809A>G	rs121907990	18	Missense	ATP hinge
	9.4	p.Ala874Val	c.2621C>T	rs376355660	11	Missense	TM5
	8	p.Leu1083Phe	c.3247C>T		15	Missense	ATP loop
Lebanon	44.7	p. Ala1003Thr	c.2299insC	rs137853287	8	Missense	TM4
(**Usta** et al., 2014)							
Saudi Arabia	32	p.Gln1399Arg	c.4196A>G		21	Missense	After TM8
(**Al Jumah** et al., 2004; **Majumdar** et al., 2004)							
	16	p.Ser774Arg	c.2230 T>C	rs535217574	21	Missense	TM3
Taiwan	29.6	p.Arg778Leu	c.2332C>T	rs28942074	8	Missense	TM4
(**Lee** et al., 2000; **Wan** et al., 2006)							
	8.9	p.Pro992Leu	c.2975C>T	rs201038679	13	Missense	TM6
	4.8	p.Gly943Asp	c.2828G>A		12	Missense	TM5

Continued

Table 3.1

Continued

Region	AF (%)	Prevalent mutations					
		Protein	Nucleotide	RS	Exon	Type	Domain
Thailand (**Panichareon** et al., 2011)	10.52	p.Arg778Leu	c.2332C>T	rs28942074	8	Missense	TM4
	7.89	p.Leu1371Pro	c.4112 T>C		20	Missense	TM8
Iran (**Zali** et al., 2011)	19	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
Africa							
Egypt (**Abdelghaffar** et al., 2008; **Abdel Ghaffar** et al., 2011)	42.2	IVS18+6 T>C	c.3903+6C>T	rs2282057	18	Splice	
	40.6	p.Ala11140Val	c.3419C>T		16	Missense	ATP loop
	26.5	p.Lys832Arg	c.2495A>G	rs1061472	10	Missense	TM4/Td
Americas							
USA (**Kuppala** et al., 2009)	40.3	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
	1.9	p.Asn1270Ser	c.3809A>G	rs121907990	18	Missense	ATP hinge
	1.9	p.Gly1266Arg	c.3796G>A	rs121907992	18	Missense	ATP hinge
Brazil (**Deguti** et al., 2004; **Machado** et al., 2008; **Bem** et al., 2013)	37.1	p.His1069Gln	c.3207C>A	rs76151636	14	Missense	ATP loop
	31.25	p.Ala1135fs	c.3400delC	rs137853281	15	Premature stop	ATP loop
	11.4	p.Ala1135GlnfsX13	c.3402delC	rs137853281	15	Premature stop	ATP loop
		p.Leu708Pro	c.2123 T>C		8	Missense	TM2
Costa Rica (**Shah** et al., 1997)	61	p.Asn1270Ser	c.3809A>G	rs121907990	18	Missense	ATP hinge
Venezuela (**Paradisi** et al., 2015)	26.9	p.Ala1135GlnfsX13	c.3402delC	rs137853281	15	Premature stop	ATP loop
	9.6	p.Gly691Arg	c.2071G>A		7	Missense	TM2

AF, allelic frequency; RS, Reference single nucleotide polymorphism (SNP) cluster identification number.

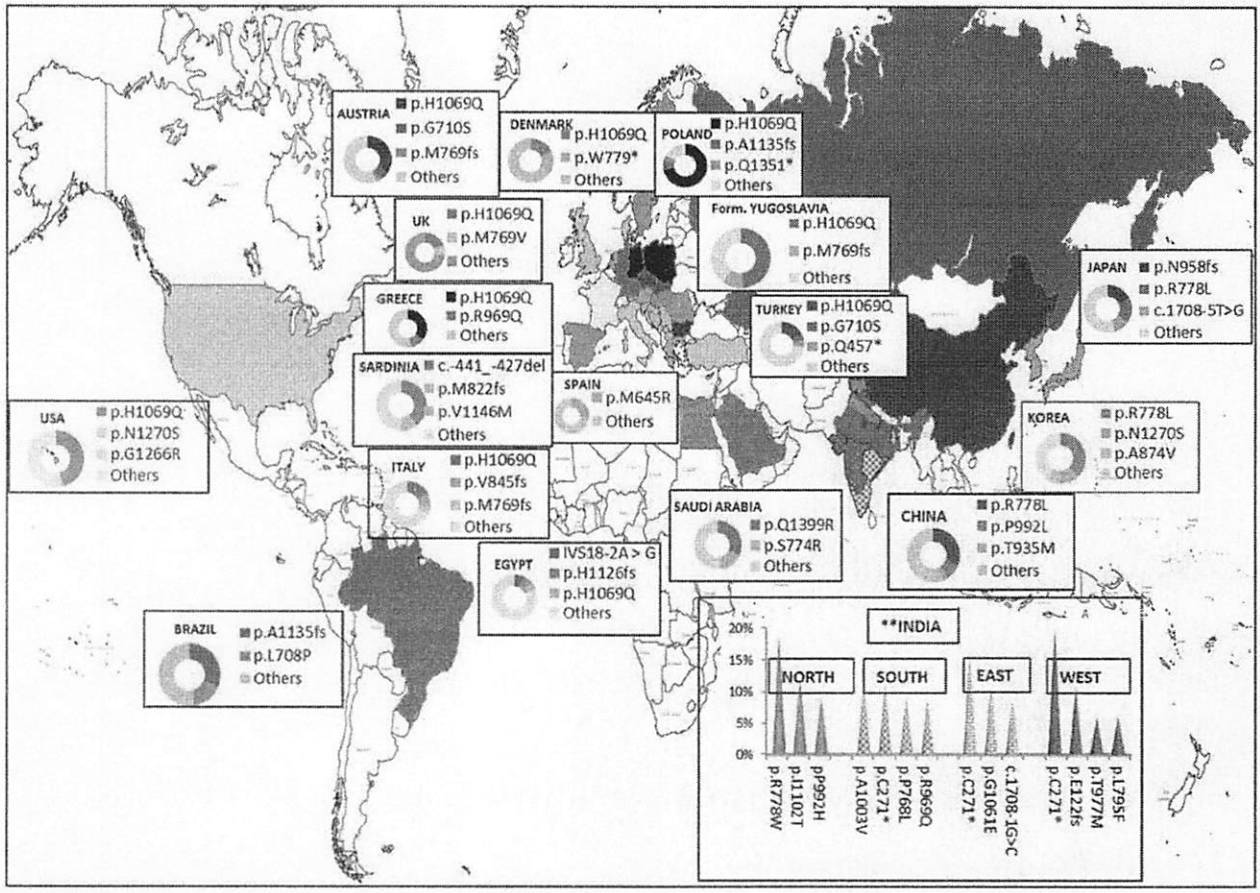

Fig. 3.4. Prevalence of *ATP7B* mutation by geographic region; the darker the gradient, the higher the allelic frequency. (Reproduced from Gomes and Dedoussis, 2016, with permission from Taylor and Francis.)

the marked variability in phenotype of Wilson disease is likely attributable to an amalgamation of genetic, metabolic, and environmental factors (Leggio et al., 2006).

The most consistent genotype–phenotype correlation in Wilson disease is that the most severe, early-onset disease with predominantly hepatic presentation is associated with mutations causing absent ATPase activity. Convincing studies have demonstrated fulminant hepatic disease in mouse models such as the toxic milk (tx) mouse and the Jackson tx mouse (tx^J), which harbor point mutations causing loss of ATP7B function, but not affecting ATP7B synthesis (Theophilos et al., 1996; Coronado et al., 2001; La Fontaine et al., 2001; Huster et al., 2006).

Genetic polymorphisms in *ATP7B*, other genes, and epigenetic factors have been shown to impact disease phenotype by affecting ATP7B protein structure and function. Of the over 600 mutations associated with Wilson disease, the majority are missense mutations that completely inactivate the copper-transporting function of ATP7B (Lutsenko, 2014). In general, individuals with protein-truncating mutations have earlier onset of disease due to decreased protein stability and quantity (Merle et al., 2010). However, other studies have demonstrated partial preservation of copper-transporting function, perhaps explaining the milder phenotypes associated with certain mutations (Rodriguez-Granillo et al., 2008; Dmitriev et al., 2011; Huster et al., 2012). Individuals with the R778L mutation have been shown to have an earlier onset of disease and predominantly hepatic presentation (Z. Y. Wu et al., 2003). In contrast, individuals with the H1069Q mutation have a mean onset of symptoms between 20–22 years old and predominantly neurologic phenotype (Stapelbroek et al., 2004; Kalita et al., 2010). There is also some evidence that Kayser–Fleischer rings are more common in H1069Q homozygous patients in Hungary at time of diagnosis than in compound heterozygous individuals (Folhoffer et al., 2007).

Moreover, pathogenic variants may affect ATP7B targeting from the TGN to cytosolic vesicles. For instance, the p.Met875Val mutation results in a less stable protein and causes reversible ATP7B localization defects. Under a low-copper environment, the p.Gly875Arg variant is sequestered in the endoplasmic reticulum. However, addition of exogenous copper to the cellular growth medium stabilizes the protein, allowing it to complete

its intended journey to the TGN and overcoming its disease-causing phenotype. Theoretically, patients with this specific variant may be more sensitive to dietary copper deficiency (Gupta et al., 2011).

The timing and location of copper buildup can also preferentially alter the hepatic transcriptome, based on homozygous *ATP7B*$^{-/-}$ mouse models. Proteomic analyses of mRNA profiles at each of these disease stages reflect unique patterns (Huster et al., 2006; Ralle et al., 2010). In the initial stage, mRNA for proteins responsible for cell cycle regulation, splicing, and cholesterol synthesis is present (Burkhead et al., 2011). This leads to early accumulation of copper bound to metallothioneins in the cytosol and free copper in the nuclei. In the progressive stage, mRNA changes throughout the cell are present, including the endoplasmic reticulum, mitochondria, and endocytic pathways, causing copper to pathologically accumulate within hepatocytes. In the later stages, mRNA for lysosomal and endosomal proteins is upregulated. In these final stages, copper concentrations decrease in the cytosol and nuclei, and accumulate in the membranous cellular compartment, causing bile duct proliferation and hepatic neoplastic changes. Therefore, the location of copper accumulation may convey more specific prognostic information about disease progression rather than total copper levels.

Other studies have compared homozygotes to compound heterozygotes of the same mutation to establish genotype–phenotype correlations. A study of 76 members of a large, consanguineous Lebanese family showed an association between c.2299insC and hepatic disease and between the p.Ala1003Thr mutation and neurologic disease (Usta et al., 2014).

Other candidate polymorphisms that are thought to modify the clinical phenotype of Wilson disease include MTHFR (Gromadzka et al., 2005), COMMD1 (Weiss, 2006), ATOX1 (Simon, 2008), XIAP (Weiss et al., 2010), PNPLA3 and hepatic steatosis (Stättermayer et al., 2012), and DMT1 (Przybyłkowski et al., 2014), although none of these genes has been demonstrated to have significant diagnostic or predictive value.

Significant phenotypic variation of Wilson disease exists between individuals with the same mutation, individuals within the same family, and even between monozygotic twins (Członkowska et al., 2009; Kegley et al., 2010). While some studies have documented high intrafamilial concordance of clinical symptoms and biochemical results (Hofer et al., 2012; Chabik et al., 2014; Ferenci et al., 2015), others have reported a wide range in age of onset and presenting symptoms amongst siblings (Ala et al., 2007; Taly et al., 2007) and families carrying the same mutation (Takeshita et al., 2002). Indeed, disparate clinical presentations in monozygotic twins raise the suspicion for epigenetic modifiers in Wilson disease. See Chapter 4 for more details about the genetic and environmental modifiers of Wilson disease.

CLINICAL MOLECULAR DIAGNOSIS

The current gold standard of diagnosis for Wilson disease is direct Sanger sequencing of the *ATP7B* gene or molecular testing for previously identified familial mutations. Historically, most pathogenic variants in *ATP7B* were identified using a combination of polymerase chain reaction (PCR)/restriction fragment length polymorphism (RFLP), single-strand conformation polymorphism (SSCP), denaturing gradient gel electrophoresis (DGGE), temporal temperature gradient electrophoresis (TTGE), denaturing high-performance liquid chromatography (DHPLC), and Sanger sequencing (Loudianos et al., 1999; Shimizu et al., 1999; Margarit et al., 2005; Vrabelova et al., 2005; G. H. Kim et al., 2008). The critical demerits of this complex tiered approach are that the detection rate is not high enough to find most mutations and the turnaround time is often extended. Although regional clusters of specific mutations have been well described, a customized screening approach taking into account these regional variants may be complicated by ethnically diverse populations and inaccurate clinical information provided with samples. Biochemical results are often imprecise, as elevations in urinary copper excretion tend to occur late in the disease process and fewer than 40% of presymptomatic patients excrete copper less than 100 μg/day (Sternlieb and Scheinberg, 1968; Nakayama et al., 2008). For these reasons, direct sequencing of the *ATP7B* gene has become the preferred standard and provides the greatest yield in clinical molecular diagnosis. Please refer to Chapter 14 for details about the diagnosis of Wilson disease.

Starting the diagnostic process with molecular testing may significantly reduce the need for invasive liver biopsy. Liver copper content alone was found to be insufficient to exclude Wilson disease, as levels may not be elevated in some affected patients. Based on several previous studies, biallelic pathogenic variants were identified in about 80% of patients with biochemical and clinical tests suggestive of Wilson disease. Currently available screening tests may not definitively rule out the disease, and no single test could permit *de novo* diagnosis. Of note, many patients may not possess the characteristic findings and may present when their clinical disease is relatively mild. Inappropriate treatment for false-positive cases has the potential of inducing copper deficiency, which can result in hematologic and neurologic sequelae (N. Kumar et al., 2003). These findings reinforce the need for reliable clinical diagnostic criteria

and underscore the benefits of DNA testing prior to invasive procedures (Ferenci, 2005).

Multiplex PCR is used to amplify all 21 exons and splice sites of *ATP7B*, including promoter regions. Although the large deletions or duplications cannot be detected with this conventional Sanger sequencing method, the chance of these being present in Wilson disease appears low (Stenson et al., 2012). If clinical suspicion is still high with only one pathogenic variant identified, then multiplex ligation-dependent probe amplification (MLPA) test should be considered. Microarray-based comparative genomic hybridization is another option to evaluate partial or full gene deletions or duplications with higher sensitivity. Cases with only one pathogenic variant present should be carefully reviewed in the context of other biochemical and clinical findings. Molecular genetic testing using direct mutation analysis is very effective in identifying affected patients and presymptomatic siblings of probands (Manolaki et al., 2009).

Wilson disease is an autosomal-recessive disorder, which means that there is a 25% chance that a full sibling of the index case is also affected. Once homozygous or compound heterozygous mutations in *ATP7B* have been established in the index patient, mutation detection becomes valuable in family screening. The same genotype in asymptomatic family members confirms diagnosis of the disease, thus allowing for early treatment before the onset of complications. In family members in whom clinical and biochemical features are uncertain, the demonstration of either heterozygous (carrier) or wild-type gene sequence prevents unnecessary treatment (Chang et al., 2007).

If the proband has secured a diagnosis of Wilson disease on the basis of clinical and biochemical evidence, but testing for *ATP7B* mutations is not available, family screening can be done by haplotype analysis of polymorphic markers flanking the disease gene (Thomas et al., 1995b; Gupta et al., 2005; Przybyłkowski et al., 2014). In this instance, the rare possibility of recombination events (typically 0.5–5% of cases) needs to be considered. The rate of recombination is dependent on which flanking markers are studied. Microsatellite or single-nucleotide polymorphisms in the *ATP7B* lateral wing are used for haplotyping, which is useful for screening relatives of patients with previously identified familial mutations. False-positive results may occur if haplotyping is used on patients with low-probability gene recombinations.

Genetic testing for *ATP7B* mutations can be valuable to confirm a diagnosis of Wilson disease, especially when presentation is unusual (Caprai et al., 2006). Attention has been drawn to this situation by the molecular confirmation of early-onset hepatic disease in a 3-year-old child (Wilson et al., 2000). Mutation analysis has also confirmed late-onset disease, including the case of two siblings in their 70s – the oldest reported patients so far at time of diagnosis (Nanji et al., 1997; Gupta et al., 2005; Perri et al., 2005; Weitzman et al., 2014).

ATP7B mutation analysis makes an important contribution to clinical practice. Unfortunately, systematic genetic testing for Wilson disease is still difficult and fairly expensive due to the plethora of different mutations, the occurrence of regulatory mutations in noncoding sequence, the large size of the gene, and the limitations of currently available methods. However, technical advances allowing high-throughput screening could be applied to the disease (Bost et al., 2012; Lepori et al., 2012). This new apparatus can sequence six million basepairs of DNA per hour with accuracy greater than 99%. Such advances might permit specialized laboratories to detect all variants by sequencing the entire genomic Wilson disease gene from patients, including not only the translated exons, but also the important noncoding sequences that are not normally investigated.

Interpretation of variants of uncertain significance has become a major challenge for accurate interpretation, genetic counseling, and prevention. Screening family members may help with the interpretation of variants of uncertain significance, but not all variants can be resolved with this approach. Functional analysis is often necessary; however, no clinical functional analysis is currently available. A computational approach to predict significance of mutations is often helpful, but a further concrete model is required to demonstrate the efficacy in guiding clinical decisions.

POPULATION SCREENING

The purpose of newborn screening is to identify treatable congenital conditions that can affect a child's long-term health and development. Recent tandem mass spectrometry (MS/MS) applications have markedly expanded the ability to screen for >50 metabolic diseases from a single dried blood spot. In addition to the original Wilson–Jungner classic screening criteria (Wilson and Jungner, 1968), the American College of Medical Genetics convened the Newborn Screening Expert Group to develop a uniform screening panel in 2006 (American College of Medical Genetics Newborn Screening Expert Group, 2006). Of the primary tenants, Wilson disease is an ideal target for screening, given its relatively high prevalence and availability of effective treatment (Hahn et al., 2002; Roberts et al., 2008). Unfortunately, despite extensive discussion on the need for population screening, no cost-effective biomarkers or methods for early detection have been developed for Wilson disease yet. Several

small pilot studies have been conducted using ceruloplasmin as a biomarker for screening, with limited findings (Yamaguchi et al., 1999; Hahn et al., 2002; Owada et al., 2002; Schilsky and Shneider, 2002; Kroll et al., 2006). Ceruloplasmin alone is not sufficient to screen for Wilson disease in newborns, as a substantial number of newborns present with physiologically low ceruloplasmin. Ceruloplasmin assay around 3 years of age may be the most appropriate population-screening method, but mandatory health checkups at this age are not universally available in the USA and worldwide.

Many treatable congenital disorders are caused by mutations that result in absent or diminished levels of proteins; thus, protein biomarkers have enormous potential in the diagnosis/screening of congenital disorders. Liquid chromatography mass spectrometry with multiple reaction monitoring (LC-MRM-MS) has emerged as a robust technology that enables highly precise, specific, multiplex quantification of signature proteotypic peptides as stoichiometric surrogates of biomarker proteins.

Our lab is currently exploring the use of peptide immunoaffinity enrichment (Whiteaker et al., 2010, 2011) to quantify ATP7B in dried blood spot (DBS). These promising proof-of-concept data open up the possibility of screening for Wilson disease in newborns. Further clinical validation on a large-scale study will be required to determine the efficacy of the assay.

CONCLUSION

Wilson disease is an autosomal-recessive disease due to pathogenic mutations in *ATP7B*. *ATP7B* is the only identified gene known to cause Wilson disease, and encodes a transmembrane copper-transporting ATPase of the same name. While biochemical testing and clinical criteria may assist in the early diagnosis and treatment, the current gold standard for Wilson disease diagnosis is direct Sanger sequencing of ATP7B or molecular testing for known familial mutations. Genotype–phenotype correlations have been studied extensively but direct causations remain nebulous. Modifier genes may affect the penetrance and phenotypes but a large-scale study for clinical validation is warranted. The overall worldwide prevalence of Wilson disease is 1 in 30 000 individuals, with significant geographic variation. The most common mutation in Northern America and Europe is the missense mutation p.H1069Q and the most common mutation in East Asian populations is the missense p.R778L. Ceruloplasmin alone is insufficient to screen for Wilson disease in newborns. While peptide immunoaffinity assays show promise for newborn screening, further large-scale clinical studies are required to determine efficacy of these population-based screening methods for Wilson's disease.

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Quantification of ATP7B Protein in Dried Blood Spots by Peptide Immuno-SRM as a Potential Screen for Wilson's Disease

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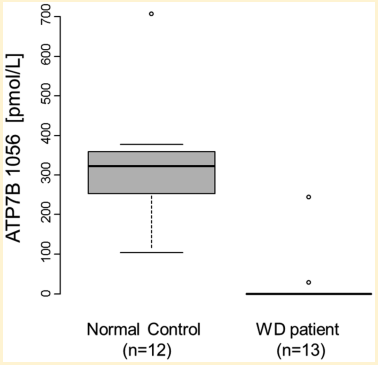
ABSTRACT: Wilson's Disease (WD), a copper transport disorder caused by a genetic defect in the *ATP7B* gene, has been a long time strong candidate for newborn screening (NBS), since early interventions can give better results by preventing irreversible neurological disability or liver cirrhosis. Several previous pilot studies measuring ceruloplasmin (CP) in infants or children showed that this marker alone was insufficient to meet the universal screening for WD. WD results from mutations that cause absent or markedly diminished levels of ATP7B. Therefore, ATP7B could serve as a marker for the screening of WD, if the protein can be detected from dried blood spots (DBS). This study demonstrates that the immuno-SRM platform can quantify ATP7B in DBS in the picomolar range, and that the assay readily distinguishes affected cases from normal controls ($p < 0.0001$). The assay precision was <10% CV, and the protein was stable for a week in DBS at room temperature. These promising proof-of-concept data open up the possibility of screening WD in newborns and the potential for a multiplexed assay for screening a variety of congenital disorders using proteins as biomarkers in DBS.



KEYWORDS: *Wilson's disease, WD, newborn screening, NBS, ATP7B, dried blood spots, DBS, immuno-SRM, peptide immunoaffinity enrichment, mass spectrometry*

INTRODUCTION

Wilson's Disease (WD) is an autosomal recessive disorder caused by mutations in *ATP7B* gene *(OMIM *606882)*.[1−3] *ATP7B* encodes a transmembrane protein ATPase (ATP7B), which is highly expressed in the liver and kidney and functions as a copper-dependent P-type ATPase. *ATP7B* is required for transmembrane transport of copper from hepatocytes into the biliary system. Absent or reduced function of ATP7B protein results in copper accumulation in the liver and subsequently in the brain, kidneys, and other organs. The impaired function of ATP7B protein also fails to incorporate copper into apoceruloplasmin, resulting in the decreased blood level of ceruloplasmin (CP) in the majority of patients with WD.[4−6]

The prevalence of WD is ∼1 in 30 000 newborns, with a carrier frequency of 1 in 90 (higher in certain populations).[7] However, regional variations exist. In particular, Costa Rica, Sardinia, the Canary Island, and Crete have all reported to have increased incidence.[8−11] WD is a slow, progressive disease. Although the biochemical defects are present from birth, patients with WD typically present with chronic hepatitis, cirrhosis, or acute liver failure in the first or second decade of life. They may have tremors, ataxia, dysarthria, and difficulty swallowing. WD was fatal until treatments were developed a half century ago. In 1955, the identification of D-penicillamine by John Walshe dramatically improved the outcome of WD by increasing the urinary excretion of copper.[12] Other treatments have since been introduced, including trientine and zinc salts, and have proven efficacious.[13−17] Unfortunately, despite the fact that effective medical treatments have been available for over 50 years and can prevent a fatal outcome if patients are diagnosed early, clinically recognizing WD remains difficult because of its slow progression and the broad clinical spectrum of symptoms. Therefore, many patients still present with irreversible multiorgan damage at the time of diagnosis.

Ideally, patients should be recognized in the presymptomatic stage. There has been a consensus that the best way to achieve early diagnosis of WD before the onset of serious symptoms is through NBS.[18−20] The current gold standard for diagnosis of WD includes multiple laboratory tests, such as copper determination in the urine and liver tissue, followed by confirmation with genetic testing of the *ATP7B* gene. These current diagnostic tools, however, are not suitable for large-scale screening.

With the discovery that CP was reduced in the majority (∼85%) of patients with WD,[5,6,20] several methods were developed to measure CP, a proposed marker for WD, in DBS or urine using different analytical platforms such as a sandwich

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ELISA assay and an LC−MS/MS assay.[19,21−25] Unfortunately, from the few pilot studies published measuring CP in infants or children, CP determination alone was insufficient to screen for WD because of high false discovery rates resulting from the fact that (1) CP is physiologically low in some unaffected newborn babies and (2) some heterozygote carriers for WD have reduced levels of CP. Pediatric screening around 3 years of age has been proposed in Japan, but it is practically difficult to screen the entire population.[26] Despite a strong need for more reliable markers or methods to meet the requirement for the universal screening of WD, no further developments have been made in attempting to screen for WD in recent years.

Most WD mutations have been shown to disrupt ATP7B stability, resulting in absent or diminished levels of the protein; thus quantifying ATP7B levels has enormous potential in the screening of WD. There are >370 mutations reported worldwide;[27,28] most are rare and infrequent, except the two most common mutations, p.H1069Q (~37−63% of the white population) and p.R778L (57% of the East Asian population).[8,29−31] In line with previous observations for disease-causing missense mutations,[32−35] some WD-associated missense mutations including p.H1069Q and p.R778L resulted in a markedly decreased level of the ATP7B protein caused by enhanced degradation.[31,36,37] Other prevalent mutations such as protein-truncating nonsense mutations (~13% of known point mutations)[38] and frameshift mutations[30] are predicted to result in the absence or decay of mRNA[39,40] or a severely truncated protein, resulting in absent or diminished levels of the protein. Taken together, it is expected that most patients with WD would have absence of or reduced levels of ATP7B. While these findings suggest that absent or diminished ATP7B levels can be an indicator for WD, it has not yet been tested.

Targeted mass spectrometry, in particular, multiple/selected reaction monitoring (M/SRM), has been used for rapid development of quantitative assays with high specificity, high-throughput, precision, and robustness,[41−45] and cross-laboratory (including international) transferability of SRM-based assays has been achieved.[45] The combination of DBS with SRM is the standard analytical approach in clinical or NBS laboratories for a variety of small metabolites that accumulate as a result of inborn errors of metabolism.[46,47] Although SRM is capable of quantifying proteins present in μg/mL and higher concentrations, many potential protein markers of greatest interest are often in the low ng/mL range. Quantification of proteins and peptides in complex samples (e.g., plasma) in the ng/mL range by SRM is challenging because of the complexity and large dynamic range of the matrix. The sensitivity of SRM is not sufficient to measure low abundance protein markers directly from DBS without an enrichment process due to interferences from more abundant analytes present in the matrix.

In recent years, peptide immunoaffinity enrichment coupled to SRM (immuno-SRM) has emerged as a promising technique for the quantification of low abundance proteins in complex matrices.[48−57] The benefits of immunoaffinity enrichment of the target peptide analyte from digests of complex samples are to greatly enrich the peptide of interest before LC−MS/MS, reducing ion suppression from background components and greatly enhancing the sensitivity of the method. Immuno-SRM has been successfully implemented to address the detection-limit challenges associated with measuring low-abundance protein biomarkers in the low- and sub-μg/L range in a wide array of studies.[48−51,58−62]

While the immuno-SRM technology has been demonstrated on clinical samples,[63,64] it has not yet been adapted for measuring low-abundance proteins from dried blood spots on filter paper. In this study, we proposed to evaluate ATP7B as a marker for WD. As proof-of-concept, we investigated the immuno-SRM methodology and applied this assay to determine the concentrations of ATP7B in DBS from unaffected and WD-affected individuals. The results demonstrate that immuno-SRM is a high-sensitivity platform for DBS analysis of proteins in the low-picomolar range and ATP7B is a potential marker for screening WD in newborns.

EXPERIMENTAL PROCEDURES

Study Approval

Blood samples involved in this study included a total of 13 WD patients and 12 healthy volunteers. Of these, 10 WD and 12 healthy subjects were from Seattle Children's Hospital and 3 WD subjects were from Asan Medical Center in Seoul, Korea. All patients signed informed consent and all research procedures were approved by the institutional review boards of aforementioned institutions. Whole blood from each subject was collected in ACD (acid citrate dextrose) tubes. Dried blood spots were prepared by pipetting 70 μL of blood/spot onto filter paper card (Protein Saver 903 Card, Whatman, Piscataway, NJ), allowed to dry at room temperature overnight, and then stored in sealed plastic bags at −80 °C until use. Fifteen year old DBS samples from proven carriers and affected patient from a previous study[21] were retrieved from −20 °C and tested as described.

Materials

ProteaseMAX Surfactant (no. V2072) was purchased from Promega (Madison, WI). Proteomics grade trypsin, bovine serum albumin protein standard (200 mg/mL), (3-[3-cholamidopropyl) dimethylammonio]-1-propanesulfonate) (CHAPS, no. PI28300) detergent, and ammonium bicarbonate (XX) were obtained from Sigma Life Science (St. Louis, MO). Acetonitrile (no. A955) and water (no. W6, LCMS optima grade), formic acid (no. PI28905), and phosphate-buffered saline (PBS, no. 10010-023) were obtained from Thermo Fisher Scientific (Waltham, MA). HepG2 cell line was obtained from the ATCC (Manassas, VA).

Generation of Immuno-SRM Assay Reagents

Rabbit polyclonal antibodies were produced by Pacific Immunology (Ramona, CA). Polyclonal antibodies were affinity purified from 25 mL of antiserum. Purified (>95% by HPLC) heavy stable isotope-labeled peptides were obtained from Anaspec (Fremont, CA). For stable isotope-labeled peptides, the C-terminal arginine or lysine was labeled with [^{13}C and ^{15}N] labeled atoms, resulting in a mass shift of +8 or +10 Da, respectively. Aliquots were stored in 5% acetonitrile/0.1% formic acid at −20 °C until use. The antibody was coupled and immobilized to 2.8 μm Protein G magnetic beads (no. 10004D, Invitrogen, Carlsbad, CA) in a 1 μg antibody-to-2.5 μL of beads ratio. In brief, 250 μL of the beads was added to 1.6 mL Eppendorf tubes and washed once with 250 μL of 1× PBS, followed by the addition of 100 μg of antibody and 1× PBS + 0.03% CHAPS (no. 28300, Thermo Scientific) to yield a total 250 μL of volume. The antibodies were allowed to couple to the beads overnight with tumbling at 4 °C. The next day, the antibodies were immobilized onto the beads as follows (the work was performed in a fume hood). The supernatant was

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Table 1. List of Candidate Peptides

peptide	sequence	molecular weight	parent ion	daughter ions[a]
ATP7B 301−313	YDPSCTSPVALQR	1435.7	718.8	579.8 (y11 + 2), 683.4 (y6), 871.5 (y8), 974.5 (y9)
ATP7B 325−339	VSLPDGAEGSGTDHR	1496.7	499.9	599.8 (y12 + 2), 656.30 (y13 + 2), 729.33 (y7), 858.37 (y8)
ATP7B 887−901	ATHVGNDTTLAQIVK	1566.8	523.3	558.4 (y5), 671.4 (y6), 772.5 (y7), 897.4 (b9)
ATP7B 1056−1077	VLAVVGTAEASSE HPLGVAVTK	2134.2	712.4	827.4 (y17 + 2), 876.9 (y18 + 2), 926.5 (y19 + 2), 966.0 (y20 + 2)

[a]Ion type for daughter ions is in parentheses.

discarded, and 250 μL of freshly prepared 20 mM DMP (dimethyl pimelimidate dihydrochloride, no. D8388, Sigma) in 200 mM triethanolamine, pH 8.5 (no. T1377, Sigma) was added. The samples were tumbled for 30 min at room temperature, and the DMP in triethanolamine was discarded. To quench the reaction, 250 μL of 150 mM monoethanolamine (no. 411000, Sigma) was added and the beads were tumbled at room temperature for 30 min. The antibody beads were washed twice using 250 μL of 5% acetic acid + 0.03% CHAPS (5 min of tumbling at room temperate each time), and washed once more using 250 μL of 1× PBS + 0.03% CHAPS. The antibody-beads suspension was finally resuspended in 250 μL of 1× PBS and stored at 4 °C until use.

Trypsin Digestion

From each DBS sample, twenty 3 mm punches (containing ∼3.7 μL of blood per disc) were obtained with a standard leather punch and placed into a 1.7 mL microcentrifuge, followed by the addition of 500 μL of 0.1% ProteaseMax in 50 mM ammonium biocarbonate (pH 8). Tubes were vortex-mixed for 1 h on Eppendorf MixMate (Eppendorf, Hamburg, Germany). At this point, aliquots were reserved for a Bradford assay. Disulfide bond reduction and trypsin digestion were performed in a single step with 2 M DTT and acetonitrile added to final concentrations of 5 mM and 15%, respectively. Trypsin was used in a 1:50 enzyme to protein ratio (w/w). The mixture was incubated in a 37 °C water bath overnight. After a 10 min centrifugation, the supernatant was transferred to a new tube, dried under a nitrogen stream, and stored at −80 °C until use.

Peptide Immunoaffinity Enrichment and Liquid Chromatography−Mass Spectrometry

The DBS digests were resuspended in 1× PBS + 0.03% CHAPS to yield a 1 μg/μL nominal protein digest concentration. Next, ∼2 mg of protein digest was combined with 4.8 μg of the antibody (immobilized on beads) in each tube and tubes were incubated overnight at 4 °C with tumbling. (The total capture volume was 500 μL.) The beads with immobilized antibodies and captured peptides were washed twice in PBS buffer + 0.03% CHAPS and washed once in PBS diluted 1:10, and peptides were eluted in 30 μL of 5% acetic acid/3% acetonitrile. The elution was frozen at −80 °C until analysis. An Eksigent Ultra nanoLC 2D system (Eksigent Technologies, Dublin, CA) with a nano autosampler was used for liquid chromatography. The peptides were loaded on a trap column (0.3 × 5 mm, C18, LCPackings, Dionex) at 10 μL/min and the LC gradient was delivered at 300 nL/min and consisted of a linear gradient of mobile phase B developed from 2 to 40% B in 18 min on a 10 cm × 75 μm column (Reprosil AQ C18 particles, 3 μm; Dr. Maisch, Germany). The nanoLC system was connected to a hybrid triple quadrupole/ion trap mass spectrometer (6500 QTRAP, ABSciex, Foster City, CA) equipped with a nano-electrospray interface operated in the positive ion SRM mode. Parameters for declustering potential (DP) and collision energy (CE) were taken from a linear regression of previously optimized values in Skyline.[65] SRM transitions were acquired at unit/unit resolution in both the Q1 and Q3 quadrupoles with 5 ms dwell time and 3 ms pause between mass ranges, resulting in a cycle time of 1.5 s. All samples were run in a blinded fashion.

Data Analysis

All SRM data were analyzed using Skyline. The presence of multiple transitions and retention time alignment with standard peptides were manually reviewed to verify detection of the correct peptide analyte. Data were exported from Skyline for analysis and plotting. The amount of the peptide in each DBS sample was determined by calculating the ratio of the peak areas for the signature peptide to that of its labeled IS present at a known concentration.

Method Assessment

Response curves were generated in a DBS matrix. The heavy stable isotope−labeled peptides were added to the tryptic digests of DBS covering the following concentrations: 0.03, 0.13, 0.67, 3.35, and 16.77 fmol/uL. Three process replicates were prepared and analyzed at all concentration points. Repeatability was determined using two samples: (i) DBS from normal control and (ii) DBS pooled from two WD affected siblings. Complete process triplicates were prepared and analyzed on three independent days. Intraassay variation was calculated as the average CV obtained within each day. Inter-assay variation was the CV calculated from the average values of the 3 days. The stability of analytes in DBS was tested by using the same DBS card stored at room temperature and at −20 °C for 0, 3, and 7 days. For each DBS sample, samples were prepared in triplicate.

RESULTS

Selection of Target Peptides

To develop a quantitative method for the quantification of ATP7B in DBS, candidate peptides for ATP7B were screened by in silico trypsin digestion, using criteria previously described,[44] followed by BLAST searching to ensure that the sequences are unique within the human genome. These peptides were screened using the tryptic digests of HepG2 cells to empirically determine which ATP7B peptides could be best detected and quantified by LC−MS/MS. Several peptides were chosen based on the intensity of the extracted ion chromatogram and their fragmentation pattern in SRM mode. Data for the four most abundant peptides are presented in Table 1, and an example of full-MS and MS/MS spectra of ATP7B 1056−1077 is shown in Figure 1. Affinity-purified, rabbit polyclonal antibodies were generated against all four peptides. Because the sequence for ATP7B 1056 is highly hydrophobic in the N-terminal region (the first five amino acids in particular), the shortened sequence, ATP7B 1061−1077, was

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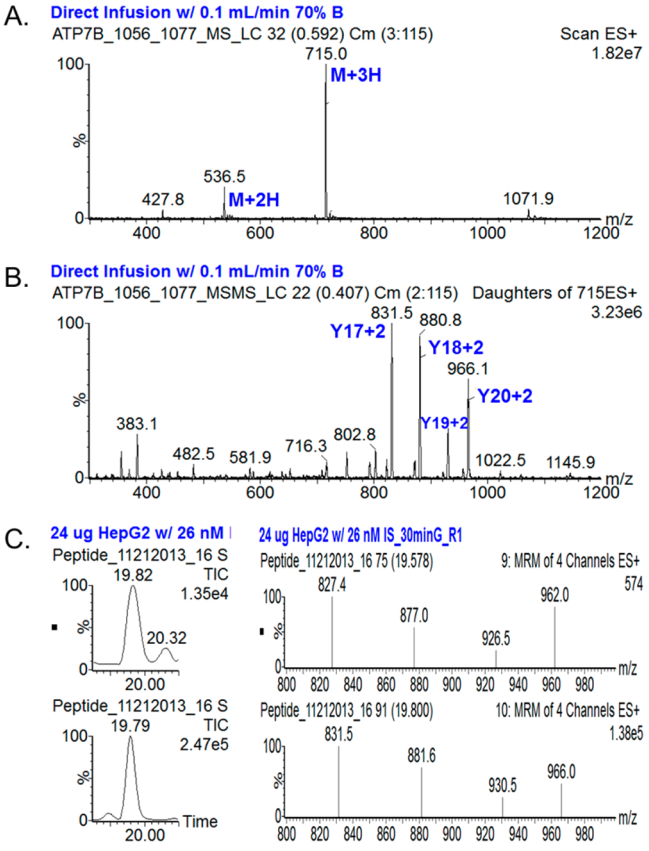

Figure 1. (A) Mass spectrum of heavy peptide 1056 for ATP7B and (B) tandem mass spectrum of the most abundant parent ion (M+3H). Abundant fragments are selected and optimized for SRM analysis. (C) Total ion chromatogram (TIC) and SRM spectra of endogenous (top) and heavy (bottom) peptide 1056 observed in HepG2 cell extract. Chromatographic peaks overlap and SRM patterns are compatible.

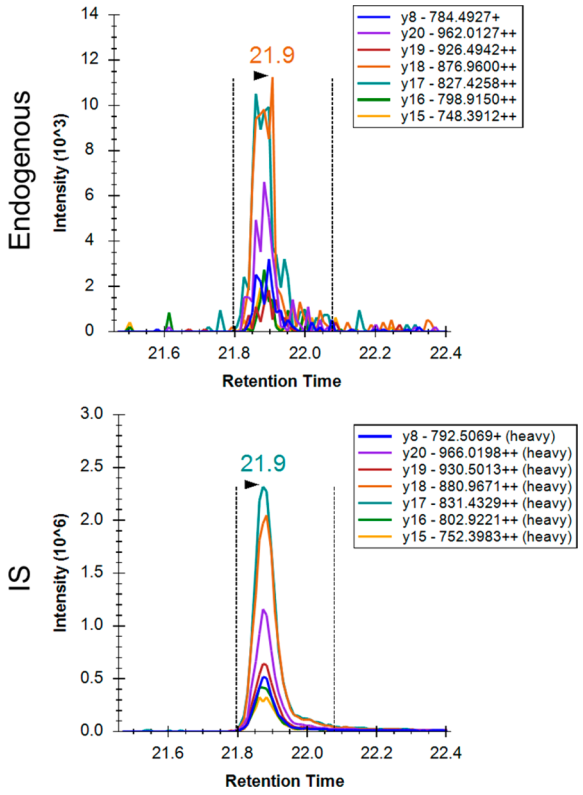

Figure 2. Extracted ion chromatograms for ATP7B 1056 peptide after peptide capture in normal PBMC. Top panel is a signature peptide found in the PBMC. Bottom panel is the isotopically labeled internal standard. Chromatographic peaks overlap, and SRM patterns are comparable. Transition labels refer to the precursor charge, fragment ion, fragment *m/z*, and fragment charge state.

used as a target for polyclonal antibody generation. While polyclonal antibodies for ATP7B 301 and 887 allowed very weak recovery of the peptides, ATP7B 325 and 1056 peptides showed recovery efficiencies ranging from ~50 to 70%. Of these two peptides, the ATP7B 1056 peptide was pursued further as a target peptide to quantify ATP7B in subsequent human samples because: (i) there was no background signal resulting from carrier peptides copurified with the antibodies[49,50,66] and (ii) the most common mutation, p.H1069Q, occurs in this peptide, taking advantage of absence of this peptide in WD patient with p.H1069Q.

Quantification of the Target Peptide by Immunoaffinity Peptide Enrichment and LC–MS/MS

While ATP7B is highly abundant in several tissues including liver, kidney, and placenta,[67] it is known to present at very low abundance in white blood cells, and it has also been observed in blood including lymphocytes and erythrocytes (gpmdb.thegpm.org). Thus ATP7B 1056 peptide was first analyzed in peripheral blood mononuclear cells (PBMCs) to see if we could identify the peptide. Isolated PBMCs were lysed and digested by trypsin, and the ATP7B 1056 immuno-SRM assay was used to enrich the peptide upstream of LC–SRM. A chromatogram of the sample eluate showing the ATP7B 1056 peptide identified in PBMC is shown in Figure 2. We next tested the feasibility of measuring ATP7B 1056 peptide in DBS. These results show the feasibility of developing an assay in DBS; we then characterized and assessed the assay for use in DBS samples.

Assay Assessment

The linearity, imprecision, and stability of the assay was assessed by following fit-for-purpose guidelines (detailed in the Experimental Procedures section).[68] The linear dynamic range was determined by generating a five-point response curve using synthetic standard peptides. Three DBS samples for each concentration level were prepared to account for any variation in protein extraction from the DBS card. These samples were analyzed in the order of increasing concentration with one blank injection between different sample concentrations. The assay showed a linear response (r^2 = 0.99) for all peptide amounts tested, spanning the peptide concentration of 27 to 16 765 pmol/L (0.7 to 417 femtomoles) (Figure 3). Results with a signal-to-noise ratio (S/N) < 10 were considered unreliable data. The low limit of quantification (LOQ) was estimated to be ~27 pmol/L based on the lowest concentration of the response curve (27 pmol/L) and patient sample no. 1 (29.5 pmol/L). The average CV was <12% for the three replicates of DBS samples prepared and analyzed at each concentration level. The linear response with the high reproducibility shows constant protein recovery and protein digestion efficiency for the target peptide. We assessed intra- and inter-assay imprecision by the LC–SRM analysis of complete process triplicates prepared from a healthy subject and a pooled patient sample and analyzed on three independent days. The results are summarized in Table 2.

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J. Proteome Res. 2017, 16, 862–871

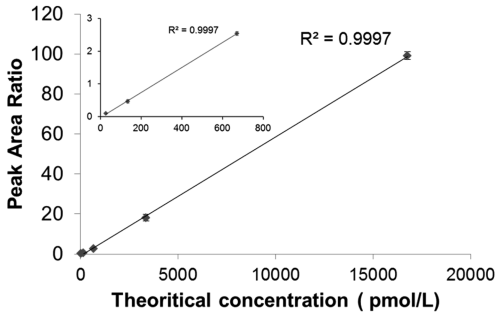

Figure 3. Response curve for ATP7B 1056 peptide. Curves are plotted for the sum of all transitions. The inset plot shows more detail of the lower end of the concentration range. Error bars are the standard deviation of three process replicates.

Table 2. Intra-Assay and Inter-Assay Imprecision of Immuno-SRM Assay for ATP7B 1056 Peptide[a]

sample	intra-assay, %	inter-assay, %
normal control	8.42	2.9
WD patient	NA	NA

[a]NA, not applicable.

Intra- and inter-assay imprecision from the normal control were 8.4% CV and 2.9% CV, respectively, suggesting the high reproducibility of both sample preparation and the method of analysis. The assay imprecision from the pooled WD patients was not calculated because most peaks observed were below the LOQ. The stability data for the ATP7B peptide is presented in Figure 4. The ATP7B peptide was stable for at least 7 days in DBS at RT and −20 °C.

Evaluation of ATP7B as a Marker for Early Screening of WD

To determine if the chosen target peptide could be used as a screening marker for WD, a total of 25 DBS samples from 12 controls and 13 confirmed WD patients were analyzed in a blinded fashion for ATP7B. Representative SRM traces

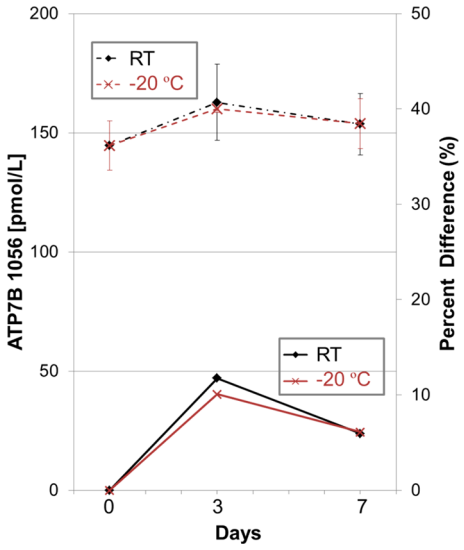

Figure 4. Stability of ATP7B 1056 peptide in normal control DBS at room temperature and −20 °C for 0, 3, and 7 days. The data represent the average of three replicates. Dashed and solid lines represent ATP7B concentrations and percent difference, respectively. Error bars are the standard deviation of three process replicates.

obtained from a healthy control and a WD patient are presented in Figure 5. The results are summarized in Table 3

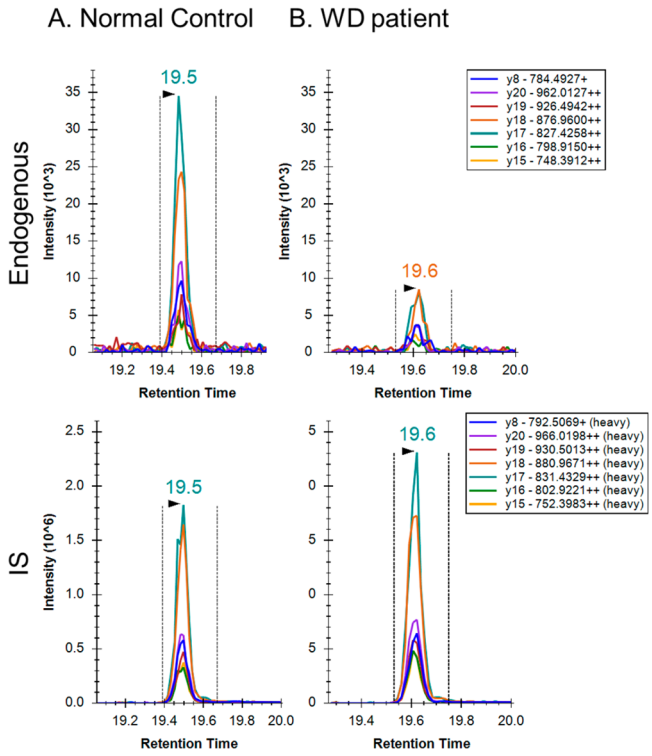

Figure 5. Extracted ion chromatograms for ATP7B 1056 peptide after peptide capture in DBS from (A) normal control and (B) WD patient. Top panel is a signature peptide found in DBS. Bottom panel is the isotopically labeled internal standard. Chromatographic peaks overlap and SRM patterns are comparable. Transition labels refer to the precursor charge, fragment ion, fragment m/z, and fragment charge state.

and Figure 6. As shown, the assay readily distinguished affected patients from controls ($p < 0.0001$). While we were able to reliably detect endogenous ATP7B ranging from 105 to 708 pmol/L in normal controls, the analyte response from WD patients was either not detected or below 29.5 pmol/L, except for one case, WD08. Of note, there was no ATP7B 1056 peptide detected in either of the WD patients who carried p.H1069Q mutation, as predicted. Case 8 presented with presumably alcoholic liver cirrhosis at the age of 56 years with a history of chronic jaundice, ascites, and hepatosplenomegaly. The copper content in the liver biopsy was marginally elevated at 116 μg/g dry weight tissue (control <35 μg/g), which prompted a genetic test for *ATP7B* gene. She was found to carry one known pathogenic variant, p.Thr974Met, and one variant of unknown significance, p.Ser391Leu in trans. Her 24 h urine copper was normal at 17 μg/24 h, serum CP was within the normal range, and no Kayser−Fleischer ring was detected in her eyes. Given her clinical and biochemical evaluations, she was suspected to be a presumtive carrier for Wilson's Disease, and the VUS was predicted to be benign in nature. Her mildly elevated copper content in the liver tissue was considered most likely secondary to long-standing liver disease.

The levels of ATP7B 1056 peptide were determined to see if the assay can distinguish between WD patients and proven carriers. Because of the high prevalence of carriers, it is important that a screening test should be able to limit or avoid

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Table 3. ATP7B 1056 Peptide Concentration in 13 DBS Samples from WD Patients

sample	ATP7B 1056 (pmol/L)	mutation
WD01	29.5	p.R778W and p.T977M
WD02	ND[a]	p.H1069Q and p.R1319*
WD03	ND	p.H1069Q and p.R1319*
WD04	ND	not available at this time
WD05	ND	p.R778L homozygote
WD06	ND	p.C2304_2305insc and p.L1083F
WD07	NA[b]	p.R778L and p.A874 V
WD08	244.8	p.Thr974Met and p.Ser391Leu
WD09	ND	p.R778G and p.K175S_fs/p.Q260P_fs
WD10	ND	p.R778G and p.K175S_fs/p.Q260P_fs
WD11	ND	p.R778L and p.E1064A
WD12	ND	p.R778L and p.E1064A
WD13	ND	p.H1069Q and p.Y1331Tfs*61
controls (N = 12)	320.9 ± 147.4	

[a]ND, not detected. [b]NA, not applicable due to S/N < 10.

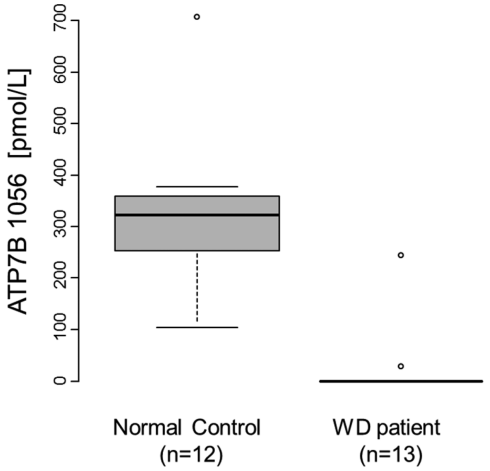

Figure 6. Distribution of the levels of ATP7B in DBS from 13 WD patients and 12 normal controls. The bold black line indicates the median, the inner quartiles are represented by boxes, and the whiskers show 95% of the data.

carrier detections. A set of DBS specimens used for this test included a WD patient, two proven carriers (the WD patient's mother and father), and two age-matched normal controls. These DBS had been stored at −20 °C for ∼15 years. The results are shown in Table 4. ATP7B 1056 peptide in the WD patient was markedly reduced compared with the two carriers and two age-matched controls. The levels of ATP7B 1056 peptide did not differ between the carriers and age-matched controls. Of note, the levels of ATP7B from the WD patient and the four controls were in the range of the WD patients and the control groups tested in this study, indicating that the protein in DBS could be stable for many years.

Table 4. ATP7B 1056 Peptide Concentration in 15 year old DBS

sample	ATP7B 1056 (pmol/L)
WD patient	60.3
Carrier 1 (mother)	270.9
Carrier 2 (father)	251.4
age-matched NC 1	369.5
age-matched NC 2	452.6

DISCUSSION

WD is a progressive and fatal disorder that is treatable, where early detection can make a significant impact on disease outcome and even be life-saving. However, there is currently no suitable marker and method available for population screening. In this study, we: (i) propose ATP7B as a potential marker to screen WD, taking advantage of an absence or decrease in the amount of the ATP7B protein in most WD patients; (ii) provide a sensitive immuno-SRM assay for the quantification of ATP7B in DBS, demonstrating the feasibility of DBS/immuno-SRM formats for screening congenital disorders lacking marker proteins; and (iii) suggest our approach can be further applied to aid diagnostics in conjunction with clinical and other biochemical test results.

We were able to reliably detect endogenous ATP7B in DBS from normal controls in the range of 105−708 pmol/L. To our knowledge, the detection and quantification of ATP7B protein in DBS has never been achieved with any method before. In 12 out of 13WD patients, the amount of the deficient ATP7B was below the control range, suggesting the feasibility of the use of ATP7B as a potential marker for WD. This approach is unique as most studies are looking for accumulated metabolites or markers, whereas this assay directly analyzes the affected protein. Although the number of samples tested in this study is quite limited, the results on the patients carrying the two most common mutations (p.R778L and p.H1069Q) are promising and demonstrate the feasibility of the immuno-SRM approach for mass screening.

The result on case 8 highlights the use of this assay for those patients with ambiguous genetic or biochemical test results, which is not an uncommon situation in the clinic. The variant of uncertain significance is not uncommon in ATP7B gene, while there is no definite diagnostic test available. The diagnosis for WD could be challenging, especially with those ambiguous or borderline results that could potentially lead to unnecessary treatment. Our result seems very promising in aiding the appropriate and accurate diagnosis in conjunction with other genetic and biochemical test results, although further studies on many clinical samples are necessary.

While the data presented in this study indicate the possible use of ATP7B in DBS for screening WD, we acknowledge that the findings are preliminary. Larger studies including both controls with proven carriers and patient samples with a broad mutational spectrum will be required to determine more

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accurate reference, disease ranges, and cutoff. In addition, patient samples tested in this study are limited to children or adults due to the difficulty of identifying newborn samples from affected patients. Although we expect no significant age dependence of its abundance, the effect of age on the level of ATP7B protein (in particular for newborns) is not known. As with all NBS assays, the implementation of this assay will need to be tested in the newborns or infants on whom the testing would be carried out.

As anticipated, there was no detectable ATP7B 1056 peptide in either of the WD patients who carried the p.H1069Q mutation that occurs within this peptide. However, it can be argued that the absence of this peptide could be due to sequence variations (mutations/polymorphisms) from presumed healthy subjects. This could be resolved by monitoring multiple peptides for each target protein, which will help ensure that negative results are truly negative. The reduced/absence of multiple peptides in a SRM assay could increase the confidence of a negative result. This applies to the quantification of any protein to prevent underestimation due to single nucleotide polymorphisms and posttranslational modifications.

Becaue of the nature of the individual variants, measurement of a target protein, ATP7B, may not be sensitive enough to identify all affected individuals. For example, some patients with mutations that affect protein function/structure but not quantity may not be detected. One approach to address this limitation is the use of additional/secondary markers such as CP. When the primary marker shows ambiguous or undetermined result, the application of secondary markers can help improve both the sensitivity and specificity of the assay. It is important to note that the ability to measure multiple analytes in the same analysis would not require additional sample process or sample collection.

DBS offers many advantages such as a less invasive sampling, a simpler storage, and an easier transfer. However, there are several variables that may affect how uniformly blood and analytes spread across the filter paper, influencing precision in quantitative analysis. These include the hematocrit values, blood volume spotted, and chromatographic effects. There are contradicting reports in the literature as to whether these variables have a direct impact on the precision of the analytical result.[69,70] Note that in our proof-of-principle study we chose to reduce the effects of these variables by spotting the same volume of blood for all DBS samples used in experiments in this study and using nearly the entire blood spots for analysis.

The performance characteristics of our immuno-SRM assay were found to be compatible with expectations for clinical sample analysis. The inter-assay and intra-assay CV of ATP7B assay using DBS from a healthy control specimen were <10%, demonstrating that the assay is precise. The response curve was linear, with an R^2 of 0.99 and the dynamic range from 27 to 16 765 pmol/L. The use of immuno-SRM also offers other compelling advantages with respect to NBS. These advantages include: (i) this assay can be multiplexed without loss of specificity and sensitivity, enabling simultaneous analysis of multiple proteins from a single sample and a single injection, permitting greater statistical power to be achieved and robust cross-correlations to be made and (ii) screening programs already have been using MS/MS technology, in particular, SRM.

DBS is an attractive alternative to the collection of plasma or serum, particularly for NBS. Analytes are known to be more stable in DBS compared with those in plasma, blood, or other solutions,[71] likely because of the dehydration of the sample on the card and consequent minimization of chemical and enzymatic hydrolysis of the analytes.[71] Note that in our bottom-up approach to quantify the protein we focused on the integrity of the targeted peptide that serves as surrogates for the protein and not the intact protein itself. Our stability test showed that the ATP7B peptide is fairly stable in DBS for a week at room temperature and −20 °C. Additionally, results from the ATP7B peptide from DBS stored at −20 °C for ∼15 years were comparable to fresh samples, suggesting good long-term stability. Our data strongly suggest that peptides in DBS may be stable, opening up the possibility of application of immuno-SRM to NBS for testing for other genetic conditions such as primary immunodeficiencies or cystinosis.

While screening programs already utilize MS/MS technology for small molecules, immuno-SRM for quantification of proteins is a novel platform in the clinical and NBS laboratories. The method for measuring proteins uses quite different procedures from measuring small metabolites. The assay requires a relatively large number of steps before LC−MS/MS: (i) proteolytic digestion of proteins in the DBS; (ii) immunoaffinity enrichment of target peptides; and (iii) MS/MS coupled to liquid chromatography. Although this is a relatively complex assay format, the implementation of robotic sample preparation for trypsin digestion and peptide enrichment and a robust chromatography configuration[45] should enable this technology to advance into routine clinical analysis for thousands of samples.

A limitation for setting up an immuno-SRM assay may be the time required and likelihood of success in generating an antipeptide antibody.[66] Monoclonal antibodies are preferable for clinical or screening assays because they provide a renewable resource for long-term supply with acceptable consistency and reproducibility. Some monoclonal antibodies outperform the polyclonal antibodies with increased recovery efficiency and sensitivity of the immuno-SRM assay.

■ CONCLUSIONS

We developed a novel, sensitive immunoaffinity LC−MS/MS assay for quantitative measurement of ATP7B levels in DBS. This study demonstrates the feasibility of the use of immuno-SRM to quantify ATP7B in DBS to screen for WD. To the best of our knowledge, this is the first published report of employing immuno-SRM strategy for measuring a clinically important, low-abundance protein in DBS. The described method opens up future opportunities for the analysis of other protein markers in DBS for many other life-threatening congenital disorders that are currently not a part of the NBS.

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Notes

The authors declare no competing financial interest.

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CLINICAL—LIVER

Direct Measurement of ATP7B Peptides Is Highly Effective in the Diagnosis of Wilson Disease

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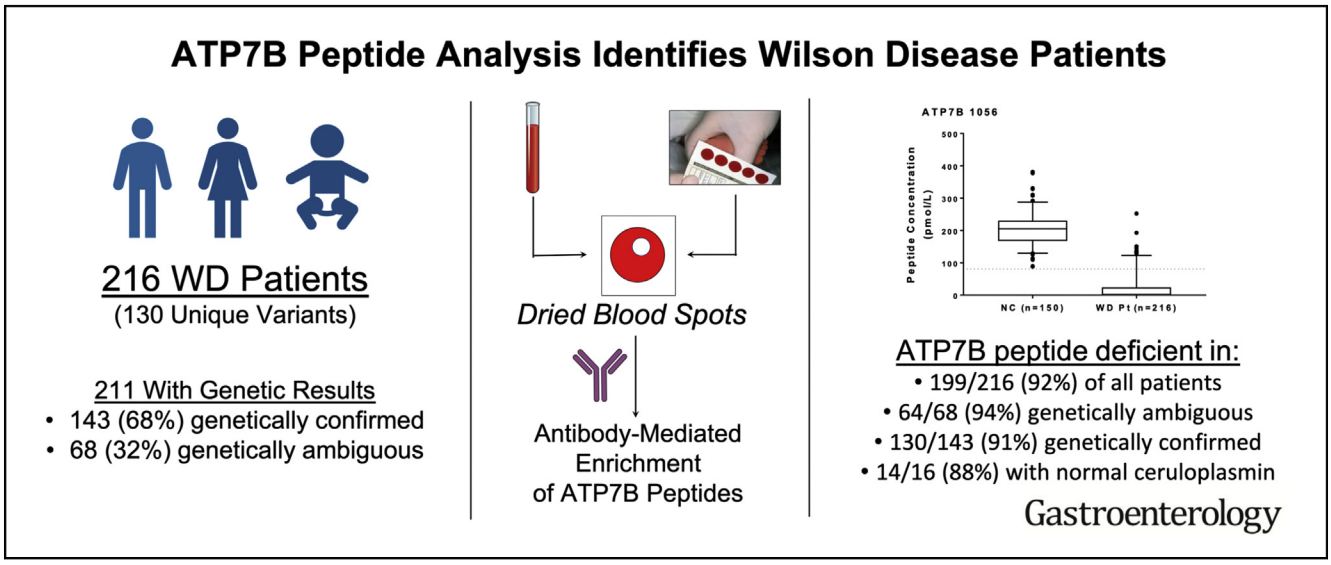

See Covering the Cover synopsis on page 2231; See editorial on page 2249.

BACKGROUND & AIMS: Both existing clinical criteria and genetic testing have significant limitations for the diagnosis of Wilson disease (WD), often creating ambiguities in patient identification and leading to delayed diagnosis and ineffective management. ATP7B protein concentration, indicated by direct measurement of surrogate peptides from patient dried blood spot samples, could provide primary evidence of WD. ATP7B concentrations were measured in patient samples from diverse backgrounds, diagnostic potential is determined, and results are compared with biochemical and genetic results from individual patients. **METHODS:** Two hundred and sixty-four samples from biorepositories at 3 international and 2 domestic academic centers and 150 normal controls were obtained after Institutional Review Board approval. Genetically or clinically confirmed WD patients with a Leipzig score >3 and obligate heterozygote (carriers) from affected family members were included. ATP7B peptide measurements were made by immunoaffinity enrichment mass spectrometry. **RESULTS:** Two ATP7B peptides were used to measure ATP7B protein concentration. Receiver operating characteristics curve analysis generates an area under the curve of 0.98. ATP7B peptide analysis of the sequence ATP7B 887 was found to have a sensitivity of 91.2%, specificity of 98.1%, positive predictive value of 98.0%, and a negative predictive value of 91.5%. In patients with normal ceruloplasmin concentrations (>20 mg/dL), 14 of 16 (87.5%) were ATP7B-deficient. In patients without clear genetic results, 94% were ATP7B-deficient. **CONCLUSIONS:** Quantification of ATP7B peptide effectively identified WD patients in 92.1% of presented cases and reduced ambiguities resulting from ceruloplasmin and genetic analysis. Clarity is brought to patients with ambiguous genetic results, significantly aiding in noninvasive diagnosis. A proposed diagnostic score and algorithm incorporating ATP7B peptide concentrations can be rapidly diagnostic and supplemental to current Leipzig scoring systems.

Keywords: Wilson disease; Leipzig Score; Immuno-SRM; ATP7B.

Wilson disease (WD) is named for Dr Samuel Alexander Kinnier Wilson, who first described the disorder in his 1912 doctoral thesis. Since then, treatments have been developed for WD and, importantly, WD has become a preventable disease. WD is an autosomal recessive disorder of copper metabolism due to mutations in the *ATP7B* gene that encodes copper-transporting P-type ATPase (EC # 7.2.2.8).[1–3] WD has an estimated prevalence of 1:30,000 and a carrier frequency of 1:90 with regional variation.[4,5] Although guidelines for the diagnosis of WD have been developed,[6,7] patient identification remains a challenge resulting in delayed diagnosis and development of irreversible severe complications, such as permanent brain or liver damage, which render treatments ineffective.[5]

The key features of WD are liver disease, neuropsychiatric abnormalities, and Kayser-Fleischer (KF) rings. The presence of KF rings with neurologic manifestation and/or low serum ceruloplasmin (Cp) is considered enough to establish WD diagnosis. However, most cases require a combination of clinical symptoms and laboratory evaluations.[8] Currently, no single test permits de novo WD diagnosis in every potential patient.[7] Serum Cp is decreased in neurologic WD, but can be in the low-normal range in up to 50% of adult patients with active liver disease[9,10] and the positive predictive value of serum Cp for diagnosis of WD is poor.[9,11,12] In children with WD, 15%–36% had Cp in the normal range.[13] Serum Cp alone is not sufficient to diagnose or exclude WD. A diagnostic score (Leipzig score, 2003) was proposed to guide clinical diagnosis and has been adopted in the clinical practice guidelines for the European Association for the Study of the Liver.[14] Although recent advances in clinical molecular diagnosis have greatly improved the accuracy of WD diagnosis in affected patients and their siblings, traditional Sanger sequencing cannot detect large deletion or duplications. In addition, there are many single nucleotide polymorphisms and variants of unknown significance (VUS) in the ATP7B gene. Interpretation of genetic sequencing results, particularly in the presence of only one identified mutation or VUS, create ambiguity in WD patient identification.

We evaluated the direct measurement of ATP7B protein from WD patient dried blood spots (DBS), through surrogate ATP7B peptides, as a diagnostic tool.[15] As reported in our previous studies for multiple primary immunodeficiency conditions, peptide measurements are made using immunoaffinity enrichment coupled to selected reaction monitoring (immuno-SRM) mass spectrometry.[16,17] This method uses antipeptide antibodies to concentrate and quantify extremely-low-concentration peptide targets from complex matrices, including DBS.[15–21] Analysis of ATP7B concentration in DBS from WD patients with a broad range of genetic backgrounds shows that ATP7B peptide levels are greatly reduced. Analysis of ATP7B protein concentration can identify WD with high diagnostic accuracy.

WHAT YOU NEED TO KNOW

BACKGROUND AND CONTEXT

Identification of patients with Wilson disease remains a challenge, resulting in delayed diagnosis and development of irreversible severe complications, such as permanent brain or liver damage, which render treatments ineffective.

NEW FINDING

Directly measuring ATP7B from dried blood spots of patients with Wilson disease with diverse genetic backgrounds showed ATP7B peptide concentrations have a high diagnostic potential.

LIMITATIONS

The study is limited in that data are mostly obtained from White patients. Sensitivity and specificity may be variable geographically.

IMPACT

ATP7B peptide analysis identifies most patients with Wilson disease, reducing ambiguities resulting from genetic analysis, and is expected to advance the use of proteomics, a promising but largely clinically untapped technology.

Methods

Dried Blood Spot Samples

This protocol was approved by the Institutional Review Board of Seattle Children's Hospital (SCH) and each of the participating institutes. All subjects gave written informed consent. Patient and carrier samples were provided by SCH, Seattle, WA; Medical University of Vienna, Austria; Medical University Innsbruck, Austria; University of Medicine and Pharmacy, Iuliu Hatieganu, Cluj-Napoca, Romania; Wilhelmina Children's Hospital, University Medical Center, Utrecht, The Netherlands; Yale University, New Haven, CT; University of Heidelberg, Germany; and Asan Medical Center, Seoul, South Korea. Samples were prepared either by fingerstick or by pipetting 70 μL of blood (per 12-mm spot) onto filter paper cards (903 ProteinSaver; Whatman, Piscataway, NJ). The samples were then dried overnight at room temperature, delivered to SCH, and stored at −80°C until use. One hundred and fifty normal control DBS samples (BioIVT, Westbury, NY) were analyzed to establish the normal reference range and cut-off.

Abbreviations used in this paper: **ACN, acetonitrile; AF, allele frequency; CHAPS, 3-([3-cholamidopropyl] dimethylammonio)-1-propanesulfonate; Cp, ceruloplasmin; CV, coefficient of variation; DBS, dried blood spot; FA, formic acid; immuno-SRM, immunoaffinity enrichment coupled to selected reaction monitoring; IS, internal standard; KF, Kayser-Fleischer; LC/MS, liquid chromatography/mass spectrometry; LLOD, lower limits of detection; LLOQ, lower limits of quantification; mAb, monoclonal antibody; SCH, Seattle Children's Hospital; VCI, variant with conflicting interpretations; VUS, variants of unknown significance; WD, Wilson disease; WT, wild-type.**

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Immunoaffinity Enrichment Coupled to Selected Reaction Monitoring Reagents

Triton X-100 (T9284, 100 mL) and Ammonium bicarbonate (A6141-25G) were purchased from Sigma-Aldrich (St. Louis, MO). TPCK-treated Worthington trypsin (LS003740) was purchased from Worthington (Lakewood, NJ). 3-([3-cholamidopropyl]dimethylammonio)-1-propanesulfonate (CHAPS) (no. 28300), acetonitrile (ACN) (liquid chromatography/mass spectrometry [LC/MS] grade), acetic acid (LC/MS grade), water (Optima LC/MS grade), formic acid (FA) (Optima LC/MS grade), 1× phosphate-buffered saline (no. 10010-023), dithiothreitol (no. 20290), and 1M Tris-(hydroxymethyl)aminomethane (pH 8) (no. 15568-025) buffer were obtained from Fisher Scientific (Waltham, MA). Protein G–coated magnetic beads (Dynabeads, no. 10004D) were purchased from Invitrogen (Carlsbad, CA).

Isotope-labeled internal standard (IS) peptides were purchased from either Atlantic Peptides (Lewisburg, PA) or Life Technologies Corporation (Carlsbad, CA). IS peptides were >95% pure and incorporated heavy stable isotope-labeled (^{13}C and ^{15}N) C-terminal lysine (+8 Da) or arginine (+10 Da). IS stock solutions are stored frozen as 500× mixtures in 1× phosphate-buffered saline + 15% ACN + 0.1% FA + 0.03% CHAPS in H_2O and diluted to 1× immediately before use.

Selection of Signature Peptides and Antibody Production

Selection and production of ATP7B 1056 peptide and antibody has been described in previous reports.[15–17]

Using the same guidelines, ATP7B 887 was selected. Antibody production was performed by Excel Biopharm (San Francisco, CA), Pacific Immunology (Ramona, CA), and ExonBio (San Diego, CA). Enzyme-linked immunosorbent assays were performed, by the companies mentioned, in bleed samples after immunization and in supernatant samples after monoclonal antibody production. Selections were further confirmed at each step by the immuno-SRM method.[22]

Antibody Bead Reagent Production

Monoclonal antibody (mAb) beads were produced by overnight 4°C incubation of Protein G Dynabeads with mAb, as reported previously.[15–17]

Dried Blood Spot Extraction, Trypsin Digestion, and Immunoaffinity Enrichment

Protein extraction and tryptic digestion were performed as reported previously, with slight modifications.[16] One 6.35-mm diameter DBS punch was placed into 96-well plates (ThermoScientific, Chicago, IL), covered with an adhesive seal (Genesee Scientific, San Diego, CA), and extracted using 0.1% Triton X-100 in 50 mM ammonium bicarbonate (200 μL) with dithiothreitol (final concentration 0.2M). After 30 minutes incubation at 37°C with agitation, trypsin (37.5 μg) was added and incubated for 2 hours at 37°C. For enrichment, 10 μL of 1M TRIS (pH 8) and 10 μL of 1× ATP7B IS mix were added (final concentrations = 0.25 nM). Extracted supernatant (200 μL) was transferred to a new plate and incubated with 2.5 μL of each mAb bead overnight at 4°C with agitation.

After incubation, mAb beads were isolated using a 96-well magnetic plate (Alpaqua Magnum EX, Beverly, MA), washed twice with 1× phosphate-buffered saline + 0.01% CHAPS, and magnetically isolated. Peptides were eluted with 30 μL of H_2O with 5% acetic acid and 3% ACN for 5 minutes and transferred to a new 96-well plate for analysis (Abegene, Chicago, IL).

Liquid Chromatography-Mass Spectrometry

LC-MS/MS was performed using a Waters Xevo TQ-XS with Ionkey source and dual M-Class chromatography pumps (Milford, MA). Chromatographic solvents were A: H_2O + 0.1% FA and B: ACN + 0.1% FA. Peptides are loaded onto an M-Class Trap Symmetry C18 column (300 μM x 25 mm, 100A, 5 μM) for 3 minutes with a constant flow of 98:2 A:B at 20 μL/min. After loading, the flow is reversed and peptides are separated using a 150 μM × 100 mm BEH C18 ionkey (130 Å, 1.7 μM). The gradients used are summarized in Supplementary Table 1 and were reported previously.[16] Precursor mass, fragment mass, and collision energy were tuned to optimize the generated signal (Supplementary Table 2). Representative chromatograms for both ATP7B 887 and ATP7B 1056 peptides were shown in Supplementary Figure 1*A–D*.

Concentration Calculation and Data Analysis

Selected reaction monitoring data captured in the MS were analyzed using Skyline (MacCoss Lab, Seattle, WA, https://skyline.ms/project/home/begin.view).[23] Specificity was assured by monitoring retention times and relative transition intensities of endogenous and IS peptides. Concentrations of endogenous signature peptides were calculated using endogenous/IS signal ratio. DBS spots are assumed to contain 70 μL of evenly distributed whole blood. The volume of blood in the punch area is calculated as 17.5 μL. Concentrations are calculated from blood volume, ratio, and IS concentration. Statistical analyses and receiver operating characteristics curves were generated using GraphPad Prism (San Diego, CA).

Method Performance Assessment

Response curves were generated for each peptide to establish assay linearity and determine the lower limits of detection (LLOD) and quantification (LLOQ). Seven concentrations (0×, 0.05×, 0.1×, 0.5×, 1×, 5×, and 50×) of IS were added across the set of pooled digest samples in triplicate. LLOD was calculated as: $LLOD = mean_{blank} + 3 \times SD_{Low}$ ($mean_{blank}$: mean signal from a triplicate blank injection, SD_{Low}: SD of IS injection below the LLOQ). LLOQ is the lowest concentration with a coefficient of variation (CV) of <20%. The linearity curves of ATP7B 1056 and ATP7B 887 are shown in Supplementary Figure 1*E* and *F*.

The assay precision and accuracy were evaluated by within-day (intra-) and between-day (inter-) assay CV, respectively. The intra- and inter-assay CV were determined using 5 replicates of an identical pooled blood DBS sample each day over a course of 5 days (Table 1).

Internal Sample Quality Control

Measurement of endogenous peptides unrelated to WD and, therefore, assumed to be present at normal concentrations, was used as an internal quality control to monitor the successful

CLINICAL LIVER

Table 1. Analytical and Diagnostic Performance for ATP7B Peptides

Peptide	LLOD, *pmol/L*	LLOQ, *pmol/L*	Intra-assay CV, %	Inter-assay CV, %	PPV, %	NPV, %	AUC
ATP7B 1056	3.81	71.43	12.9	15.3	96.1	91.3	0.98
ATP7B 887	2.17	7.14	11.0	13.0	98.0	91.5	0.98

AUC, area under the curve; NPV, negative predictive value; PPV, positive predictive value.

extraction, digestion, and enrichment of target peptides. These peptides are ADA 93, representing adenosine deaminase, CD42 128, representing glycoprotein Ib, and IDUA 462, representing α-L-iduronidase.[16] A sample run was assumed to be of sufficient quality if the measured concentration of 2 of these 3 peptides was within 1.75 SD of the mean for the cohort (Supplementary Tables 3–5). Samples failing these acceptance requirements were repeated. With confirmatory test failure, the DBS was removed from the sample cohort due to inadequate matrix and an additional sample was requested.

Results

Characteristics of Patient Cohort

WD DBS samples were obtained from 198 White and 18 Far Eastern Asian patients with WD (114 male and 102 female) and 48 obligate carriers (Supplementary Tables 3 and 4). All carriers are obligate heterozygote from the family members of index case patients with 2 confirmed variants. Patient age range spanned from 2 months to 73 years. For the purpose of stability validation, samples from 11 patients and 1 healthy normal subject were collected from both fresh and blood samples stored up to 11 years prior (Supplementary Table 7). Clinical information including Cp concentrations, Leipzig scores, liver copper content, presence of KF rings, initial presentation, and presence of cirrhosis are presented where available (Supplementary Table 3). Control DBS samples (n = 150) were obtained from healthy subjects ranging from 18 to 73 years of age.

An analysis of the specific variants in the sample set showed that the cohort contained 130 unique variants (Figure 1*B*, Supplementary Table 6), including 83 pathogenic or likely pathogenic variants, 43 VUS, 3 benign or likely benign variants, and 1 with conflicting interpretations (VCI). In affected patient samples, 143 exhibited only pathogenic or likely pathogenic mutations according to a public database (ClinVar, gnomAD [Genome Aggregation Database]), including 31 patients homozygous for p.H1069Q, the most common variant in WD patients (Figure 1*A*, Supplementary Table 3). In addition, 20 patients were homozygotes for other variants. Seven patients exhibited 2 VUS (2 of them are homozygotes). In addition, 37 patients were compound heterozygous for 1 VUS and 1 pathogenic or likely pathogenic mutation. Eighteen patients had only 1 variant detected by Sanger sequencing. No second variant was detected. Three clinically suspected patients were compound heterozygotes, with 1 likely benign variants according to gnomAD and the second variant with likely pathogenic, pathogenic, and unknown, respectively.

Forty-eight samples from obligate carriers, all of them family members of affected patients, presented with 2 variants, had a single pathogenic variant and a single wild-type allele or a benign variant (Supplementary Table 4).

Surrogate Peptide Markers for ATP7B

The mean ± SD signature peptide concentration in normal control was 257.7 ± 57.5 pmol/L for ATP7B 887 (range, 136.4–447.0 pmol/L; 5^{th}–95^{th} percentile range, 165.0–359.6 pmol/L) and 203.0 ± 48.9 for ATP7B 1056 (range, 88.2–381.3 pmol/L; 5^{th}–95^{th} percentile range, 129.7–287.3 pmol/L). These cut-offs were set at –2.5 (114.0 pmol/L) and –2.5 SD (80.8 pmol/L) below the mean normal concentration for ATP7B 887 and ATP7B 1056, respectively.

Analytical Performance

The analytical figures of merit for immuno-SRM quantification of ATP7B peptides are given in Table 1. The LLODs of ATP7B quantification were determined to be 3.81 pmol/L and 2.17 pmol/L for ATP7B 1056 and ATP7B 887,

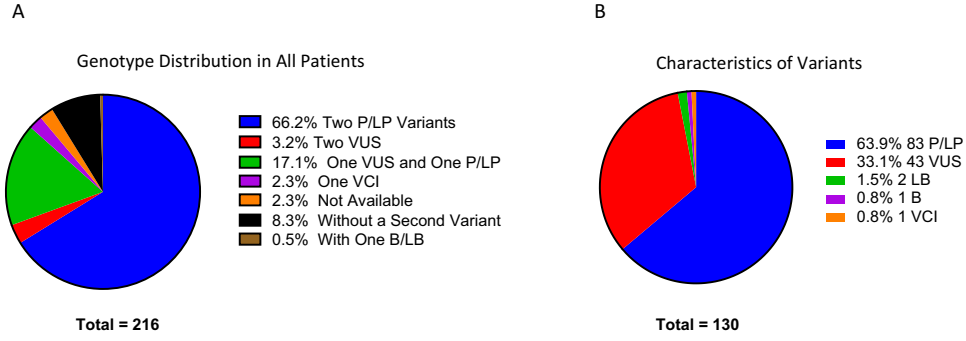


Figure 1. Patient cohort characteristics. Analysis of the genotypes of patients (*A*) and the characteristics of the variants present (*B*) show the diversity of variants and variant combinations present.

respectively. LLOQs were determined to be 71.43 pmol/L and 7.14 pmol/L. The intra-assay CVs were 12.9% and 11.0% for ATP7B 1056 and ATP7B 887. The inter-assay CVs were 15.3% and 13.0%, respectively.

ATP7B Concentration Measurements and Primary Diagnostic Performance

Signature peptide levels in patient DBS were below cut-off in 195 of 216 (90.3%) of samples for both ATP7B 1056 and ATP7B 887 (Figure 2*A* and *B*). There were 17 WD patients (7.9%) that had ATP7B level above the cut-off for both ATP7B peptides. There were 2 WD patients had only ATP7B 887 and 2 WD patients had only ATP7B 1056 levels above the cut-off. In all, 199 of 216 patients (92.1%) had at least 1 peptide below cut-off. In WD carriers, 8 of 48 (16.7%) and 4 of 48 (8.3%) samples were below diagnostic cut-offs for ATP7B 1056 and ATP7B 887, respectively. These patient samples generate potential false positives.

As a primary diagnostic, receiver operating characteristics curve analysis (Figure 2*C* and *D*) constructed from this DBS sample cohort found that both ATP7B 1056 and ATP7B 887 peptide analysis have an area under the curve of 0.98 (ATP7B 1056 [SE = 0.006; 95% confidence interval, 0.97–0.99; $P < .0001$] and ATP7B 887 [SE = 0.007; 95% confidence interval, 0.96–0.99; $P < .0001$]). ATP7B 887 analysis was found to have a sensitivity of 91.2%, specificity of 98.1%, positive predictive value of 98.0%, and a negative predictive value of 91.5%. ATP7B 1056 showed positive predictive value of 96.1% and negative predictive value of 91.3% (Table 1).

Effects of Common Variants

In the cohort of 216 patients with WD, a total of 130 variants were identified (Supplementary Table 6). Many common pathogenic variants, including p.H1069Q (allele frequency [AF], 0.103%), p.R778L (AF, 0.013%), p.M645R (AF, 0.047%), and p.E1064A (AF, 0.015%), were associated with either an undetectable or significantly reduced level of ATP7B (Figure 3*A*–*D*).

Variants Leading to Potential False Negatives

In this cohort, 17 of 216 patients (7.9%) have ATP7B concentrations above the cut-off for both signature peptides. The genetic information is summarized in Table 2. In these 17 WD patients, 13 variants were commonly involved and could contribute to normal levels of ATP7B. According to gnomAD (https://gnomad.broadinstitute.org), these variants are rare, with an AF <0.0089%; the remaining variant, p.M665I, has a VCI designation. Of note, the 4 variants above (p.R616W, p.G710S p.M769V, and p.R969Q) have been reported to show the ATP7B protein distribution similar to wild-type (WT) in an in vitro study (Figure 3*E* and *F*).[24]

ATP7B Analysis and Variant Pathogenicity

Of the 216 WD patients, 211 had genetic test results available (Figure 2*E*). One hundred and forty-three were genetically confirmed to be WD patients by being compound heterozygous or homozygous for known pathogenic or likely pathogenic mutations (Figure 1*A*). One hundred and thirty of these patients (91.0%) had concentrations of at least 1 signature peptide below established cut-offs (Figure 4*D*). Alternatively, 68 patients had ambiguous genetic test results preventing straightforward genetic identification. Sixty-four (94%) of these patients were deficient in ATP7B by peptide analysis (Figure 2*E*). Seven patients were compound heterozygous or homozygous for 2 VUS. All samples (100%) contained significant reductions in ATP7B peptides (Table 2). Thirty-seven patients were compound heterozygous for 1 VUS and 1 known pathogenic or likely pathogenic variant (Table 2, Figure 4*E*). ATP7B concentrations were below cut-off in 35 of 37 (94.6%) of these cases. One VCI, p.M665I, was found in 5 patients. In this case, 3 patients had peptide concentrations below established cut-offs and the remaining 2 were compound heterozygous p.G710S, known to cause false negatives. Three patients had known benign or likely benign in combination with known pathogenic or likely pathogenic mutations. Two are likely carriers with normal ATP7B, but 1 patient with a likely benign mutation in combination with a known pathogenic mutation had nondetectable ATP7B, indicating possible misannotation (#64). Finally, in 18 WD cases that have only 1 variant with no second mutation detected, their ATP7B peptide levels were all (100% of samples) reduced below the cut-off (Table 2, Figure 4*F*).

ATP7B Analysis and Ceruloplasmin Concentration

The 200 patients for whom Cp values were provided were stratified into the following 3 subgroups: 107 patients with Cp <10 mg/dL, 77 patients with Cp between 10 and 20 mg/dL, and 16 patients with Cp >20 mg/dL (Figure 4*A*–*C*). Within these groups, 101 of 107 (94.3%) with Cp <10 mg/dL, 70 of 77 (90.9%) with Cp between 10 and 20 mg/dL, and 14 of 16 (87.5%) with Cp >20 mg/dL had DBS ATP7B peptide concentrations below diagnostic cut-offs.

ATP7B Analysis and Other Clinical Indications

Where possible, clinical information including age, Leipzig scores, liver copper content, presence of KF rings, initial presentation, and presence of cirrhosis are presented (Supplementary Table 3). The exact number of treated patients and their regimens is unknown. No differences in ATP7B concentration were found among the patients based on any of these factors. Fifty-nine patients had hepatic copper measurements available (Figure 4*G*–*I*). Fifty-one patients (86.4%) had elevated liver copper (>250 μg/g), 46 of them (90.2%) had deficient ATP7B levels. Eight patient had liver copper that ranged from 25 to 248 μg/g, 7 of them with deficient ATP7B levels. Of note, 3 samples were received with suspicion of WD by elevated liver copper, but with no ATP7B variants identified, indicating they are not WD patients. As expected, these samples had normal ATP7B concentrations and were not contained within the final 216 WD patient cohort.

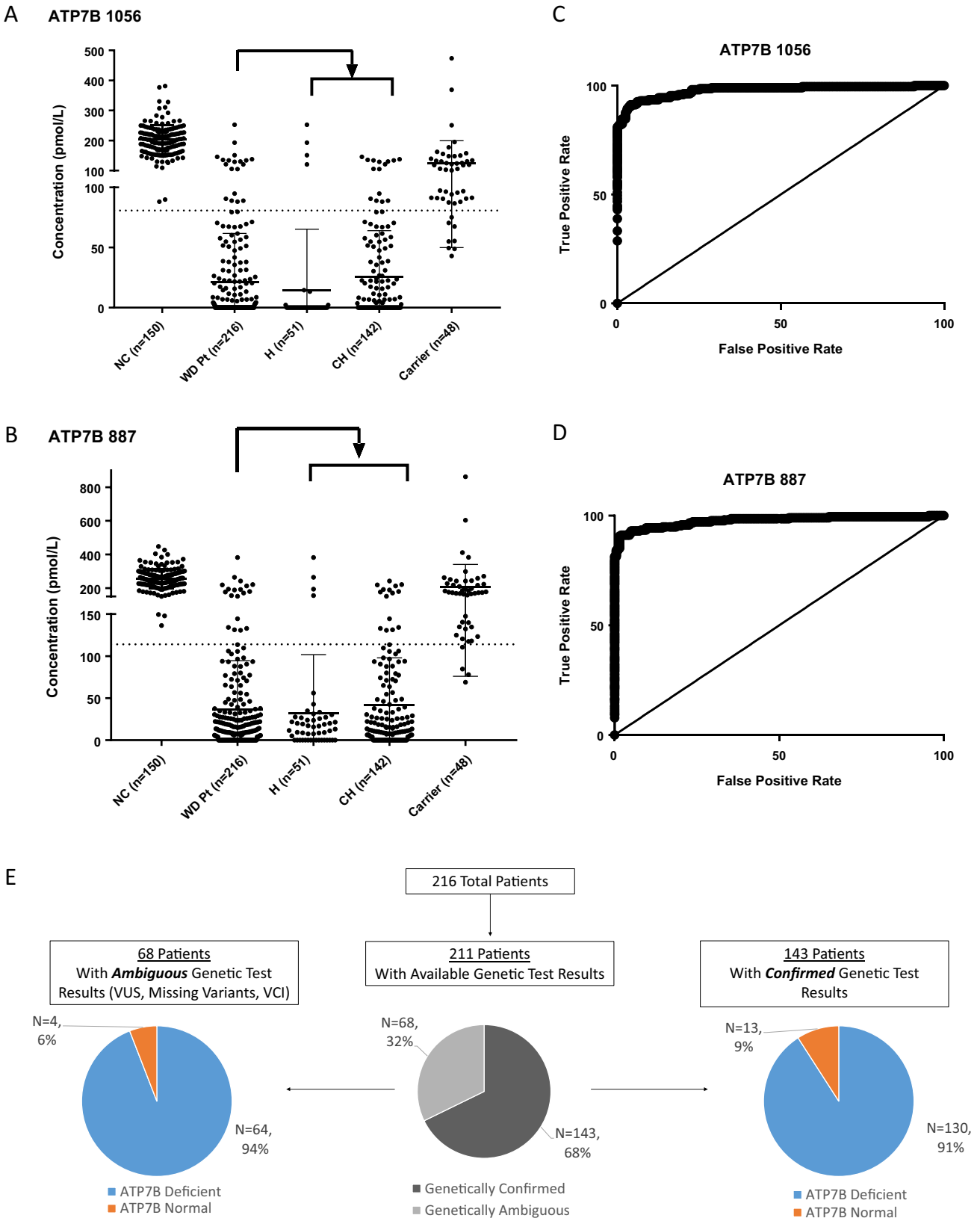


Figure 2. Diagnostic performance of ATP7B peptide analysis. Comparison of ATP7B peptide measurements for ATP7B 1056 (*A*) and ATP7B 887 (*B*) peptides in normal control patients (NC), patients, homozygotes (H), compound heterozygotes (CH), and carriers. *Dotted lines* represent diagnostic cut-offs for each peptide. Receiver operating characteristics curves show the diagnostic performance of ATP7B 1056 (*C*) and ATP7B 887 (*D*). WD patients with genetic test results are readily identified even in subgroups where genetic results are ambiguous (*E*).

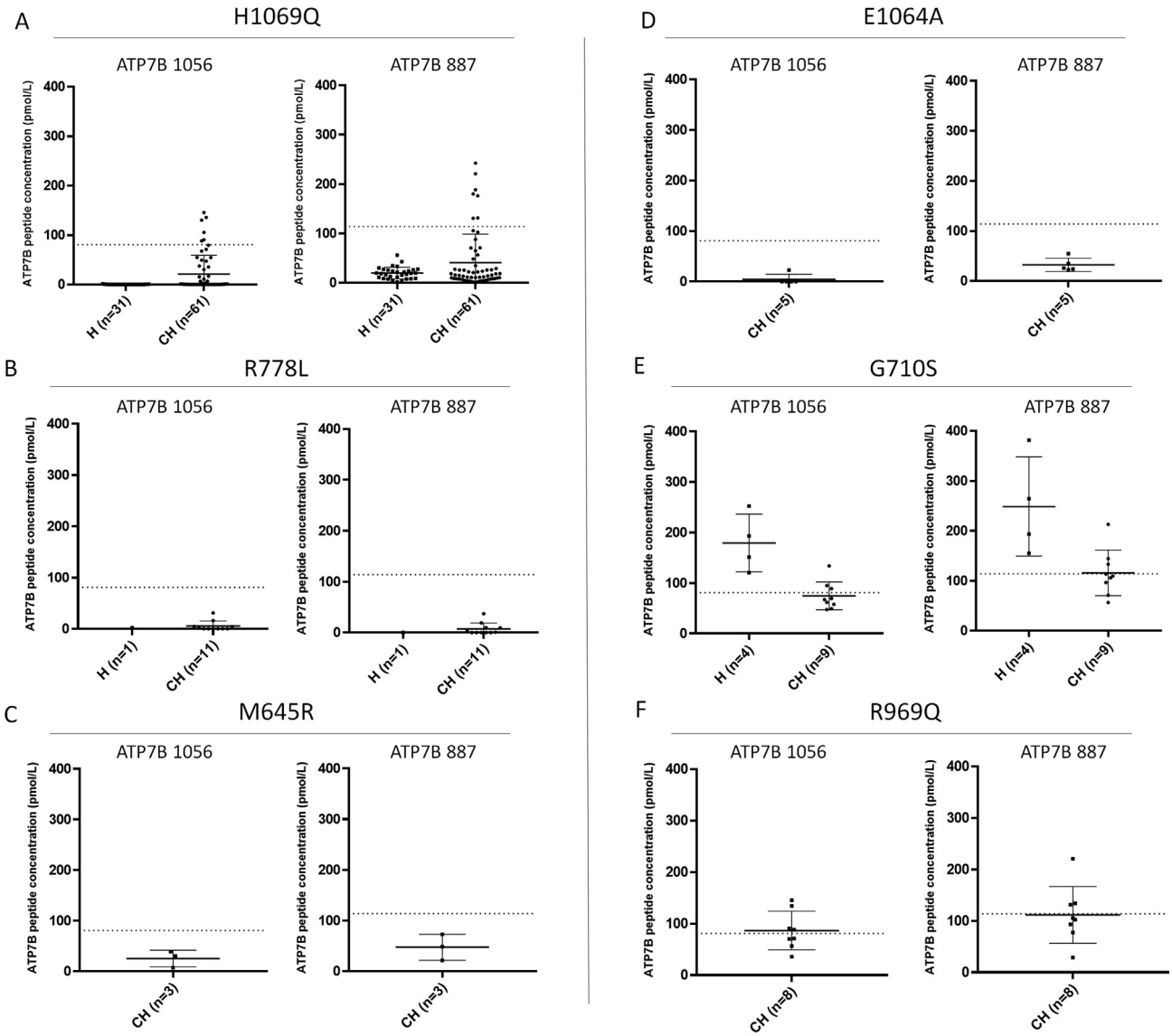


Figure 3. ATP7B peptide concentrations in patients with common variants are often reduced and variants causing false negatives are rare. Patients homozygous and heterozygous for p.H1069Q (*A*), R778L (*B*), M645R (*C*), and E1064A (*D*) are largely reduced. False negatives within the patient cohort are possible with the presence of specific variants, including G710S (*E*) and R969Q (*F*). Patients can have variable peptide concentrations depending on the second variant. CH, compound heterozygote; H, homozygote. *Dotted lines* represent peptide diagnostic cut-offs.

Discussion

This is the first large cohort study directly measuring ATP7B from DBS of WD patients with diverse genetic backgrounds. It showed ATP7B peptide concentrations have a high diagnostic potential. This test is a novel approach to WD testing and screening with a high sensitivity and specificity. It can be applied successfully in patients who do not present with clear clinical and laboratory criteria for WD as a first "second"-line test and expand the clinician's ability to noninvasively diagnose WD by reducing the need for liver biopsy. As the assay measures the ATP7B peptides produced in peripheral blood, liver synthetic dysfunction would have little or no effect on the concentration of ATP7B peptides in DBS, which is another advantage of the assay. In suspected cases, no single test is diagnostic, and a combination of laboratory tests and clinical investigation is required to establish the diagnosis. These include ophthalmologic testing for KF rings, Cp measurement, 24-hour urine copper measurement, liver biopsy to determine copper content, and *ATP7B* gene sequencing. In patients with hepatic WD, KF rings may be absent and Cp in the low to normal range can contribute to diagnostic ambiguity.

Genetic sequencing can give a definitive diagnosis when 2 known pathogenic variants are found. However, many mutations found in the *ATP7B* gene are VUS, VCI, or extremely rare. Now, more than 1300 variants in the ATP7B gene are listed in Varsome (varsome.com)[25]; 649 of them were pathogenic or likely pathogenic, and 692 of them were classified as VUS. Variant interpretation remains a challenge

Table 2. Selected Patients From Cohort

Patient/ gender/age	Variant 1	Annotation 1	Variant 2	Annotation 2	ATP7B 1056 (pmol/L)	ATP7B 887 pmol/L	CPL (mg/dL)	Leipzig score	Liver Copper (ug/g Tissue)	KF Ring	Presentation
False negative (n=17)											
110/F/17	p.G710A	Path	p.G710S	Path	88.9	133.0	14	7	-	NP	-
10/M/34	p.G710S	Path	p.G710S	Path	120.4	155.3	<9	8	-	Y	N
11/M/17	p.G710S	Path	p.G710S	Path	193.2	264.3	12	10	-	Y	B*
12/F/44*	p.G710S	Path	p.G710S	Path	151.1	193.6	9.1	11	164	Y	N
13/M/19	p.G710S	Path	p.G710S	Path	252.4	381.7	20.7	8	1243	Y	B
111/M/47	p.M665I	Conflicting	p.G710S	Path	133.9	213.0	15.5	7	324	NP	H
112/M/NA	p.M665I	Conflicting	p.G710S	Path	94.9	144.4	NA	6	324	NP	H
140/F/29	p.R616Q	Path	p.L1305P	Path	105.5	151.7	2	8	-	Y	B
149/F/17	p.H1069Q	Path	p.M769V	Likely Path	130.2	180.1	10	8	2047	NP	H
150/F/31	p.N41S /p.I1021V	Likely Path	p.M996T	Likely Path	129.1	218.6	4	6	>250	NP	H
153/F/37	p.H1069Q	Path	p.P1273L	Path	105.3	188.5	8	10	-	Y	N
154/F/18	p.M769H-fs	Path	p.P1273L	Path	121.4	170.4	5	10	-	Y	N
170/M/NA	p.H1069Q	Path	p.R969Q	Path	90.5	131.4	NA	4	-	-	-
124/M/17	p.Q7D-fs*14	VUS	p.H1069Q	Path	135.8	242.5	19	5	-	NP	H
171/F/9	p.H1069Q	Path	p.R969Q	Path	145.6	220.9	16	5	-	NP	H
176/M/NA	p.R616W	Path	p.R969Q	Path	134.2	134.2	25	7	-	NP	H
183/F/31	p.T977M	Path	p.T991A	VUS	137.9	191.7	12	3	-	Y	H
Patients with one pathogenic/likely pathogenic variant in combination with one VUS (n=37)											
124/M/17	p.H1069Q	Path	p.Q7D-fs*14	VUS	135.8	242.5	19	5	-	NP	H
125/M/9	p.H1069Q	Path	p.Q7D-fs*14	VUS	79.4	176.3	16	5	-	NP	H
131/F/13	p.H1069Q	Path	p.I1007T-fs	VUS	ND	5.4	14	5	-	Y	H
55/M/59	p.H1069Q	Path	arr[GRCh37] 13q14.3 (52541594_52548863)x1	VUS	ND	16.1	13	5	-	Y	H
87/F/19	p.H1069Q	Path	p.D1447G-fs	VUS	67.5	130.7	13	6	-	NP	H
159/M/40	p.R827W	Likely Path	p.R1320T	VUS	58.7	93.4	13	7	-	NP	N
133/M/18	p.H1069Q	Path	p.K1028S-fs	VUS	ND	3.9	12	7	842	NP	H*
183/F/31	p.T977M	Path	p.T991A	VUS	137.9	191.7	12	3	-	Y	H
164/F/NA	p.H1069Q	Path	p.R778P	VUS	ND	ND	11	9	1332	NP	H

Table 2. Continued

Patient/ gender/age	Variant 1	Annotation 1	Variant 2	Annotation 2	ATP7B 1056 (pmol/L)	ATP7B 887 pmol/L	CPL (mg/dL)	Leipzig score	Liver Copper (ug/g Tissue)	KF Ring	Presentation
118/M/6	p.H1069Q	Path	p.G1011X	VUS	ND	10.0	10	6	-	NP	H
157/F/8	p.R1041W	Likely Path	p.D765Y	VUS	6.8	20.7	10	8	1211	NP	H
163/M/2 mo	p.R778L	Path	p.V1106I	VUS	4.0	9.7	10	-	-	NP	A
182/M/40	p.T850I	Likely Path	p.G515S	VUS	11.6	13.4	10	6	-	Y	B
67/F/17	c.2865+1G>A	Path	p.Ser135*	VUS	ND	ND	<13	9	-	NP	H
167/F/19	p.H1069Q	Path	p.R919L	VUS	3.6	22.1	9	10	-	Y	N
95/M/53	p.H1069Q	Path	p.F1343dup	VUS	ND	ND	9	3	-	Y	H
108/M/15	p.H1069Q	Path	p.G1341E	VUS	ND	10.4	6.7	8	-	Y	H
181/F/41	p.G1266R	Path	p.T807I	VUS	8.3	5.9	6	9	291	NP	H
193/F/15	p.S1365C-fs*12	Path	p.Y743I-fs*19	VUS	ND	ND	<6	7	-	NP	H
80/M/40	p.A874V	Path	c.2299InsC	VUS	6.0	10.5	5.5	10	1575	Y	H*
120/F/13	p.H1069Q	Path	p.Lys269*	VUS	ND	ND	5	5	923	NP	H
142/M/39	p.T977M	Path	p.L1350P	VUS	22.6	32.6	5	10	-	Y	B
76/M/5	p.A1018V	Path	p.E458X	VUS	26.4	47.5	4.1	8	793	NP	H
141/F/46	p.H1069Q	Path	p.L1333P	VUS	ND	6.3	4	7	-	Y	H
184/M/10	p.A874V	Path	p.V1106I	VUS	20.1	15.5	4	-	-	-	-
119/M/38	p.H1069Q	Path	p.K844E-fs*10	VUS	ND	8.7	<4	4	-	NP	N
79/M/59	p.L1088X	Likely Path	p.A1135Q-fs*13	VUS	ND	ND	<4	6	-	Y	H
165/M/15	p.R778W	Path	p.K35N-fs*6	VUS	21.8	25.1	<3	4	-	NP	H
143/F/26	p.R778L	Path	p.L770L	VUS	ND	ND	<3	9	-	Y	H
69/F/16	p.T1029I	Path	c.3060+5G>C	VUS	2.8	ND	<3	8	-	Y	H
74/F/12	p.H1069Q	Path	IVS19-1C>G	VUS	ND	13.5	3	9	-	Y	N
188/F/8	p.M645R	Path	p.V997-fs	VUS	38.7	72.4	2.4	8	>250	NP	H*
102/M/9	p.G1341D	Path	p.F1026F-fs	VUS	ND	ND	2	10	-	Y	N
134/F/10	p.M769H-fs	Path	p.K1028S-fs	VUS	ND	ND	2	10	-	Y	N
72/F/24	p.G710S	Path	c.3400delC	VUS	57.7	96.6	0.6	12	448	Y	B
68/M/11	c.1708-1g>c	Likely Path	c.2866-3c>g	VUS	22.4	37.7	NA	6	-	Y	H
88/F/8	p.M769H-fs	Path	p.D1460Y	VUS	20.3	39.3	low	6	-	NP	H

Table 2. Continued

Patient/ gender/age	Variant 1	Annotation 1	Variant 2	Annotation 2	ATP7B 1056 (pmol/L)	ATP7B 887 pmol/L	CPL (mg/dL)	Leipzig score	Liver Copper (ug/g Tissue)	KF Ring	Presentation
Patients with two VUS's (n=7)											
86/F/68	p.E332K	VUS	p.D1047V	VUS	51.7	79.3	27	6	26	NP	B
3/M/18	p.G1335E	VUS	p.G1335E	VUS	ND	ND	<2	-	-	YP	N
9/F/15	p.G1341E	VUS	p.G1341E	VUS	2.2	ND	1.4	6	-	NP	H
84/M/NA	p.I1336V	VUS	p.C709T	VUS	10.5	15.5	10	8	552	NP	H
85/F/NA	p.I1336V	VUS	p.C709T	VUS	ND	6.3	13	7	900	NP	H
186/M/NA	p.S932L	VUS	p.V1364V-fs	VUS	ND	ND	10	9	-	NP	B
130/M/20	p.T59H-fs*19	VUS	p.H1247Q	VUS	23.6	27.2	<4	9	54	Y	N
Patients with only one variant found (no 2nd variant detected) (n=18)											
208/M/17	c.2299delC	VUS	Unknown	-	ND	ND	<2	-	-	NP	H
209/M/19	c.2299delC	VUS	Unknown	-	ND	ND	<2	-	502	NP	N
195/F/43	c.51+4a>t	Path	Unknown	-	25.6	37.6	4	8	-	Y	N
196/F/28	p.A1049A-fs	VUS	Unknown	-	2.2	9.1	3	7	-	Y	N
194/M/50	p.D765N	Path	Unknown	-	20.4	21.4	<4	8	-	Y	N
197/M/22	p.G1176R	Path	Unknown	-	3.6	7.3	3	5	-	NP	H
199/M/17	p.H1069Q	Path	Unknown	-	8.6	32.9	<10	5	-	NP	N
201/M/14	p.H1069Q	Path	Unknown	-	31.4	29.0	3.4	-	-	NP	N
198/F/NA	p.H1069Q	Path	Unknown	-	ND	5.0	12	6	-	NP	H
200/M/14	p.H1069Q	Path	Unknown	-	ND	6.7	13	4	-	Y	H
211/F/22	p.H1069Q	Path	Unknown	-	ND	7.9	2	5	-	NP	N
210/M/NA	p.L1305P	Path	Unknown	-	ND	6.0	NA	3	-	-	-
202/F/16	p.M769H-fs	Path	Unknown	-	ND	ND	13.8	6	525	Y	H
203/M/37	p.R1319X	Path	Unknown	-	3.0	5.2	<10	9	1042	Y	B
204/M/12	p.R778L	Path	Unknown	-	ND	9.5	<3.0	5	-	NP	H
205/M/25	p.T1220M	Likely Path	Unknown	-	67.7	77.0	<10	4	191	NP	B
207/F/18	p.W779X	Path	Unknown	-	38.0	65.6	15.3	6	258	NP	H
206/M/45	p.W779X	Path	Unknown	-	ND	ND	<-4	6	-	Y	H

*, cirrhosis; A, asymptomatic; B, both hepatic and neurologic; H, hepatic; N, neurologic; NA, not available; ND, not detected; NP, not present; VUS, variant of uncertain significance; Y, present.

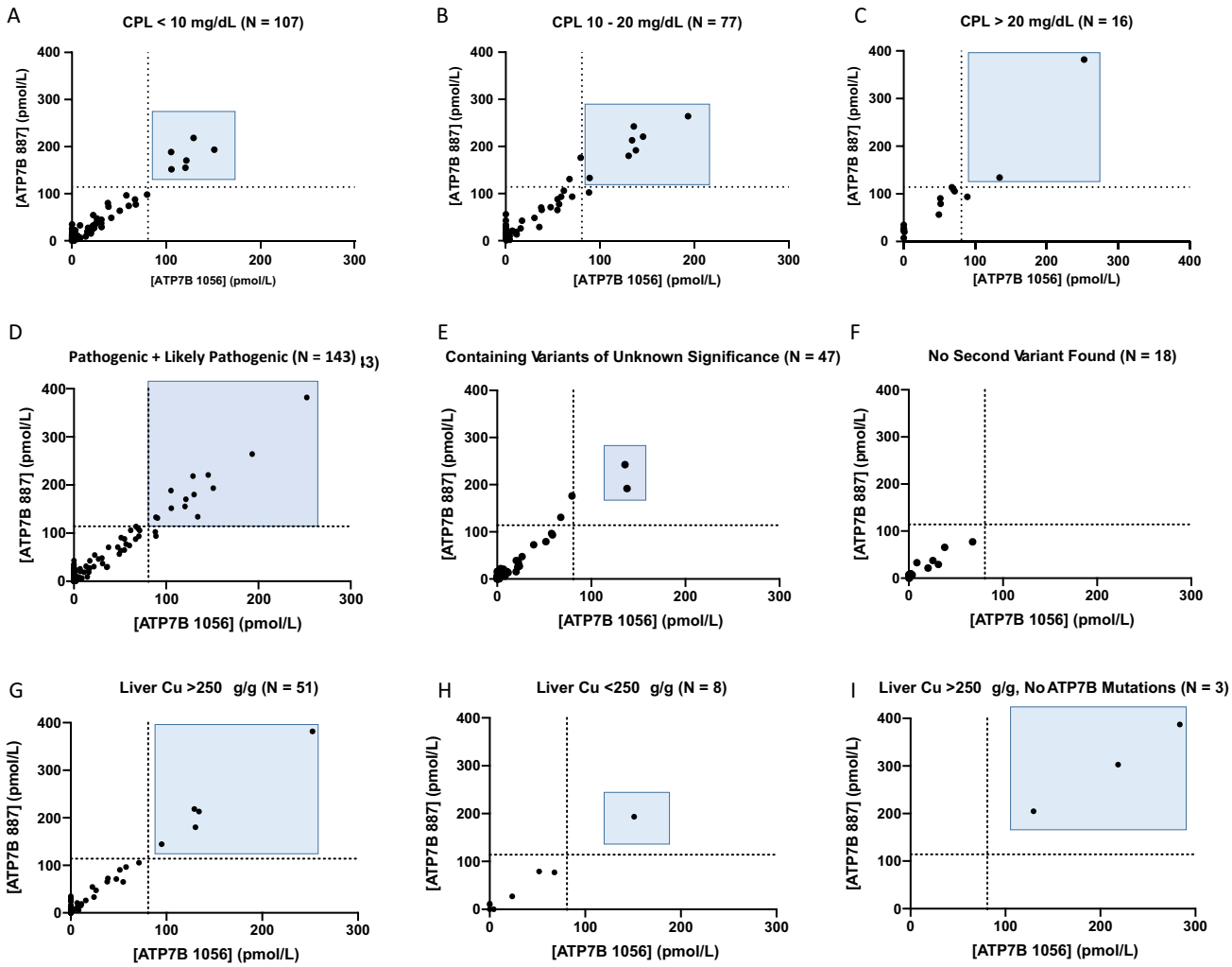
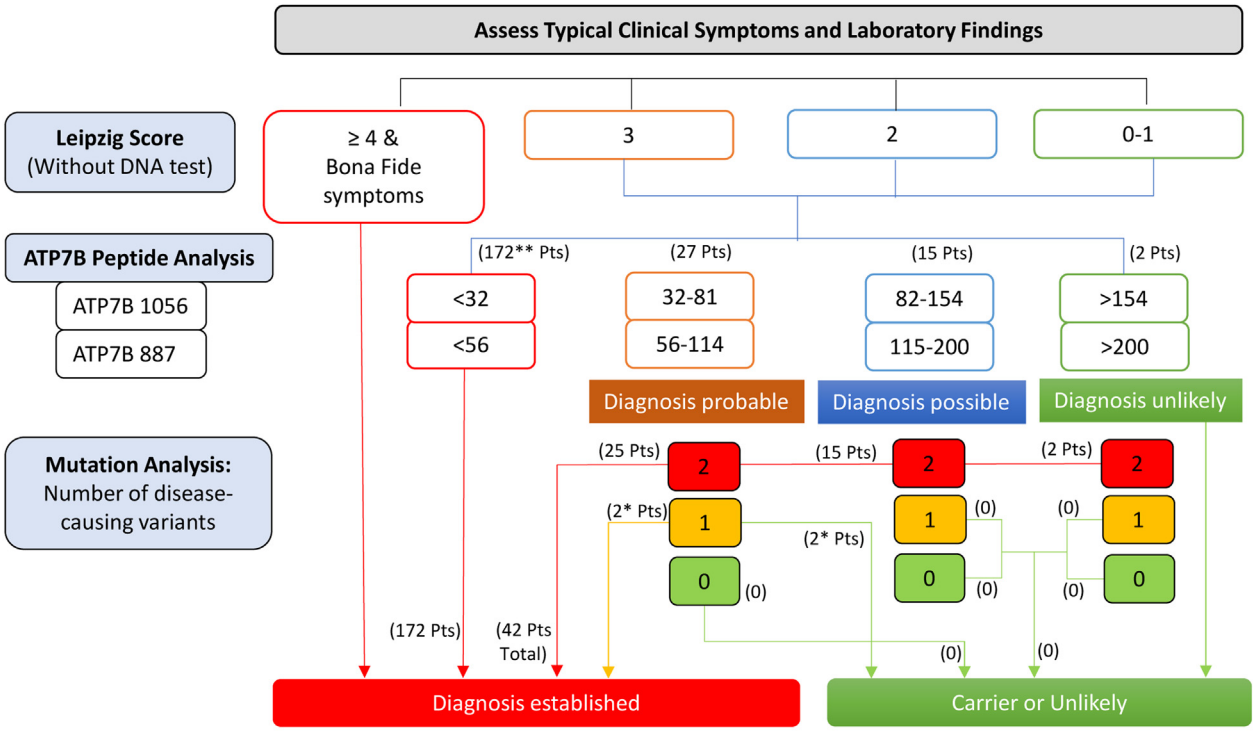


Figure 4. ATP7B peptide concentration analysis can provide clear results where Cp results and genetic analysis are ambiguous. Patients with significantly (*A*), moderately (*B*), and normal (*C*) Cp were readily identified. ATP7B concentrations are reduced regardless of variant status, including in patients with 2 pathogenic or likely pathogenic variants (*D*), at least 1 VUS (*E*), or where no second variant was found (*F*). *Dotted lines* represent peptide diagnostic cut-offs. Patients with liver copper above (*G*) or below 250 μg/g (*H*) are shown. In 3 samples from non-WD patients with elevated liver copper, ATP7B concentrations are normal (*I*).

for clinical laboratories.[26,27] The recommended Leipzig score for diagnosis of WD assigns numerical values to the number of disease-causing mutations to give probable WD diagnoses. The definition of "disease"-causing variant is based on a variety of databases, which can give conflicting answers.[28]

We hypothesized that direct measurement of ATP7B could identify WD patients as a majority of pathogenic mutations often result in protein misfolding, absence of decay of messenger RNA and enhanced degradation. To explore this goal, 2 WT ATP7B peptides were chosen for antipeptide antibody generation. Several factors influence peptide selection, as they must be unique to ATP7B, detectable by mass spectrometry, and elicit specific antibodies for isolation. ATP7B 1056 contains the most common WD-causing mutation, p.H1069Q. If the patient is homozygous for this mutation, WT ATP7B 1056 will not be found because the WT sequence is not present. Having a second peptide, ATP7B 887, builds a redundancy into the assay to ensure accurate performance.

As a primary diagnostic test, quantification of ATP7B from DBS effectively identified WD patients (Figure 2*A* and *B*, Table 1). Reduction of ATP7B concentrations below diagnostic cut-offs for at least 1 ATP7B peptide was evident in 92.1% of WD patients. Because the cut-offs set are based on the number of normal control patients analyzed, receiver operating characteristics curves were constructed showing ATP7B analysis to be highly sensitive and specific for diagnosis. The calculated area under the curve for the dataset is 0.98 regardless of peptide quantified (Figure 2*C* and *D*). Here, 211 patients had available genetic results (Figure 2*E*). WD is genetically confirmed in 143 patients with 2 evident pathogenic or likely pathogenic variants. This leaves 68 patients (32%) without a straightforward genetic diagnosis. Within the genetically confirmed subgroup, 91% were ATP7B-deficient, agreeing with sequencing results.



*# 205 and #207 fall under both outputs: "Diagnosis established" and "Carrier or Unlikely".

**includes 156 patients with 2 variants and 16 patients with 1 variant

ATP7B	Average	SD	-3.5	-2.5	-2	-1
1056.0	203.0	48.9	31.9	80.8	105.2	154.1
887.0	257.7	57.5	56.5	114.0	142.7	200.2

Figure 5. Proposed Wilson disease diagnostic algorithm.

More importantly, 94% of patients without clear genetic results (containing VUS, VCI, or missing variants on sequencing) were ATP7B-deficient. ATP7B peptide concentration analysis can be highly useful in these patients.

ATP7B peptide concentrations were measured in patient samples collected up to 11 years prior, to study whether ATP7B degradation impacts stored samples (Supplementary Table 7). Individuals with reduced ATP7B in freshly or recently collected samples had reduced concentrations across time. This suggests that older samples are not being identified as patients due to ATP7B degradation. No diagnosis in this group changed due to date of sampling. This includes 1 normal control patient with 3 separate samples taken over 6 months. All measured concentrations clearly identified this individual as normal and the CV for the measurements in these samples was approximately 11.4% and 12.4% for ATP7B 1056 and 887, respectively.

Certain variants are highly prevalent in the population and are more commonly seen in clinic. These represent important test cases for the discriminatory ability of ATP7B quantification by immuno-SRM. These variants had predominantly low or undetectable peptide concentrations, supporting the hypothesis that protein levels are reduced in vivo. Four of these high-frequency variants (H1069Q, R778L, E1064A, and M645R) have significantly reduced ATP7B concentrations in both homozygotes and compound heterozygotes (Figure 3*A*–*D*). Patients with these common mutations and, therefore, a significant percentage of patients overall, should be readily discriminated by the use of immuno-SRM as an index test.

Direct measurement of ATP7B peptides means that disease-causing mutations that affect protein activity but not protein concentration will generate false-negative results. In each of the 17 false-negative patients, both ATP7B 887 and ATP7B 1056 were above diagnostic cut-offs, indicating significant production of nonfunctional protein. Identifying these variants and their frequency will be important in interpreting immuno-SRM in the context of other clinical results. If ATP7B concentrations in patient DBS are in the established normal ranges but clinical suspicion for WD is high, continued patient workup is obligatory to confirm the diagnosis. Several such variants were found here and ATP7B levels often depended on the nature of the second variant (Table 2). The variants found in these patients have AFs ranging from unknown to 0.0089% and would therefore represent a small percentage of the overall patient population.

The most common variants with ATP7B levels above the cut-off are p.G710S and p.R969Q (Table 2; Figure 3*E* and *F*). There were 13 WD patients carrying the p.G710S variant and 8 patients with p.R969Q. Seven p.G710S patients had normal concentrations of ATP7B peptides when they were

either homozygous for p.G710S or compound heterozygous with p.G710A and p.M665I. Three p.R969Q patients were false negatives. In 2 of these cases, p.R969Q is in combination with p.H1069Q, which is shown to significantly reduce ATP7B concentrations. p.G710S is a variant associated with severe liver disease, 2 patients required an emergency liver transplantation due to fulminant hepatic failure.[29] These variants are known pathogenic by causing significantly impaired copper transport activity while maintaining normal ATP7B trafficking and phosphorylation in vitro.[24,30,31] Patients with these variants could be missed by immuno-SRM when in combination with variants producing significant ATP7B. When in combination with a variant severely affecting ATP7B concentrations, as in the p.H1069Q cases, ATP7B levels may give false negatives (Table 2). The mechanisms of variant interaction in vivo are currently under study. Diagnostic potential will therefore depend on the specific genotype of these patients. However, knowing that these variants have the potential to generate normal levels of ATP7B will be useful when evaluating genetic analysis after ATP7B measurement.

Like the method-dependent cut-offs used for Cp, 24-hour urinary copper[32] and hepatic copper for diagnosing WD,[33,34] measurement of ATP7B peptides require validation in the population in which it will be used. This will aid in generating clearly defined cut-offs. A true description of the final diagnostic performance of ATP7B concentration measurement will come with conducting a large cohort validation or pilot study, such as newborn screening.

Cp is not useful as a screening test for WD,[9] but levels of Cp <10 mg/dL are regarded as useful for diagnosis (score 2 in Leipzig score) of WD. A recent Chinese study found that Cp levels <12 mg/dL are strongly indicative of a diagnosis of WD.[32] Here, 200 patients had Cp values available, among them 77 (38.5%) had moderate Cp values of 10–20 mg/dL and an additional 16 (8%) had normal Cp levels >20 mg/dL; 92.9% of patients with Cp <10 mg/dL had ATP7B concentrations below diagnostic cut-offs (Figure 4*A*–*C*). When Cp levels were moderately reduced, 91.6% of patients would be identified by ATP7B analysis. Even in those with normal Cp values ATP7B identified 87.5% of cases. Measurement of DBS ATP7B can provide clarity when Cp levels are ambiguous. This is likely because ATP7B is not a measurement of secondary disease effects. Cp and copper measurements are often confounded by external processes or disease influences, including liver cirrhosis and malnutrition. As Cp is an acute-phase reactant possessing ferroxidase activity, the concentration can be elevated by acute inflammation. A prospective study on serum Cp as a screening test for WD in patients referred with liver disease reported a positive predictive value of only 6%.[9] Here, immuno-SRM analysis of ATP7B clearly outperforms Cp measurement.

Clinical information, including age, Leipzig scores, liver copper content, presence of KF rings, initial presentation, and presence of cirrhosis, was obtained when possible (Supplementary Table 3). No significant differences in ATP7B concentration were found based on age (pediatric vs adult), presence of KF ring, presentation (hepatic, neurologic, both hepatic and neurologic, or asymptomatic), or presence of cirrhosis. The mean ± SD ATP7B 887 peptide concentrations in hepatic and neurologic presentation were 33.8 ± 51.7 pmol/L and 38.2 ± 57.1 pmol/L, respectively. This appears aligned with previous observations in a large cohort showing the absence of any phenotype–genotype correlation regarding initial symptomatic manifestation.[35] Fifty-nine patients had hepatic copper measurements and 86.4% of these had elevated liver copper >250 μg/g. This includes 5 with normal ATP7B levels, 3 of them with p.G710S variant, and supports their diagnosis as WD patients despite negative immuno-SRM results (Figure 4*G*–*I*). Of interest, 3 samples were received with suspicion of WD by elevated liver copper but with no ATP7B variants identified. These samples had normal ATP7B concentrations, supporting their status as non-WD patients and providing an example of immuno-SRM analysis in ruling out WD (Figure 4*I*). Finally, it is unknown how many patients were being treated and what their treatment regimens were. Current WD therapies are focused on copper depletion and not restoration of ATP7B protein. As such, treatments would not affect the measured levels of ATP7B.

Examples From Dataset Where Diagnosis Is Simplified

The ability of ATP7B measurement to clarify case results extends to ambiguities in genetic analysis and contributes significantly to advancing noninvasive clinical diagnosis of WD (Figures 2*E* and 4*D*–*F*). There are 7 cases presented in which patients were compound heterozygous or homozygous for 2 VUS, indicating a lack of strong understanding of how these variants will contribute to phenotype (Table 2). All of these patients had significantly reduced ATP7B peptides levels suggesting affected patient status. Three patients in this group had only moderately reduced Cp between 10 and 20 mg/dL. In 1 case (no. 186), sequencing returned 1 VUS computationally predicted to be pathogenic and 1 (p.V1364V-fs) predicted to be benign. This patient had entirely nondetectable DBS ATP7B, indicating the consequence of this genetic combination is disease-causing. One patient (no. 86) presented an interesting clinical situation in which a VUS was found on sequencing and Cp concentrations were clearly within the normal range (27 mg/dL) (Table 2). Here, ATP7B measurement provided a clear indication that these patients severely lacked ATP7B protein and were very likely affected by WD in a way that genetic analysis, prediction, and Cp measurement could not.

Similarly, there were 37 patients in which a known pathogenic or likely pathogenic variant was found in combination with a VUS (Figure 4*E*, Table 2). Thirty-five (94.6%) of these had reduced ATP7B concentrations of at least 1 peptide, indicating positive identification as a WD patient despite a lack of knowledge of the consequences of their specific mutations. In 14 of these patients, Cp was found to be ≥10 mg/dL. Here, a significant reduction in patient ATP7B is particularly valuable for assigning a clinical designation and treatment course.

Of concern are situations where Sanger sequencing and/or next-generation sequencing identifies only 1 variant. Sequencing analysis can be robust for targeting known variants but can return negative results if disease-causing mutations are small or large deletions, duplications, in the deep intronic or promoter regions, or in poly-A tails. These situations can have a significant impact on patient identification and place greater emphasis on Cp measurements and other WD diagnostics while the genetic basis for disease is not known. In 18 patients from our cohort with high suspicion for WD, only 1 *ATP7B* variant was identified (Figure 4*F*, Table 2). All of them had ATP7B measurement with peptide concentrations below established cut-offs. This includes 4 patients with Cp concentrations between 10 and 20 mg/dL, where a single variant and a moderate Cp reduction would be insufficient to establish the diagnosis. In 3 patients, the single variant detected from sequencing was a VUS causing a further complication in genetic interpretation. ATP7B measurement can provide direct evidence of the consequences of existing variants even if the second variant was not detected from sequencing workflows.

It is notable that some carriers with ATP7B peptide concentrations below cut-off create potential false positives. ATP7B 887 analysis showed 4 of 48 carriers had peptide levels below diagnostic cut-offs. The mechanisms by which a single affected allele reduce ATP7B concentrations are unknown and require study. Polymorphisms affecting protein concentration or factors affecting possible co-translated protein interactions may exist but are currently unknown.

Finally, we propose a new algorithm for WD diagnosis incorporating ATP7B measurement findings for each peptide (Figure 5). Here we define 4 possible patient groups based on ATP7B peptide level: directly established WD (<32 pmol/L for ATP7B 1056 and <56 pmol/L for ATP7B 887); probable WD (ATP7B between group 1 and the diagnostic cut-off); possible WD (ATP7B 1056: between cut-off and 154 pmol/L and ATP7B 887: between cut-off and 200 pmol/L); and unlikely WD (>154 pmol/L for ATP7B 1056 and >200 pmol/L for ATP7B 887). In validating the algorithm on the current retrospectively collected patients, we found that 172 of 216 (79.6%) showed ATP7B within the range of patient group 1, making their diagnosis very highly likely. Applying this method to the remaining cases along with mutation analysis show that all are able to reach WD diagnosis, except for 2 cases in which diagnosis or carrier status cannot be determined. Full validation of this proposed algorithm will be necessary through a large cohort prospective study.

Limitations

The study is limited in that data are predominantly for White patients. Patients from the Indian subcontinent, Africa, and South America may have different genotype distributions and possibly differing ATP7B peptide levels. There is also an unavoidable selection bias due to the analysis of only well-described WD cases. A significant limitation comes from the fact that these samples have been collected in a retrospective manner. It will be important to prospectively analyze a large cohort samples before genetic analysis in future applications. Full validation in a large prospective study will allow for more accurate definition of ATP7B variability in healthy normal samples and diagnostic parameters, including sensitivity, specificity, positive predictive value, and negative predictive value. The current statistical measures of predictive significance might change upon conducting a validating study. Furthermore, very few cases of fulminant WD and hemolysis were investigated. Patients presenting with hemolysis might have differing ATP7B peptide concentrations due to red blood cell lysis. In addition, ATP7B analysis is unable to delineate patient groups based on phenotype, that is, hepatic or neurologic types or disease severity. Finally, only 2 signature ATP7B peptides have been analyzed. Alternative peptide sequences may have different discriminating capability such that study of additional candidate peptides may be helpful.

LC-MS/MS is considered highly specialized technology, although it has been used in clinical laboratories in a wide array of fields, such as toxicology, drug monitoring, and newborn screening. Following the standard validation process in the clinical laboratory, we anticipate the assay can be successfully implemented into clinical practice.

ATP7B peptide analysis identified WD patients in a large majority of cases and reduced ambiguities resulting from genetic analysis and Cp levels. This noninvasive assay can serve as an adjunctive test for the diagnosis of WD and is expected to fundamentally advance the use of proteomic technology for a rapid screening tool, an area that holds great promise but is largely untapped.

Supplementary Material

Note: To access the supplementary material accompanying this article, visit the online version of *Gastroenterology* at www.gastrojournal.org, and at http://dxdoi.org/10.1053/j.gastro.2021.02.052.

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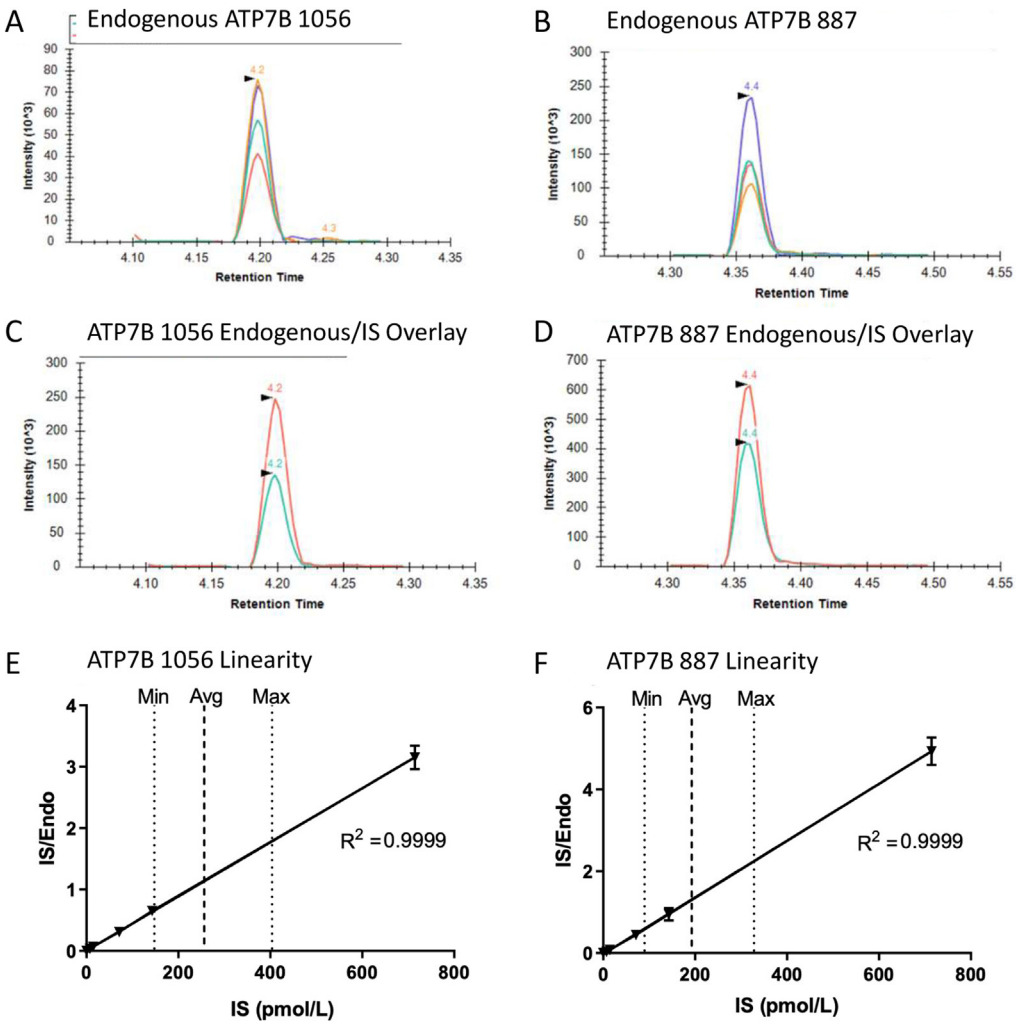

Supplementary Figure 1. Representative chromatograms of ATP7B peptides in MS/MS. (*A*) endogenous ATP7B 887; (*B*) endogenous ATP7B 1056; (*C*) overlay of endogenous and IS ATP7B 887; and (*D*) overlay of endogenous and IS ATP7B 1056. Linearity curves of ATP7B 1056 (*A*) and ATP7B 887 (*B*). *Vertical lines* represent the average (avg), minimum (min), and maximum (max) concentration determined in normal controls.

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Expanding the Diagnostic Toolkit of Wilson Disease with ATP7B Peptides

See "Direct measurement of ATP7B peptides is highly effective in the diagnosis of Wilson disease," by Collins CJ, Yi F, Dayuha R, et al, on page 2367.

The diagnosis of Wilson disease (WD) can be determined by a combination of parameters aiming to detect copper accumulation. In this article from Collins et al,[1] a new approach to the diagnosis of WD is proposed. In current clinical practice, low ceruloplasmin levels are used as a minimal screening test, followed by Kayser–Fleischer ring assessment, 24-hour urine collection, liver biopsy for copper quantification, and brain magnetic resonance imaging for demonstration of copper-related changes in the basal ganglia. Although scoring systems can aid in the diagnostic process,[2] the traditional laboratory tools lack sensitivity and specificity for WD. In particular, the major limitation of ceruloplasmin determination is that most clinical laboratories quantify the enzyme levels, but not its oxidase activity, with consequent determination of erroneous elevated levels owing to quantification of both ceruloplasmin and biologically inactive apoceruloplasmin.[3]

Total serum copper is even less valuable as a screening test because it is influenced by ceruloplasmin level and its measurement methods. Genetic testing with *ATP7B* sequencing, whole-exome sequencing[4] and whole-genome sequencing are considered confirmatory tests and their costs are becoming more approachable. Although *ATP7B* variants are associated with various degrees of functional impairment,[5] their clinical correlate is uncertain. Therefore, the major challenge is the lack of genotype–phenotype correlation,[6] with the additional concern that some gene variants may not be associated with clinical manifestation development. Other diagnostic options, including the radioactive copper incorporation test[7] or the exchangeable copper,[8] are interesting but will likely offer several challenges in their access and execution outside selected academic centers. Therefore, although the diagnosis of WD is frequently achievable with available methods, many cases are trapped in a grey area of diagnostic uncertainty characterized by borderline ceruloplasmin levels, gene variants of unknown significance, and ambiguous clinical presentations. These borderline cases often overlap with common conditions, including fatty liver, autoimmune hepatitis, and movement disorders, and require refined clinical judgment and expertise for the final diagnosis.

Striving for a gold standard and improved diagnostic assessment is important because WD is a treatable disease both in the acute and chronic phases and timely diagnosis can prevent, improve, and even resolve many of the clinical manifestations. Particularly concerning are the neuropsychiatric manifestations, which are debilitating and only responsive after years of anticopper treatment and physical therapy. In addition, new treatment options are on the horizon, including next-generation chelating agents[9] and gene therapy approaches.[10] Medical treatment is often limited by drug toxicity, and unsatisfactory improvement, especially of the severe neuropsychiatric symptoms, is frequent. Liver transplantation can be an effective treatment and is often inevitable in cases of acute liver failure, but chronic immunosuppression is not desirable.[11] Therefore, when the phenotypic characterization of WD improves beyond the traditional definitions of prevalent hepatic and neuropsychiatric involvement,[12,13] the goal will be to tailor and optimize medical treatment to the phenotype or predicted disease course.

In this issue of *Gastroenterology*, Collins et al[1] adopt a new approach to the diagnosis of WD based on the quantification of ATP7B protein concentration derived from measurement of 2 surrogate peptides in patient dried blood

spot samples, as direct evidence of WD diagnosis. Two ATP7B peptides, ATP7B 887 and ATP7B 1056, were selected and measured with specific antibodies. The studied samples derived from pre-existing biorepositories originated from European, North American, and South Korean institutions providing samples from patients with WD (n = 216), obligate *ATP7B* variant carriers (n = 48), and healthy patients (n = 150). Patients with WD carried different *ATP7B* variants, mostly highly prevalent in the population of origin. The age range was 2 months to 73 years, which is important because it is now established that the diagnosis of WD should be considered at any age.

The approach was first to identify the analytical and diagnostic performance of the peptides and identify the diagnostic cutoffs. A large proportion of the studied patients had available genetic test results and 92.1% of the patients with genetically confirmed WD had ≥1 of the peptides below the established minimal cutoffs. Among the 67 patients in whom genetic tests could not establish diagnosis of WD, 63 (94%) had their diagnosis clarified by low peptide levels. Of note, ≤16% of individuals heterozygous for WD had peptide levels below the cutoff, but could also have been WD cases carrying unknown variants. ATP7B 887 analysis demonstrated a sensitivity of 91.2% and specificity of 98.1%. In line with the challenges related to varied WD presentation, a small but sizable percentage of genetically confirmed WD cases presented peptide levels above the cutoffs, therefore representing a border line zone of patients that will likely still be difficult to diagnose or rule out as WD.

Owing to the retrospective nature of the sample collection, available clinical data were not extensive and information about current medical treatments were not detailed. Regardless, the possibility of a new diagnostic test to add to the WD toolkit available to practitioners not familiar with WD, is appealing and ultimately beneficial to patients. This study and the significance of the ATP7B peptides is timely as it positions itself in the middle of an ongoing debate about the full penetrance of WD,[14] the role of extra hepatic ATP7B copper transporter on clinical manifestations,[15] and the significance of ATP7B variants on disease phenotype.

Genetic testing and the knowledge to interpret these data remain incomplete. This study on ATP7B peptides opens clinical and research opportunities aiming to correlate peptide levels with disease severity and phenotype on larger populations. As suggested by the authors, integrating peptide levels into a validated diagnostic scoring system which includes copper metabolism parameters and genetic testing should be a major research focus and could lead to improved phenotypic and prognostic patient characterization. Ultimately, the proposed approach may be helpful to diagnose both adult and pediatric cases of WD.

In summary, expanding the diagnostic toolkit of WD is a priority and this study offers an additional option that could have a role as an initial screening or confirmatory test and will ensure that fewer diagnoses are delayed or missed.

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COVID-19 Gastrointestinal Symptoms and Attenuation of the Immune Response to SARS-CoV-2

See "Intestinal host response to SARS-CoV-2 infection and COVID-19 outcomes in patients with gastrointestinal symptoms," by Livanos AE, Jha D, Cossarini F, et al, on page 2435.

As we eclipse year 1 of the severe acute respiratory syndrome novel coronavirus-2 (SARS-CoV-2) pandemic, our understanding of this virus is only beginning to unfold despite an exponentially growing body of literature. Early descriptions of the novel respiratory illness coronavirus disease 2019 (COVID-19) were quickly followed by reports of a multisystem disease.[1] Gastrointestinal (GI) symptoms are frequently reported in patients with COVID-19, which raised questions of gut infection and the possibility of fecal–oral transmission. Indeed, viral RNA was detected in stool of infected patients by quantitative reverse transcriptase polymerase chain reaction, which frequently remained positive beyond the duration of positivity of their nasopharyngeal samples.[2–4] Multiple groups have reported GI epithelial coexpression of the host receptor ACE2 and TMPRSS2, a protease enabling cell entry through cleavage of the eponymous spike protein.[5–8] In line with the susceptibility of the GI epithelium to SARS-CoV-2 entry, viral RNA by in situ hybridization[9] and viral nucleocapsid protein (NP) by immunofluorescence have been detected in infected patient intestinal biopsies,[2] whereas viral particles have been demonstrated by transmission electron microscopy.[9] Moreover, recent studies using ex vivo enteroid or organoid models convincingly demonstrated that GI epithelium is permissive to SARS-CoV-2 infection.[6,9–11] Notably, human intestinal enteroids inoculated with the virus or nasopharyngeal aspirates from infected patients showed intracellular NP and increasing viral titers over time, indicating productive viral replication within intestinal epithelium.[9–11] Enteroids also exhibited double-stranded RNA, a requisite intermediate produced during the replication of this positive-sense RNA virus.[11] What happens in vivo remains unclear owing to the inherent logistical challenges of studying large cohorts of patients infected with this novel pathogen. Beyond the possibility of GI infection, viral interactions with GI tract tissues and the immune system, and how this influences clinical outcomes in COVID-19 remain largely unknown.

In this issue of *Gastroenterology*, Livanos et al[12] attempt to correlate clinical observations in patients with COVID-19 with immunologic changes within the GI tract on histologic, transcriptomic, and proteomic levels. The authors analyzed endoscopic biopsy specimens from COVID-19–afflicted patients, finding no gross abnormalities when compared with matched controls.[12] Although they found angiotensin-converting enzyme 2 (ACE2) diffusely expressed throughout the GI tract, viral NP was found more prominently in the ileum versus duodenum in the limited number of specimens studied.[12] They attribute this finding to increased MUC2+ goblet cell prevalence in distal small intestine, which colocalized with NP immunostaining, and their transmission electron microscopy images showing viral particles present primarily in exit vesicles of goblet cells.[12] This finding may corroborate findings from bronchial air–liquid interface cultures demonstrating tropism for goblet cells,[13] but were nevertheless surprising given relatively low ACE2 expression in intestinal goblet cell clusters,[5] evidence of goblet cell depletion in an infected patient's colonic biopsies,[9] and data from human intestinal organoids demonstrating enterocytes were susceptible to infection whereas goblet cells were not.[6,11] Livanos et al[12] also find evidence of NP staining in a nongoblet crypt base cell population but do not discern this population as Paneth, stem cells, or another population.[12] This raises additional questions of cellular tropism and host entry determinants because enterocytes in the villus compartment express the highest levels of ACE2.

The authors also performed a mass cytometry immunophenotypic analysis of endoscopic biopsies and blood samples from these patients.[12] They found that specific dendritic cell populations were diminished in the lamina propria of infected patients, possibly supporting some alteration in antigen presentation.[12] Bulk RNA sequencing of these tissues found decreased expression of several inflammatory pathways in the lamina propria and a trend toward the up-regulation of antiviral pathway signatures within the epithelial compartment.[12]

Notably, Vero E6 cells inoculated with homogenized biopsy tissue obtained from patients did not show evidence of productive infection.[12] This finding may simply result from sampling or technical error, but it may also reflect the biology of SARS-CoV-2 in the gut. There remains a striking lack of evidence of infectious virion isolation from the gut,

Management of Wilson Disease Diagnosed in Infancy: An Appraisal of Available Experience to Generate Discussion

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ABSTRACT

Increased access to molecular genetic testing is changing the demographics for diagnosing inherited disorders and imposing new challenges for medical management. Wilson disease (WD), typically diagnosed in older children and adults, can now be detected in utero and in infants (children younger than 24 months, including neonates) via genetic testing. An evidence-based approach to management of these neonates and extremely young children, who are typically asymptomatic, has been hampered by lack of clinical experience. We present a case of an infantile diagnosis of WD, review available experience, and discuss current trends in antenatal genetic testing of parents and fetus that may lead to a very early diagnosis of WD. Based on physiological and nutritional considerations, we propose an algorithmic approach to management of infantile WD as a starting point for further discussion. Future collaboration amongst specialists is essential to identify evidence-based approaches and best practice for managing treatment of infants with genetically diagnosed WD.

Key Words: *ATP7B*, copper, genetics, screening, zinc salts

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What Is Known

- ATP7B molecular genetic testing can identify homozygotes or compound heterozygotes for pathogenic mutations associated with Wilson disease.
- Screening first-degree relatives of patients with Wilson disease is recommended to identify those affected with Wilson disease.
- Zinc therapy is effective in asymptomatic patients with Wilson disease, but few data are available about optimal treatment for infants with genetically diagnosed Wilson disease.

What Is New

- Testing infants born to parents who are known carriers of *ATP7B* pathogenic mutations can identify infantile Wilson disease.
- Breast milk and formula provide approximately >150% of estimated copper and zinc needs, because copper is critical for normal development.
- Treatment with zinc in infantile Wilson disease may prevent development of significant copper accumulation and end-organ damage; however, timing when to start treatment is uncertain.
- When general newborn screening for Wilson disease becomes feasible, the resultant change in Wilson disease demographics will enhance the need for a coherent approach to management of infants with genetically diagnosed Wilson disease.

Wilson disease (WD) displays toxic accumulation of copper in liver and brain due to dysfunctional or absent ATP7B, the Wilson ATPase, an intracellular copper-transporting P-type ATPase expressed mainly in hepatocytes. ATP7B facilitates production of holoceruloplasmin and, importantly, biliary excretion of copper (1). The average age at diagnosis reported in pediatric WD cohorts is 10 years (2–4). Children older than 24 months with WD, herein referred to as having ''infantile WD,'' are almost invariably asymptomatic (5,6).

With availability of affordable expanded carrier screening-panels, we can detect the carrier status for various genetic diseases in parents. When each parent carries a mutation in *ATP7B*, genetic testing of the infant can lead to an early diagnosis of WD. Alternatively, whole-exome sequencing or sequencing for *ATP7B* mutations independent of the parents' status can also detect WD.

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According to the American College of Obstetricians and Gynecologists Committee on Genetics, criteria for inclusion of a disease in expanded carrier screening-panels include a carrier frequency of ≥1:100, a well-defined phenotype, and onset of disease in childhood (7). Carrier rates for 1 *ATP7B* mutation are estimated at 1:90, except in some isolated populations; a recent British study reported a much higher carrier rate (8). WD is clinically pleomorphic, but its phenotypes are well described: ranging from mild with only liver test abnormalities to severe with hepatic decompensation during childhood, including acute liver failure requiring liver transplantation (2,9). Neurological and psychiatric symptoms are less frequent in childhood (2). With early institution of treatment, disease progression can be prevented and the morbidity associated with neurologic and psychiatric WD can be avoided (10). Thus, WD fits American College of Obstetricians and Gynecologists recommendations for carrier screening inclusion criteria.

Here we present a case of infantile WD as a basis for addressing the implications of genetic diagnosis during infancy. We have collectively reviewed current experience with infantile WD and examined infants' diets for exposure to copper and zinc. Copper is required for normal growth and development in infancy but is potentially toxic in excess. We propose an algorithmic approach for surveillance and management of infantile WD. Our purpose is to stimulate discussion around this emerging issue of how to manage this unique population, and promote multicenter collaborations to gather requisite data.

CASE PRESENTATION

The patient's mother was a healthy 35-year-old woman of Italian-Greek descent who had difficulty with conception due to a uterine septum. Before surgical correction, her fertility evaluation included carrier screening-panel testing. A heterozygous mutation in *ATP7B*, c.3207C>A (p.His1069Gln), was identified, which leads to rapid degradation of ATP7B in the endoplasmic reticulum (11). Her partner, of Italian descent, underwent genetic testing which identified a different heterozygous *ATP7B* mutation: c.845delT (p.Leu282Profs*2) producing a truncated protein (12). These 2 individuals were not known to be consanguineous.

The patient was born following a healthy pregnancy and delivery at term. Birth weight was normal. Genetic testing for *ATP7B* was obtained in the infant at 42 days. Results available by 2 months old revealed that she was compound heterozygote for the 2 parental mutations. Knowledge of the parental genotypes excluded that these 2 mutations were cis, thus affecting both alleles encoding ATP7B. At the initial hepatology evaluation, she was 3 months old, exclusively formula-fed and growing well (weight 76th percentile, length 98th percentile). On examination, she was well-appearing, anicteric, without hepatosplenomegaly. A routine unrestricted pediatric diet was recommended; foods with high copper content were not yet in the diet.

By 8 months of age, the infant was taking 24 oz of formula per day, equating to 329 μg of copper and 3.3 mg of zinc per day. She was ingesting a spectrum of infant purees, all in moderation, and demonstrated appropriate growth.

The infant was evaluated at 12, 15, and 18 months and was consistently asymptomatic with normal development, appropriate growth, and no hepatosplenomegaly. Laboratory results at these visits are included in Table 1. Abdominal ultrasonography at 12 months demonstrated a normal-appearing liver without increased echogenicity or hepatosplenomegaly (Fig. 1).

The parents' utmost concern had been the optimal strategy to avoid any copper overload in their child. At 18 months, she was clinically well and was eating a full diet of table foods, apart from foods high in copper (liver, nuts, mushrooms, chocolate, shellfish). Following discussion of risks and benefits with her parents, at this time treatment was initiated with zinc gluconate 25 mg twice daily, spaced away from meals. Since she was iron deficient before zinc initiation, iron supplementation was administered with meals twice a day. Zinc therapy was well-tolerated. We have not measured basal 24-hour urinary copper (or zinc) excretion as she was not yet toilet trained.

Cases of Infantile Wilson Disease in the Literature

Fourteen cases of infantile WD have been reported plus 2 unpublished cases (J.M.V., R.A.) (Table 2) (4,6,13–21). Genetic testing of infants born to children of known carriers of *ATP7B* mutations will identify patients with WD who are asymptomatic with normal alanine aminotransferase (ALT) and aspartate aminotransferase (AST). Our review of the WD literature revealed that in cases diagnosed during infancy ALT values ranged between normal (12 IU/L) and approximately 10 times the upper limit of normal (556 IU/L). Data are skimpy; however, around the age of 11 to 13 months some had elevated aminotransferases (Table 2). Medical management of these cases was highly variable or else not described.

Physiological Considerations: Copper Metabolism During Fetal Development

Fetal copper trafficking is complicated. Both *ATP7A*, which is defective in Menkes disease, and *ATP7B* encode transporters involved in copper homeostasis in the placenta. ATP7A, the Menkes ATPase, allows copper to be imported across the placenta to the fetus from the maternal circulation (22). ATP7B is also expressed in the placenta and is expected to allow efflux of copper across the placenta back to the maternal circulation (22). In the third trimester this efflux, however, becomes less important as the growing fetus has increased copper needs. In infants whose placental ATP7B is defective, it is unknown if the total body copper is

TABLE 1. Longitudinal serum biochemistry measurements in an infant with Wilson disease before initiation of treatment

Age at testing	8 mo	12 m	15 mo	18 mo
ALT, U/L (normal <33)	21	25	15	20
AST, U/L (normal <32)	54	40	42	39
GGT, U/L (normal <42)	18	14	12	14
Direct bilirubin, mg/dL (normal <0.4)	0.2	0.1	< 0.2	<0.2
Ceruloplasmin, mg/dL (normal > 20)	8	11	11	7
Serum copper, μg/dL (normal 20–70)	25	27	28	25

ALT = alanine aminotransferase; AST = aspartate aminotransferase; GGT = gamma-glutamyltransferase.

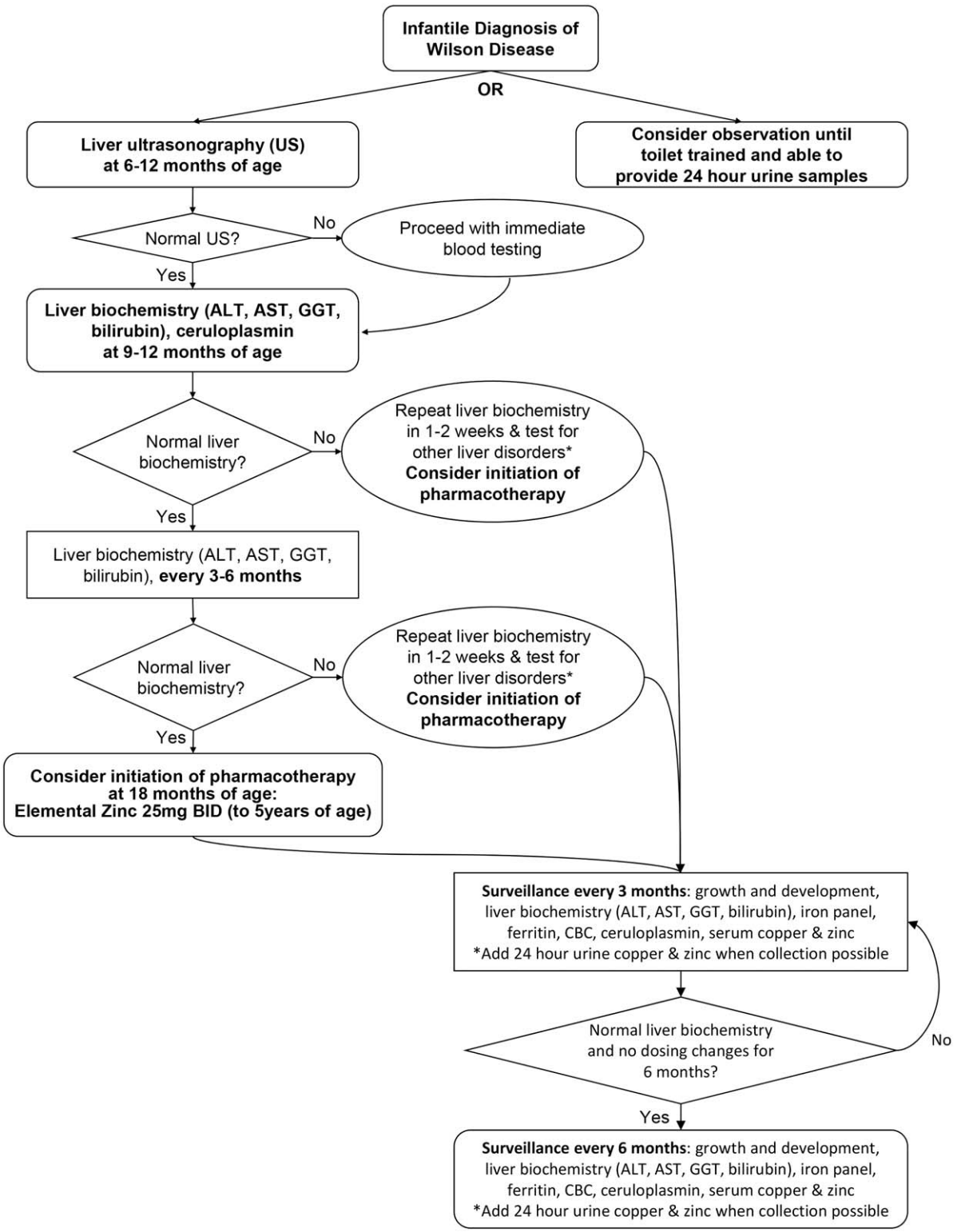

FIGURE 1. Proposed surveillance and treatment algorithm for infants with genetically diagnosed Wilson disease. *Etiologies of other liver disorders include alpha-1-antitrypsin deficiency, viral hepatitis B and C, and autoimmune liver diseases. ALT = alanine aminotransferase; AST = aspartate aminotransferase; GGT = gamma-glutamyltransferase.

TABLE 2. Reported and authors' experience with infantile Wilson disease

Presentation	Origin	ATP7B mutation	Ceruloplasmin, mg/dL	Urinary copper, μg/24 h	Aminotransferases at presentation	Management	Reference
Newborn boy; parents had prenatal genetic testing	USA	p.H1069Q/p.P1379S	26 (at 1 y)	220 (at 1 y)	ALT 110 IU/L AST 57 IU/L (at 1 y)	Low-dose zinc at 16 mo	Bennett et al, 2013 (13)
2-Mo-old girl; parents had prenatal genetic testing	USA (Italian/ Greek ethnicity)	p.H1069Q /c.845delT	8 (at 8 mo)	Not yet performed	ALT 21 IU/L AST 54 IU/L (at 8 mo)	Zinc started at 18 mo of age	Case presented here
3-Mo-old girl; parents had prenatal genetic testing	USA	c.4051C>T / c.2304dupC	<3 (at 6 mo)	Not yet performed	ALT 12 IU/L AST 23 IU/L (at 6 mo)	Dietary counseling	*J. Vittorio, unpublished observations
3-Y-old boy; parents had prenatal genetic testing	USA	p.H1069Q (homozygous)	26 (at 3 y)	23 (at 3 y)	ALT 30 IU/L AST 45 IU/L (at 3 y)	Zinc acetate started at 3 y	*R. Arnon, unpublished observations
4-Mo-old girl; family screening	Greek	Not available	Not available	<100	Not available	Not available	Manolaki et al, 2009 (4)
8-Mo-old boy; unclear reason for ceruloplasmin measurement	Japanese	c.2302insC (homozygous)	9.5 (at 8 mo)	17 (at 8 mo)	Within normal	Not reported	Shimizu et al, 1997 (14)
8-Mo-old boy; ALT measured with an acute diarrheal illness	Chinese	p.G1186S / c.4006delA, and 5 other single nucleotide polymorphisms	7.9 (at 11 mo)	Not reported	ALT 247 IU/L AST 193 IU/L (at 11 mo)	Zinc gluconate 10 mg TID started at 11 mo	Abuduxikuer et al, 2015 (6)
9-Mo-old boy; ALT measured with an acute diarrheal illness	South Korean	p.G1186S / c.4006delA	<9 (at 22 mo)	14.9 (at 23 mo)	ALT 122 IU/L AST 102 IU/L	Zinc 24 mg BID started at ~27 mo. Penicillamine initiated due to increasing aminotransferases	Kim et al, 2013 (15)
10-Mo-old girl; unclear why	Chinese	R776L/none found	Not reported	302	ALT 556 IU/L AST 274 IU/L	Trientine (dose and start date not reported)	Jang et al, 2010 (16)
11-Mo-old boy; family screening	Chinese	G943D/c.2299delC)	16	9 (0.14 μmol)	Not reported	Not reported	Mak et al, 2006 (17)
1-y-old; family screening	Lebanese	c.2299insC / c.2299insC	<2	10	ALT 44 IU/L AST 37 IU/L	Not reported	Usta et al, 2014 (18)
13-Mo-old girl; family screening	Italian	Molecular genetic testing did not reveal a mutation	8	4 (150 After penicillamine challenge)	ALT 210 IU/L AST 168 IU/L	Zinc sulfate ~23 mg BID started at ~16 mo	Iorio et al, 2003 (19)
13-Mo-old girl; unclear why	Italian	c.2299insG ho	7	Not reported	Not reported	Not reported	Nicastro et al, 2010 (20)
15-Mo-old girl; family screening	Chinese	L692P/L1015P	13.4	Not reported	Not reported	Not reported	Zhu et al, 2015 (21)
16-Mo-old girl; unclear why	Italian	P840L/N1270S	3	15	Not reported	Not reported	Nicastro et al, 2010 (20)
19-Mo-old boy; unclear why	Italian	c.2299insG ho	6	15	Not reported	Not reported	Nicastro et al, 2010 (20)
23-Mo-old girl; family screening	Greek	Not available	Not available	Not available	Not reported	Not reported	Manolaki et al, 2009 (4)

ALT = alanine aminotransferase; AST = aspartate aminotransferase.

appropriate or if there is already an over-accumulation present at birth. The experience with the toxic milk mouse models of WD suggest that offspring of an affected dam, who are themselves homozygous for the same *ATP7B* mutation and destined to have murine ''WD,'' are relatively copper deficient: they do not survive unless foster-suckled by a normal mouse (23–25). Even less is known about the contribution to fetal copper homeostasis by a mother who is heterozygous for an *ATP7B* mutation; however, the normal circulating level of copper in these individuals suggests there should be no risk for copper deficiency in infants with breastfeeding. Further study is required to understand the copper balance at birth in infants with WD to guide nutritional and pharmacological recommendations.

With copper restriction or chelation therapy, copper deficiency can potentially arise. Infantile copper deficiency is rarely severe unless the child has Menkes disease. Copper deficiency may, however, develop in infants with malnourishment, premature birth, severe gastrointestinal disorders, intractable diarrhea, and parenteral/enteral nutrition dependence (26). Signs and symptoms of copper deficiency include anemia, neutropenia, bone or vascular lesions, and central nervous system disorders (26). Although copper deficiency is rare overall, the consequences to neurological development can be severe and irreversible (26).

Nutritional Considerations: Sources of Copper and Zinc in Infant Diet

Dietary counseling for patients with WD typically includes a recommendation to avoid foods with high copper content (27). Some pediatric practitioners attach little importance to these restrictions (28). Supplemental Table 1 (Supplemental Digital Content, *http://links.lww.com/MPG/B762*) includes a list of ''high'' copper foods, defined as copper content of >0.2 mg per portion. Foods that are most typically discussed in a restrictive diet are organ meat (liver), shellfish, mushrooms, chocolate, and nuts. These foods are not commonly consumed by infants; thus, their dietary exclusion is not a challenge. The concern for infants is the amount of copper contained in infant formula and baby foods, which tend to be heavy on copper (Supplemental Table 2, Supplemental Digital Content, *http://links.lww.com/MPG/B762*).

Another consideration is the amount of zinc in infants' diets, because dietary zinc may blunt the effect of copper intake by inhibiting gut absorption. The bioavailability of zinc in breast milk is higher than that in infant formulas (29). After birth, the concentration of zinc in breast milk is initially abundant, but by the age of 6 months, like other nutrients, it decreases quite rapidly, potentially putting an infant at risk for zinc deficiency (30). A vegetarian diet of plant-based foods, such as whole grains and legumes, contains less bioavailable zinc (29).

During the first 6 months of life the primary source of nutrition is almost exclusively breast milk or formula, which provides approximately >150% estimated copper and zinc needs. The more hydrolyzed protein formulas provide higher amounts of both copper and zinc. Copper and zinc content of these formulas must be balanced against the presence of gastrointestinal disease.

In infants 6 to 12 months old, breast milk and formula remain a primary source of nutrition, whereas complementary baby foods are added (Supplemental Table 2B, Supplemental Digital Content, *http://links.lww.com/MPG/B762*). Although many families continue to offer pureed baby foods, there has been a recent movement toward the practice of ''baby-led weaning'' where solid foods are offered in their whole form (31). Notably, many of the foods provided are high in copper (>>0.2 mg copper per portion [Supplemental Table 1, Supplemental Digital Content, *http://links.lww.com/MPG/B762*]), including beans/lentils, liver (as an alternative source of iron), avocado, and sweet potatoes.

In the second year of life, infants eat a more varied diet. An average balanced diet in children 12 to 24 months old, with the associated copper and zinc content, is presented in Supplemental Table 2C (Supplemental Digital Content, *http://links.lww.com/MPG/B762*). Many infants demonstrate picky eating behaviors, which may affect copper intake.

Management of Infantile Wilson Disease: Surveillance

Monitoring WD in infancy cannot simply be a modification of how we monitor adults. Infancy is a time of rapid growth and development and obtaining blood samples and timed urine collections is difficult. Reported clinical experience suggests that serum aminotransferases are seldom abnormal before the infant is 9 to 12 months old. This is a reasonable age at which to begin monitoring liver biochemistry: ALT, AST, gamma-glutamyl transferase, total and conjugated bilirubin, albumin, and serum ceruloplasmin. Liver biochemistry can be repeated every 3 to 6 months until starting pharmacotherapy. Detection of elevated liver biochemistry (>1.5 times the upper limit of normal) requires further investigation for other etiologies of liver disease including alpha-1-antitrypsin deficiency, viral hepatitis B and C, and autoimmune liver diseases. In the absence of other infantile liver disease, abnormal liver biochemistries may warrant initiation of therapy. The identification of an additional liver disorder may make it reasonable to delay commencing WD therapy until the effect of copper overload can be confirmed as the cause of elevated liver biochemistry. Such delays should not be unduly protracted.

Liver ultrasonography provides a noninvasive examination of the liver. It can provide pertinent information about hepatic steatosis (32). Obtaining a liver ultrasound around 6 to 12 months old in a healthy infant with genetically diagnosed WD, or earlier if symptomatic, may allay parental anxiety or provide a justification for earlier intervention.

The role of liver biopsy for surveillance is unclear. In general, it should be reserved for patients with liver test abnormalities in whom the diagnosis of WD is uncertain. The role of noninvasive surveillance markers, such as transient elastography, is indeterminate as few infants have been included in studies, and no studies have been reported for infants with WD.

Importantly, this strategy is also suitable for infants whose genetic testing reveals *ATP7B* alterations characterized as variants of unknown significance. Diagnosis in this situation is perplexing. Biochemical confirmation of copper overload is necessary in patients with genetic variants of unknown significance before starting life-long treatment; consultation with a geneticist may be indicated (27).

Management of Infantile Wilson Disease: Treatment

Little published data or experience are available to determine the optimal timing for starting pharmacotherapy in infants. We require data on copper status in this young population to help guide management. A 24-hour urine collection for copper and zinc quantification is not feasible in infants who are not yet toilet trained, unless a urinary catheter is placed (20). The risks of infection or trauma from 24-hour urinary catheterization are disproportionate. Because treatment monitoring is challenging during infancy, a conservative strategy may be most appropriate. The challenge is

to balance achieving disease control against placing the infant at risk of copper deficiency during this critical phase of development.

Oral zinc is a well-established pharmacotherapy for the treatment of WD (10,33–36). Zinc, once absorbed, leads to increased synthesis of metallothionein (MT) in the enterocyte. MT binds copper preferentially over zinc, leading to sequestration of the MT-bound copper in the enterocyte and preventing copper uptake into the portal system (33). Copper is then removed via fecal excretion during regular enterocyte senescence. Zinc salts have fewer adverse effects than oral chelators; the main problem is adherence due to frequent dosing (ie, 2–3 times daily) spaced away from meals. The actual salt is not important for efficacy but may affect tolerability. In pediatric experience, zinc sulfate has been associated with notable gastric distress (33,37). A few zinc preparations exist and may theoretically differ in their tolerability and effect on appetite, nausea, vomiting, and diarrhea (38–40). Zinc as primary treatment for children with asymptomatic WD appears efficacious and well-tolerated (6,36,41,42) with the occasional dissenting report (43). Importantly, chronic treatment with zinc does not decrease hepatic parenchymal copper.

The dose of elemental zinc for treating young children (2–5 years old) is deduced from adult dosing regimens: 25 mg twice daily. This is a relatively high, truly pharmacological daily intake of zinc. Over the long term in adults with WD, zinc treatment may lead to actual copper deficiency, usually evident as anemia with neutropenia or leukopenia, although neurological deficits can develop. Little is known about any potential increased sensitivity of infants to the adverse effects of zinc treatment.

Thus, it seems reasonable to recommend zinc as initial therapy for infantile WD, based on available literature regarding management of presymptomatic children with WD diagnosed through first-degree relative screening (10). Admittedly, treatment options are limited since liquid preparations of chelators are not currently available. Based on review of previous pediatric WD case series, we would consider initial elemental zinc dosing of 25 mg per dose, twice daily, spaced 1 hour before, or 2 hours after meals, as suitable for the infant at the age of 18 months (34,44). If a suspension is unavailable, zinc should be dissolved in water. A more aggressive regimen would be smaller doses of elemental zinc (12.5 mg BID) starting at 12 months or earlier. Patients need to be monitored for copper deficiency with clinical examinations and complete blood count testing (45). Should evidence of copper deficiency develop, zinc can be held for approximately 2 to 3 months while monitoring neurological development and resolution of gastrointestinal symptoms or cytopenias. Further research is needed to determine the actual risk of copper deficiency with zinc treatment in infants with WD: it appears uncommon. Some infants may not benefit from zinc if they have advanced liver damage or persistently abnormal serum aminotransferases while on zinc therapy. They may actually require an oral chelator.

Another reasonable approach could be delaying treatment until liver biochemistry becomes abnormal or until the child is toilet-trained and can provide samples for 24-hour urinary copper measurement (46). Reported ages for successful toilet training vary in the literature, although many children are continent by 22 to 29 months.

Proposed Approach to Management of Infantile Wilson Disease: A Basis for Discussion

The broad outline of our proposal is confirmation of WD, preferably by molecular genetic testing identifying 2 trans mutations; close surveillance in the first 18 months of life; attention to dietary copper and zinc intake; initiation of zinc treatment at the age of 18 months; flexibility to alter this approach if the infant develops symptoms or biochemical evidence of liver disease earlier; and reassurance and support to parents throughout. The possibility that an infant could have WD plus some other more common liver disease cannot be disregarded. The difficulties of treatment monitoring need to be acknowledged upfront.

Special considerations relate to diet during infancy, warranting consultation with a dietician. Breast milk and baby formulas are surprisingly high in copper. Foods introduced at time of formula weaning are often high in copper. As the older infant transitions to an ordinary diet, exposure to most foods in moderation seems like a good plan. Parents may be fearful to allow their infant any intake of high copper foods, but relevant literature should be shared with parents to let them weigh risks and benefits of food choices. Addition of a chewable multivitamin (not containing copper) may be appropriate. Infants with WD should receive all routine vaccinations, including those against hepatitis A and B.

Arguably, given the variability of when WD becomes symptomatic, early treatment of asymptomatic, genetically diagnosed WD may be inappropriate. Further experience is required to address this issue. One possible strategy is that the nature of the mutation needs to be considered. Mutations predicted to result in absent ATP7B may be associated with earlier disease onset. The 3-year-old child presenting with cirrhosis due to WD was homozygous for a mutation predicted to interfere completely with production of the ATP7B (47). In a study of 59 genetically confirmed WD patients, those with 2 ''severe mutations'' (frameshift, nonsense, splice site mutations) had an earlier diagnosis of WD (median 13 years, interquartile range 9–13), whereas ''mild mutations'' were in patients diagnosed later (median 22 years, interquartile range 14–27) (48). An understanding of the infant's specific genetic mutations may influence when to start treatment, but this needs to be confirmed in formal study.

Future Directions

Family screening of first-degree relatives is already recognized as necessary following the identification of an index case. Infants born subsequent to such screening may be found to have WD (27). The scope of the problem of how to manage infants identified as having WD will increase substantially with the implementation of newborn screening. Early detection of WD via newborn screening may be feasible by quantifying ATP7B protein on dry blood cards (49). This approach is still in development.

Likewise, new treatments for WD may affect management of affected infants. Alternative therapies may include agents which ameliorate the effects of an ATP7B mutational folding defect (50). Recent findings show that inhibition of p38 and JNK pathways rescues ATP7B protein with a H1069Q mutation (51). Gene therapy, mediated by the adeno-associated virus, may become an option to correct mutations (52). Infantile WD may be the optimal setting for utilizing some of these treatments to prevent future organ damage.

CONCLUSION

Few data are available in the current literature to guide management of infants diagnosed with WD. Monitored clinical practice and multicenter collaboration will allow for an analysis of aggregate data and help guide future best management. Increased awareness of this issue and critical discussion are needed. Given a

coherent management strategy, newborn screening for WD becomes more practical. Early detection and effective management of WD hold promise of avoiding severe WD in children.

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Wilson Disease

Synonym: Hepatolenticular Degeneration

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Summary

Clinical characteristics. Wilson disease is a disorder of copper metabolism that, when untreated, can present with hepatic, neurologic, or psychiatric disturbances – or a combination of these – in individuals ages three years to older than 70 years. Manifestations in untreated individuals vary among and within families.

- Liver disease can include recurrent jaundice, simple acute self-limited hepatitis-like illness, autoimmune-type hepatitis, fulminant hepatic failure, or chronic liver disease.
- Neurologic presentations can include dysarthria, movement disorders (tremors, involuntary movements, chorea, choreoathetosis), dystonia (mask-like facies, rigidity, gait disturbance, pseudobulbar involvement), dysautonomia, seizures, sleep disorders, or insomnia.
- Psychiatric disturbances can include depression, bipolar disorder / bipolar spectrum disorder, neurotic behaviors, personality changes, or psychosis.
- Other multisystem involvement can include the eye (Kayser-Fleischer rings), hemolytic anemia, the kidneys, the endocrine glands, and the heart.

Diagnosis/testing. The diagnosis of Wilson disease is established in most instances by a combination of biochemical findings (low serum ceruloplasmin concentration, low serum concentration of total copper, and increased urinary copper excretion) and/or detection of biallelic pathogenic (or likely pathogenic) variants in *ATP7B* identified by molecular genetic testing, based on the diagnostic scoring system developed at the 8th International Meeting on Wilson Disease.

Management. *Treatment of manifestations:* Lifelong medical interventions to prevent/treat copper accumulation need to be instituted as soon as possible in all individuals with Wilson disease whether they are asymptomatic (i.e., individuals with biallelic *ATP7B* pathogenic variants who have no clinical manifestations or tissue damage related to Wilson disease), clinically asymptomatic (i.e., individuals with biallelic *ATP7B* pathogenic variants who have no clinical manifestations of Wilson disease, but have Wilson disease-related tissue damage), or symptomatic (i.e., individuals with clinical manifestations of Wilson disease and Wilson disease-related tissue damage), regardless of age and including pregnant women. As Wilson disease treatment decisions might be complex, the consultation of disease experts (primarily hepatologists and neurologists) or Wilson disease centers of excellence is advised. The first-line therapy is copper chelating agents (D-penicillamine and trientine). Zinc salts (which interfere with absorption of copper from the gastrointestinal tract) cannot be used with a copper chelating agent and are most effective after initial decoppering with a chelating agent; however, in some individuals zinc salts can be used as an initial treatment. Orthotopic liver transplantation is used for individuals who fail to respond to medical therapy or present with fulminant acute liver failure.

The goals of supportive treatment for extrahepatic manifestations of individuals with symptomatic Wilson disease are individualized to maximize function and reduce complications. Depending on their clinical manifestations, symptomatic individuals may require specialists in neurology, occupational therapy, physical therapy, physiatry, orthopedics, nutrition, speech-language pathology, social work, and psychology/psychiatry.

Surveillance: To assess treatment effectiveness and adherence to medical interventions that prevent/treat copper accumulation, the following are recommended:

- At least twice annually: assessment of serum copper and ceruloplasmin levels, liver biochemistries, international normalized ratio, complete blood count, urinalysis, and physical examination including neurologic assessment
- At least once annually: measurement of 24-hour urinary excretion of copper

Monitoring the individual's response to supportive treatment for extrahepatic manifestations and the emergence of new manifestations is per the recommendations of the treating clinical specialists.

Agents/circumstances to avoid: Foods very high in copper (liver, brain, chocolate, mushrooms, shellfish, and nuts) should be avoided, especially at the beginning of treatment.

In case of biochemical abnormalities in liver function tests or transaminases, alcohol consumption is strongly discouraged.

Evaluation of relatives at risk: It is appropriate to clarify the genetic status of asymptomatic older and younger at-risk relatives of an affected individual in order to identify as early as possible those who would benefit from prompt initiation of medical interventions to prevent/treat copper accumulation.

Pregnancy management: Treatment must be continued during pregnancy because of the risk for fulminant hepatic failure or irreversible neurologic deterioration. Because of possible adverse effects on the fetus from chelating agents, the dose should be kept as low as possible.

Genetic counseling. Wilson disease is inherited in an autosomal recessive manner. If both parents are known to be heterozygous for an *ATP7B* pathogenic variant, each sib of an affected individual has at conception a 25% chance of being affected, a 50% chance of being heterozygous, and a 25% chance of inheriting neither of the familial pathogenic variants. Once both *ATP7B* pathogenic variants have been identified in an affected family member, carrier and predictive genetic testing for at-risk relatives, prenatal testing for a pregnancy at increased risk, and preimplantation genetic testing for Wilson disease are possible.

Diagnosis

The diagnostic algorithm for Wilson disease in the European Association for Study of Liver (EASL) Clinical Practice Guidelines [European Association for Study of Liver 2012] is based on a diagnostic index ("Leipzig" score) proposed by an expert panel [Ferenci et al 2003]. This score includes clinical, biochemical, and molecular findings, but has not been validated in large patient series. The most recent diagnostic pathway of the American Association for Study of the Liver Diseases (AASLD) highlights diagnostic approaches when clinical and biochemical evaluations are ambiguous [Schilsky et al 2022b].

Suggestive Findings

Wilson disease **should be suspected** in individuals ages three to 45 years, but age alone should not exclude consideration of the diagnosis, as affected individuals have been diagnosed in their early 70s. At diagnosis, individuals with Wilson disease may have varying combinations of the following clinical findings, brain MRI findings (in those with neurologic manifestations), biochemical findings, and family history [Schilsky et al 2022b].

Clinical Findings

Children under age 18 years often present with hepatic disease exclusively.

Adults often present with hepatic disease with or without concurrent neuropsychiatric disease.

- **Liver disease** can range from recurrent jaundice, persistently elevated serum aminotransferase activity (AST, ALT), fatty liver, acute hepatitis (varying in severity, including acute liver injury), autoimmune-type hepatitis, and cirrhosis (compensated or decompensated) to acute liver failure (ALF).

  Note: Specific instances when Wilson disease should be considered is ALF with nonimmune hemolytic anemia or autoimmune hepatitis.
- **Neurologic manifestations,** resulting from central nervous system damage as a result of copper storage, can include the following:
  - Dysarthria
  - Movement disorders (tremors, involuntary movements, chorea, choreoathetosis)

- Dystonia (mask-like facies, rigidity, gait disturbance, pseudobulbar involvement)
- Dysautonomia
- Seizures
- Sleep disorders / insomnia

- **Psychiatric disturbances** can include depression, bipolar disorder / bipolar spectrum disorder, neurotic behaviors, personality changes, and psychosis.
- **Other extrahepatic involvement** can include the following:
  - Eye: Kayser-Fleisher rings, copper deposits in the periphery of the cornea, are observed by slit lamp examination and anterior segment optical coherence tomography (see Członkowska et al [2018], Figure 8). Sunflower cataracts and corneal nerve alterations can also occur.
  - Self-limited hemolytic anemia, with or without acute liver failure
  - Kidney abnormalities: aminoaciduria and nephrolithiasis
  - Hypoparathyroidism, pancreatitis
  - Cardiomyopathy, arrhythmias
  - Premature osteoporosis and arthritis
  - Infertility, recurrent miscarriages

Brain Imaging

Modalities such as magnetic resonance imaging (MRI) are of limited value in determining the extent of clinical neurologic disease but may help initially in supporting a diagnosis of Wilson disease and excluding other neurologic disorders.

Brain MRI findings consistent with Wilson disease include signal changes in the basal ganglia, thalami, pons, and white matter, as well as atrophy. Although the "face of the giant panda" sign (see Schilsky et al [2022b], Figure 1; full text), which consists of increased T_2 signal in the midbrain, has been considered pathognomonic for Wilson disease, several other findings are more commonly seen.

Biochemical Findings

Suggestive biochemical findings in a symptomatic individual relies on a combination of the following findings:

- Low serum ceruloplasmin concentration
  - **In children,** interpretation of test results requires age correction or age-specific reference ranges.

    Note: Healthy newborns have low serum ceruloplasmin concentrations. The concentrations increase during the first six months of life and peak by age two to three years at a concentration that may exceed the healthy adult reference range.
  - **In adults** with Wilson disease, serum ceruloplasmin concentration is often below the normal range (<0.2 g/L) and typically very low (<0.1 g/L).

    Note: A normal serum ceruloplasmin concentration is found in at least 5% of individuals with Wilson disease with neurologic manifestations and up to 40% of individuals with hepatic findings [Steindl et al 1997]. Serum ceruloplasmin concentration is, therefore, not a reliable screening test for Wilson disease.
- **Low serum concentration of total copper.** Most individuals with Wilson disease have a subnormal serum copper concentration that is proportional to the serum ceruloplasmin concentration (as ceruloplasmin is the main copper transporter in blood). The copper bound to ceruloplasmin (i.e., ceruloplasmin-bound copper) is considered nontoxic.

Note: Serum copper is low in healthy newborns. The concentrations increase during the first six months of life and peak by age two to three years at a concentration that may exceed the healthy adult reference range.

- **High urinary copper.** Measurement of copper in three 24-hour urine collections, free from contamination by external sources of copper, is advised. The testing laboratory should be consulted regarding its trace element urine collection protocol prior to initiating urine specimen collection.
  - **Basal urinary copper excretion** (without the use of chelating agents) is almost invariably elevated above 40 µg or ~0.6 µmol/24 hours in most individuals with Wilson disease, and above 100 µg or ~1.6 µmol/24 hours in symptomatic individuals.
  - **A provocative test of urinary copper excretion** following oral administration of D-penicillamine has been validated only in pediatric cohorts, but has proven useful in some adults [Martins da Costa et al 1992]; however, levels in affected individuals can overlap with those of heterozygotes. Note: The use of a lower value for basal urinary copper excretion of 40 µg or ~0.6 µmol/24 hours increases diagnostic sensitivity and may obviate the need for the D-penicillamine provocation test.
- **Hepatic copper quantification.** Although liver biopsy is an invasive procedure, it can be helpful when clinical findings, biochemical findings, and/or molecular genetic test results are ambiguous. Hepatic copper concentration in Wilson disease is usually greater than 250 µg/g dry weight (normal: <55 µg/g dry weight [Nuttall et al 2003]); however, such levels may be seen in other chronic liver disorders as well as cholestatic conditions [Schilsky et al 2022b].

  Note: (1) In later stages of Wilson disease, copper is distributed unevenly in the liver and measurement of hepatic copper concentration is less reliable. (2) Some individuals have only a moderately elevated hepatic copper concentration (100-250 µg/g dry weight), which overlaps with values occasionally found in heterozygotes. Thus, hepatic copper concentration in this range does not exclude the diagnosis of Wilson disease.

Family History

Family history is consistent with autosomal recessive inheritance. The family history may include affected sibs (e.g., sibs with liver disease, neurologic manifestations, and/or psychiatric disturbance) and/or parental consanguinity. Absence of a known family history does not preclude the diagnosis.

Establishing the Diagnosis

The diagnosis of Wilson disease, using clinical, biochemical, and molecular genetic findings, is based on the diagnostic scoring system developed at the 8th International Meeting on Wilson Disease, Leipzig 2001 [Ferenci et al 2003, Członkowska et al 2018] (see Table 1).

Table 1.

Diagnostic Scoring System for Wilson disease

Test	Parameter	Score
Typical clinical symptoms & signs		
Kayser-Fleischer rings	Present	2
	Absent	0
Neurologic manifestations [1]	Severe	2
	Mild	1
	Absent	0
Serum ceruloplasmin	Normal (>0.2 g/L)	0
	0.1-0.2 g/L	1
	<0.1 g/L	2

Test	Parameter	Score
Coombs-negative hemolytic anemia	Present	1
	Absent	0
Other tests		
Liver copper (in the absence of cholestasis)	>250 µg (>4 µmol)/g dry weight	2
	50-249 µg (0.8–4 µmol)/g dry weight	1
	Normal: <50 µg (<0.8 µmol)/g dry weight	-1
	Rhodanine-positive granules [2]	1
Urinary copper (in the absence of acute hepatitis)	Normal	0
	1-2x ULN	1
	>2x ULN	2
	Normal but >5x ULN after D-penicillamine	2
ATP7B molecular genetic testing	Biallelic pathogenic variants detected	4
	One pathogenic variant detected	1
	No pathogenic variants detected	0
Evaluation	**Total score**	
	Diagnosis established	≥4
	Diagnosis possible, more tests needed	3
	Diagnosis very unlikely	≤2

Adapted with permission from Ferenci et al [2003]

ULN = upper limit of normal

1. Or typical abnormalities on brain MRI
2. If no quantitative liver copper available

Per the diagnostic scoring system (see Table 1), the diagnosis of Wilson disease **can be established** in a proband with suggestive findings and biallelic pathogenic (or likely pathogenic) variants in *ATP7B* identified by molecular genetic testing (see Table 2).

Note: (1) Per ACMG/AMP variant interpretation guidelines, the terms "pathogenic variants" and "likely pathogenic variants" are synonymous in a clinical setting, meaning that both are considered diagnostic and both can be used for clinical decision making [Richards et al 2015]. Reference to "pathogenic variants" in this section is understood to include any likely pathogenic variants. (2) Identification of biallelic *ATP7B* variants of uncertain significance (or of one known *ATP7B* pathogenic variant and one *ATP7B* variant of uncertain significance) does not establish or rule out a diagnosis.

Molecular genetic testing approaches can include a combination of **gene-targeted testing** (single-gene testing, multigene panel) (see Option 1) and **comprehensive genomic testing** (exome sequencing, genome sequencing) (see Option 2). Gene-targeted testing requires that the clinician determine which gene(s) are likely involved, whereas genomic testing does not.

Option 1

Single-gene testing. When clinical and biochemical findings strongly suggest the diagnosis of Wilson disease, sequence analysis of *ATP7B* is performed first to detect small intragenic deletions/insertions and missense, nonsense, and splice site variants. Note: Depending on the sequencing method used, single-exon, multiexon, or whole-gene deletions/duplications may not be

detected. If only one or no variant is detected by the sequencing method used, the next step is to perform gene-targeted deletion/duplication analysis to detect exon and whole-gene deletions or duplications.

Note: Targeted analysis can be performed first in individuals from populations with known founder variants (e.g., Ashkenazi Jewish, Canary Islands, Druze, Sardinia; see Table 7).

A multigene panel that includes *ATP7B* and other genes of interest (see Differential Diagnosis) is most likely to identify the genetic cause of the condition while limiting identification of variants of uncertain significance and pathogenic variants in genes that do not explain the underlying phenotype. Note: (1) The genes included in the panel and the diagnostic sensitivity of the testing used for each gene vary by laboratory and are likely to change over time. (2) Some multigene panels may include genes not associated with the condition discussed in this *GeneReview*. (3) In some laboratories, panel options may include a custom laboratory-designed panel and/or custom phenotype-focused exome analysis that includes genes specified by the clinician. (4) Methods used in a panel may include sequence analysis, deletion/duplication analysis, and/or other non-sequencing-based tests.

For an introduction to multigene panels click here. More detailed information for clinicians ordering genetic tests can be found here.

Option 2

Comprehensive genomic testing does not require the clinician to determine which gene is likely involved. **Exome sequencing** is most commonly used; **genome sequencing** is also possible.

For an introduction to comprehensive genomic testing click here. More detailed information for clinicians ordering genomic testing can be found here.

Table 2.

Molecular Genetic Testing Used in Wilson Disease

Gene [1]	Method	Proportion of Probands with Pathogenic Variants [2] Detectable by Method
ATP7B	Sequence analysis [3]	98% [4]
	Gene-targeted deletion/duplication analysis [5]	Rare [6]

1. See Table A. Genes and Databases for chromosome locus and protein.
2. See Molecular Genetics for information on variants detected in this gene.
3. Sequence analysis detects variants that are benign, likely benign, of uncertain significance, likely pathogenic, or pathogenic. Variants may include small intragenic deletions/insertions and missense, nonsense, and splice site variants; typically, exon or whole-gene deletions/duplications are not detected. For issues to consider in interpretation of sequence analysis results, click here.
4. Data derived from the subscription-based professional view of Human Gene Mutation Database [Stenson et al 2020]
5. Gene-targeted deletion/duplication analysis detects intragenic deletions or duplications. Methods used may include a range of techniques such as quantitative PCR, long-range PCR, multiplex ligation-dependent probe amplification (MLPA), and a gene-targeted microarray designed to detect single-exon deletions or duplications.
6. Large deletions and duplications, encompassing one or more exons, are rare. Exon and multiexon deletions have been reported (see, e.g., Møller et al [2005], Incollu et al [2011], Møller et al [2011], Tatsumi et al [2011]).

Clinical Characteristics

Clinical Description

Untreated symptomatic Wilson disease can manifest in individuals ages three years to older than 70 years as hepatic, neurologic, psychiatric, or hematologic disturbances, or a combination of these. Phenotypic expression varies even within families. The understanding of the phenotypic spectrum has further expanded through the widespread use of molecular genetic testing, which has confirmed the diagnosis in individuals with atypical clinical and biochemical findings.

Table 3 outlines the typical presenting clinical findings of untreated Wilson disease. Of note, the "classic triad" of liver disease, movement disorder, and Kayser-Fleischer ring is uncommon.

Table 3.

Clinical Findings in Individuals with Untreated Symptomatic Wilson Disease by Presenting Finding

Presenting Finding	% of Persons	Typical Age of Presentation (Range)	Liver Disease	Neurologic Disease	Psychiatric Disturbance	Kayser-Fleischer Rings
Liver disease	~40%	6-45 yrs (3-70 yrs)	+	+/–	+/–	~50%
Neurologic disease	~40%	Mid-teen to mid-adult (6-50 yrs)	–/mild	+	+/–	~90%
Psychiatric disturbance	~20%	Adolescent to young adult	–/mild	+/–	+	~90%
Hemolytic anemia	Few		+	–	–	+

Bruha et al [2011], Weiss et al [2011], Hofer et al [2012], Weiss et al [2013b]

Untreated Symptomatic Wilson Disease

Liver disease. Untreated Wilson disease manifests as liver disease more commonly in children and younger adults, typically between ages six and 45 years; however, severe liver disease can be the initial finding in preschool-aged children [Wilson et al 2000] and in older adults. The clinical manifestations vary and can include the following findings:

- **Recurrent jaundice,** possibly caused by hemolysis
- **Simple, acute, self-limited hepatitis-like illness** with fatigue, anorexia, and/or abdominal pain
- **Autoimmune hepatitis,** often manifesting acutely with fatigue, malaise, arthropathy, and rashes. This form of liver disease responds well to chelation therapy even if cirrhosis is present (see Management).
- **Fulminant hepatic failure** with severe coagulopathy, encephalopathy, acute Coombs-negative intravascular hemolysis, and often rapidly progressive renal failure. Serum activity of aminotransferases is only moderately increased, and serum concentration of alkaline phosphatase is normal or extremely low. These individuals do not respond to chelation treatment and require urgent liver transplantation (see Management).
- **Chronic liver disease** with portal hypertension, hepatosplenomegaly, ascites, low serum albumin concentration, and coagulopathy
- **Fatty liver** of mild-to-moderate degree with abnormal liver function

Neurologic involvement follows two general patterns: movement disorders or rigid dystonia.

- Movement disorders tend to occur earlier and include tremors, poor coordination, loss of fine motor control, micrographia (abnormally small, cramped handwriting), chorea, and/or choreoathetosis.
- Spastic dystonia disorders manifest as mask-like facies, rigidity, and gait disturbance [Svetel et al 2001].

Pseudobulbar involvement such as dysarthria, drooling, and difficulty swallowing is more common in older individuals, but also occurs in children and adolescents.

In contrast to the neurologic findings in individuals with a frank neurologic presentation, the neurologic findings in individuals with a hepatic presentation may be subtle. Mood disturbance (mainly depression; occasionally poor impulse control), changes in school performance, and/or difficulty with fine motor skills (especially handwriting) or gross motor skills may be observed.

In individuals with a neurologic presentation, extensive changes on brain imaging (such as evidence of tissue cavitation) suggest structural, irreversible brain damage. These individuals are less likely to improve with treatment [Sinha et al 2007].

Psychiatric manifestations are variable. Depression is common. Neurotic behavior includes phobias, compulsive behaviors, aggression, or antisocial behavior. Older individuals may have subtle psychopathology (e.g., progressive disorganization of personality with anxiety) and affective changes (e.g., labile mood and disinhibition). Pure psychotic disorders are uncommon.

Intellectual deterioration may also occur with poor memory, difficulty in abstract thinking, and shortened attention span.

Hemolytic anemia, with either acute or chronic hemolysis, indicates a high serum concentration of non-ceruloplasmin-bound copper, which leads to destruction of erythrocytes. Liver disease is likely to be present in such individuals, as are Kayser-Fleischer rings. Recurrent hemolysis predisposes to cholelithiasis, even in children.

Other extrahepatic involvement

- Kayser-Fleischer rings result from copper deposition in Descemet's membrane of the cornea and reflect a high degree of copper storage in the body. They do not affect vision and are reduced or disappear with effective decoppering treatment (see Management).
- Kidney involvement: low molecular weight proteinuria, microscopic hematuria, Fanconi syndrome, aminoaciduria, and nephrolithiasis
- Arthritis: involvement of large joints from synovial copper accumulation
- Reduced bone mineral density with an increased prevalence of osteoporosis (in approximately 10% of affected individuals)
- Pancreatitis, cardiomyopathy, cardiac arrhythmias, rhabdomyolysis of skeletal muscle, and various endocrine disorders
- Sunflower cataracts: observed occasionally on slit lamp examination

Hepatocellular carcinoma rarely develops in Wilson disease; the estimated incidence is below 1% [Devarbhavi et al 2012].

Fertility and pregnancy. Most individuals with Wilson disease are fertile.

Successful pregnancies of women with Wilson disease who received treatment have been reported [Brewer et al 2000, Tarnacka et al 2000, Furman et al 2001]. Prior to diagnosis and treatment of Wilson disease, affected women may experience amenorrhea, infertility, or recurrent miscarriage [Członkowska et al 2018].

Treated Wilson Disease

The mainstay of treatment for Wilson disease remains lifelong oral pharmacotherapy and dietary copper restriction [Schilsky et al 2022b] (see Management, Medical Interventions to Prevent/Treat Copper Accumulation). Liver transplantation, which corrects the underlying hepatic defect in Wilson disease, is reserved for individuals with chronic or acute liver failure and those resistant to pharmacotherapy.

- **"Asymptomatic individuals with Wilson disease"** are those who have biallelic *ATP7B* pathogenic variants who are clinically asymptomatic and **do not have any Wilson disease-related tissue damage**. Typically these individuals are young infants, born to parents known to be carriers, identified by genetic testing during family screening. These children should remain asymptomatic on treatment, even if they have biochemical abnormalities but not Wilson disease-related tissue damage. (See Management, Evaluation of Relatives at Risk and Medical Interventions to Prevent/Treat Copper Accumulation.)
- **"Clinically asymptomatic individuals with Wilson disease"** are those who have biallelic *ATP7B* pathogenic variants who are clinically asymptomatic but **have Wilson disease-related tissue damage**. Treatment at this stage of disease is highly successful and is focused on stabilizing and reversing tissue injury and preventing the progression of symptoms.
- **Individuals with Wilson disease with symptomatic liver disease.** Improvement in synthetic function and clinical signs such as jaundice and ascites begins during the first two to six months of treatment, with further recovery possible over time.
- **Individuals with Wilson disease with neurologic or psychiatric manifestations.** Most stabilize within six to 18 months after initiation of consistent therapy. However, neurologic findings may not respond to medical treatment, and in a few instances individuals with preexisting neurologic findings might show a paradoxical worsening, with acceleration of neurologic involvement or development of new manifestations.

Genotype-Phenotype Correlations

No genotype-phenotype correlations for *ATP7B* have been identified [Członkowska et al 2018, Ferenci et al 2019].

Nomenclature

The neurologic form of Wilson disease has also been known as Westphal-Strumpell pseudosclerosis.

Prevalence

The prevalence of Wilson disease is estimated at one in 30,000 in most populations, with a corresponding carrier frequency in the general population of one in 90 [Sandahl et al 2020].

In some population-based studies, the genetic prevalence was three to four times higher than clinically based estimates [Olivarez et al 2001, Coffey et al 2013], pointing to the complexity when classifying variants regarding its disease-causing potential and raising the question of whether penetrance is really 100%, as generally assumed.

Recent studies suggest a prevalence as high as one in 10,000, especially in isolated populations such as Sardinia [Gialluisi et al 2013].

Founder variants have been identified in persons of Ashkenazi Jewish and Druze heritage, as well as individuals from the Canary Islands and Sardinia (see Table 7).

Genetically Related Disorders

No phenotypes other than those discussed in this *GeneReview* are known to be associated with germline pathogenic variants in *ATP7B*.

Differential Diagnosis

The complete differential diagnosis of Wilson disease is extensive and includes:

- Copper metabolism disorders;
- Hereditary disorders involving the liver;
- Hereditary disorders involving the nervous system; and
- Acquired conditions such as viral hepatitis, severe drug toxicity, and nonalcoholic steatohepatitis (NASH).

Note: Wilson disease must be specifically excluded in individuals thought to have NASH, or the opportunity for life-saving treatment will be missed.

Table 4 lists selected genetic disorders of interest in the differential diagnosis of Wilson disease (see also Schilsky et al [2022b], Table 5).

Table 4.

Hereditary Disorders of Known Genetic Cause in the Differential Diagnosis of Wilson Disease

Gene(s)	Disorder	MOI	Copper Metabolism
Copper metabolism disorders			
AP1S1	MEDNIK syndrome (OMIM 609313)	AR	Low ceruloplasmin
ATP7A	Menkes disease (See *ATP7A*-Related Copper Transport Disorders.) [1]	XL	Low serum copper & low ceruloplasmin
	Occipital horn syndrome (See *ATP7A*-Related Copper Transport Disorders.)	XL	
	ATP7A-related distal motor neuropathy (See *ATP7A*-Related Copper Transport Disorders.)	XL	Normal
CP	Aceruloplasminemia [2]	AR	Low ceruloplasmin
SLC33A1	Huppke-Brendel syndrome [3]	AR	Low serum copper & low ceruloplasmin
Liver diseases [4]			
ABCB4	MDR3 deficiency (PFIC3) (See Pediatric Genetic Cholestatic Liver Disease Overview.)	AR	Hepatic copper retention due to cholestasis

Gene(s)	Disorder	MOI	Copper Metabolism
HFE	*HFE* hemochromatosis [5]	AR	Hepatic copper retention due to cholestasis is possible.
SERPINA1	Alpha-1 antitrypsin deficiency [5]	AD [6]	
Neurologic disorders			
ATN1	DRPLA	AD	Normal
DNAJC6 *FBXO7* *PARK7* *PINK1* *PRKN* *SYNJ1* *VPS13C*	Early-onset Parkinson disease (See Parkinson Disease Overview.)	AR	
GCH1 *TOR1A*	Inherited forms of dystonia incl DYT1 early-onset isolated dystonia & GTPCH1-deficient dopa-responsive dystonia	AD	
HTT	Huntington disease	AD	
NPC1 *NPC2*	Niemann-Pick disease type C	AR	
Many genes [7]	Hereditary ataxia	AD AR XL Mat [8]	

AD = autosomal dominant; AR = autosomal recessive; Mat = maternal; MOI = mode of inheritance; PFIC = progressive familial intrahepatic cholestasis; XL = X-linked

1. Onset during infancy
2. Iron overload due to lack of oxidase activity of ceruloplasmin
3. Characterized by cataract, sensorineural deafness, and severe developmental delay
4. Primary sclerosing cholangitis (OMIM 613806) and primary biliary cirrhosis (OMIM 109720) also present with abnormal liver biochemistries with or without hepatomegaly. The genetic basis of these disorders is unknown.
5. Presents with abnormal liver biochemistries with or without hepatomegaly
6. Alpha-1 antitrypsin deficiency is inherited in an autosomal codominant manner.
7. See Hereditary Ataxia Overview, Causes.
8. The mode of inheritance depends on the genetic etiology of ataxia.

Management

Evaluations Following Initial Diagnosis

To establish the extent of disease and needs in an individual with symptomatic untreated Wilson disease, the evaluations summarized in Table 5 (if not performed as part of the evaluation that led to the diagnosis) are recommended.

Table 5.

Recommended Evaluations Following Initial Diagnosis in Individuals with Symptomatic Untreated Wilson Disease

System/Concern		Evaluation	Comment
Primary manifestations			
Liver disease		Liver biopsy or biochemical testing & imaging of liver	• Establish baseline copper studies (serum ceruloplasmin & serum copper & 24-hr urinary copper excretion). • Consider additional upper GI endoscopy to exclude or confirm esophageal varices.
Neurologic		Neurologist assess for: • Movement disorders • Gait & balance disturbance	Using validated neurologic rating scale (neurologic subscale of Unified Wilson's Disease Rating Scale) [1]
Speech		For those w/dysarthria: eval by speech-language pathologist	
Musculoskeletal/ADL		Eval by physiatrist/OT/PT	To assess gross motor & fine motor skills, gait, ambulation, need for adaptive devices
Cognitive		Assess for cognitive dysfunction.	
Psychiatric		Eval by psychiatrist, psychologist, neuropsychologist if needed	For personality & mood disorders
Eyes		Complete eye exam	To incl assessment for Kayser-Fleischer rings, sunflower cataracts
Possible secondary manifestations			
Endocrine disorders	Glucose intolerance	Basic biochemical profile	Underlying liver disease might affect hormone metabolism.
	Parathyroid insufficiency		
	Disordered growth		
	Males: gynecomastia		
	Females: menstrual irregularity / amenorrhea		
	Frequent miscarriage		
Cardiac involvement	Cardiac arrhythmia	By cardiolologist	
	Cardiomyopathy		
Renal involvement		By nephrologist	Assess for: • Tubular dysfunction (e.g., aminoaciduria, hypercalcuria, hyperphosphaturia) • Nephrolithiasis, nephrocalcinosis
Genetic counseling		By genetics professionals [2]	To inform affected persons & their families re nature, MOI, & implications of Wilson disease to facilitate medical & personal decision making

System/Concern	Evaluation	Comment
Family support & resources	By treating clinicians, social workers	Assess need for: • Community or online resources; • Social work involvement for parental/caregiver support; • Home nursing referral.

ADL = activities of daily living; GI = gastrointestinal; OT = occupational therapist; PT = physical therapist

Adapted from Schilsky et al [2022b]

1. Członkowska et al [2007], Leinweber et al [2008]
2. Medical geneticist, certified genetic counselor, certified advanced genetic nurse

Treatment of Manifestations

Medical Interventions to Prevent/Treat Copper Accumulation in Individuals with Wilson Disease Who Are Asymptomatic, Clinically Asymptomatic, or Symptomatic

See extensive review by the American Association for the Study of Liver Diseases [Schilsky et al 2022b] (full text) and EASL Clinical Practice Guidelines: Wilson's disease [European Association for Study of Liver 2012] (full text).

Individuals with Wilson disease can be clinically categorized as:

- "Asymptomatic" (individuals who have no clinical manifestations or tissue damage related to Wilson disease);
- "Clinically asymptomatic" (individuals who have no clinical manifestations of Wilson disease but have Wilson disease-related tissue damage); or
- "Symptomatic" (individuals who have clinical manifestations of Wilson disease and Wilson disease-related tissue damage).

The goal of therapy is to institute treatment with chelating agents as soon as possible in individuals with Wilson disease who are asymptomatic, clinically asymptomatic, or symptomatic.

- Treatment is lifelong, including during pregnancy.
- If one treatment is discontinued, an alternative modality must be substituted to prevent disease progression.
- Discontinuation of all treatment leads to hepatic and neurologic decompensation that is usually refractory to further medical intervention.
- During lifelong treatment, failure of any medication used to treat Wilson disease may occur, either at initiation of treatment or during maintenance therapy. Once concurrent disease and nonadherence are excluded, pharmacologic therapy should be re-evaluated and likely altered. For individuals who have more advanced liver disease or develop liver failure, evaluation for liver transplantation should be considered. Currently, no surrogate markers are established for evaluating treatment failure.

Asymptomatic individuals should be treated either with lower dosages (10-15 mg/kg) of a copper chelating agent (D-penicillamine or trientine) or zinc salts.

Clinically asymptomatic individuals should be treated with 15-20 mg/kg of a copper chelating agent (D-penicillamine or trientine).

Symptomatic individuals should be treated with 15-20 mg/kg of a copper chelating agent (D-penicillamine or trientine). However, some individuals with advanced liver disease may require more intensive therapy, and temporally separated combination therapy may be utilized.

Copper chelating agents that increase urinary excretion of copper are the first-line treatment for persons with symptomatic Wilson disease. Note: Routine institution of chelation therapy before age three years has not been adequately assessed and may have adverse effects on growth.

- **D-penicillamine (chelator).** Used since the 1950s as first-line therapy for Wilson disease [Durand et al 2001, Walshe 2003], D-penicillamine is given as tablets by mouth two or three times daily. Pyridoxine must be given along with D-penicillamine. Twenty-four-hour urine copper excretion is used to confirm chelation and increased excretion of copper. Urinary copper values should be five to ten times normal; if the values are lower, noncompliance may be an issue, or body copper stores may have been adequately depleted.
    - Complete blood count and urinalysis must be monitored regularly during D-penicillamine therapy. Serious side effects can occur in up to 30% of individuals, and include severe thrombocytopenia, leukopenia, aplastic anemia, proteinuria, nephrotic syndrome, polyserositis, Goodpasture syndrome, and severe skin reactions. An early allergic reaction with fever, rash, and proteinuria may occur. Evidence of any such side effects may require discontinuation of D-penicillamine and substitution of an alternate treatment. If such alternate therapies are unavailable, D-penicillamine-induced adverse events may be manageable by coadministration of steroids.
    - D-penicillamine inhibits collagen cross-linking and has some immunosuppressant properties. After decades of treatment, individuals may have abnormal skin and connective tissue collagen, and possible chronic depletion of copper and (possibly) other trace metals.
    - D-penicillamine should NOT be used simultaneously with zinc, pending adequate clinical assessment of this treatment strategy.
- **Trientine (chelator),** also known as triethylene tetramine dihydrochloride (2,2,2-tetramine) or trien, has been the usual second-line treatment for individuals who cannot tolerate D-penicillamine. However, a clinical trial of an alternative formulation, triethylene tetramine tetrahydrochloride, revealed good efficacy and better tolerance than D-penicillamine, supporting the concept of its use as first-line therapy [Schilsky et al 2022a].
    - Complete blood count and urinalysis must be monitored regularly in all individuals on trientine.
    - Rare side effects include gastritis with nausea and, in cases of overtreatment, iron deficiency anemia.
    - Trientine should NOT be used simultaneously with zinc pending adequate assessment of this combination. Current reports suggest that the combination of trientine and zinc, temporally dispersed throughout the day such that each drug is administered five to six hours apart from the other, may be effective in severely decompensated hepatic Wilson disease [Santos Silva et al 1996, Askari et al 2003].

Zinc (metallothionein inducer). High-dose oral zinc interferes with absorption of copper from the gastrointestinal tract, presumably by inducing enterocyte metallothionein, which preferentially binds copper from the intestinal contents and is lost in the feces as enterocytes are shed in normal turnover. Zinc therapy is most effective after initial decoppering with a chelating agent [Brewer 2001, Brewer et al 2001]. In selected individuals, it can be used as an initial treatment [Milanino et al 1992, Linn et al 2009].

Zinc is taken as tablets by mouth at least twice (usually 3 times) daily before meals. The dose is based on the elemental zinc in the tablet.

Twenty-four-hour urine copper excretion is used to monitor total body copper stores, which should decrease. Increase of urinary copper excretion under zinc therapy may indicate insufficient treatment efficacy [Weiss et al 2011]. Serum or urinary zinc concentration can be measured to monitor compliance in individuals taking zinc.

Note: (1) Gastritis, a common side effect, can be reduced with the use of zinc acetate or zinc gluconate. (2) Zinc should NOT be used simultaneously with any chelator, pending further clinical investigation.

Restriction of foods very high in copper (liver, brain, chocolate, mushrooms, shellfish, and nuts) is likely prudent, especially at the beginning of treatment. It is recommended that individuals with special dietary needs (e.g., vegetarians) consult with a trained dietitian [Schilsky et al 2022b].

Orthotopic Liver Transplantation

Orthotopic liver transplantation (OLT) is reserved for individuals who fail to respond to medical therapy or cannot tolerate it because of serious adverse side effects [Schilsky et al 2022b].

It remains controversial whether orthotopic liver transplantation should be a primary treatment for individuals with Wilson disease who have severe neurologic disease [Medici et al 2005, Weiss et al 2013a, Litwin et al 2022].

Supportive Treatment for Extrahepatic Manifestations

The goals of supportive treatment for extrahepatic manifestations of individuals with symptomatic Wilson disease are individualized to maximize function and reduce complications. Ideally each individual consults with multidisciplinary specialists in fields such as neurology, occupational therapy, physical therapy, physiatry, orthopedics, nutrition, speech-language pathology, social work, and psychology/psychiatry, depending on the clinical manifestations.

Surveillance

Assessment of Treatment Effectiveness and Adherence to Medical Interventions to Prevent/Treat Copper Accumulation

Monitoring of individuals under therapy should include routine assessments of treatment efficacy by biochemical testing and clinical evaluation.

- Insufficient therapy, underdosage, or poor compliance could lead to reaccumulation of copper and development of new symptoms.
- Adverse events related to medical treatment (especially under D-penicillamine treatment) should be evaluated.
- Excessive long-term treatment could result in copper deficiency, leading to immobilization of iron (as observed in aceruloplasminemia) and neurologic symptoms of copper deficiency [Horvath et al 2010, da Silva-Júnior et al 2011].

According to current guidelines (AASLD [Schilsky et al 2022b] and EASL Clinical Practice Guidelines [European Association for Study of Liver 2012]), routine monitoring should include the following examinations:

- At least twice annually: serum copper and ceruloplasmin, liver biochemistries, international normalized ratio, complete blood count, urinalysis, and physical examination including neurologic assessment

  Note: Individuals receiving chelation therapy require a complete blood count and urinalysis regularly, no matter how long they have been on treatment.
- At least once annually: 24-hour urinary excretion of copper

  Note: Measurements are recommended more frequently if there are questions on compliance or if dosage of medications is adjusted.

Supportive Care

To monitor the individual's response to supportive care and the emergence of new manifestations, the evaluations in Table 6 are recommended based on the supportive treatment required by the individual.

Table 6.

Recommended Surveillance of Extrahepatic Manifestations for Individuals with Symptomatic Wilson Disease

System/Concern	Evaluation	Frequency
Neurologic	Assess for new manifestations such as seizures, changes in tone, & movement disorders.	At each visit
	Consider neuroimaging if new manifestations occur.	Per treating neurologist
Cognitive	Monitor educational needs.	At each visit
Speech & language	By speech-language pathologist & consideration of alternative means of communication	When clinically evident
Feeding	Eval of nutritional status & safety of oral intake	At each visit
Psychiatric/Behavioral	Behavioral assessment for depression, bipolar disorder, personality changes, & aggressive or self-injurious behavior	

System/Concern	Evaluation	Frequency
Musculoskeletal	Physical medicine, OT/PT assessment of mobility, self-help skills	
Family/Community	Assess family need for social work support (e.g., palliative/respite care, home nursing, other local resources), care coordination, or follow-up genetic counseling if new questions arise (e.g., family planning).	

OT = occupational therapy; PT = physical therapy

Agents/Circumstances to Avoid

Foods very high in copper (liver, brain, chocolate, mushrooms, shellfish, and nuts) should be avoided, especially at the beginning of treatment.

In case of biochemical abnormalities in liver function tests or transaminases, alcohol consumption is strongly discouraged.

Evaluation of Relatives at Risk

It is appropriate to clarify the genetic status of apparently asymptomatic older and younger at-risk relatives of an affected individual in order to identify as early as possible those who would benefit from prompt initiation of medical interventions to prevent/treat copper accumulation (see Medical Interventions to Prevent/Treat Copper Accumulation). Asymptomatic and clinically asymptomatic individuals with Wilson disease should remain asymptomatic on treatment, even if they have biochemical abnormalities, histologic findings, or imaging evidence of organ damage. Evaluations can include:

- Molecular genetic testing if the *ATP7B* pathogenic variants in the family are known;
- If the *ATP7B* pathogenic variants in an affected family member are not known, biochemical assessment of parameters of copper metabolism (serum copper, urinary copper, ceruloplasmin) and liver function tests as well as ultrasound imaging of the liver (the finding of a "fatty liver" is common, even in young or asymptomatic individuals) and slit lamp examination for the presence of Kayser-Fleischer rings.

  Note: Asymptomatic individuals with Wilson disease generally have a low serum concentration of ceruloplasmin and mildly increased basal 24-hour urinary copper excretion; however, sometimes asymptomatic individuals with Wilson disease cannot be easily distinguished from heterozygotes.

Although Wilson disease is an autosomal recessive disorder and the risk to the parents and offspring of a proband is low, screening of all first-degree relatives is recommended in order to ascertain clinically asymptomatic family members in whom treatment may prevent liver disease and other manifestations of Wilson disease [Schilsky et al 2022b].

See Genetic Counseling for issues related to testing of at-risk relatives for genetic counseling purposes.

Pregnancy and Lactation Concerns

Pregnancy. Treatment must be continued during pregnancy because of the risk of fulminant hepatic failure and/or neurologic decline in the affected pregnant woman. Baseline biochemical and clinical assessment as soon as a pregnancy is recognized is important. This includes evaluation for portal hypertension in those who have cirrhosis because of the risk of peripartum variceal hemorrhage [Członkowska et al 2018].

- D-penicillamine has been used in many pregnancies with no adverse outcomes; however, congenital connective tissue disorders encompassing inguinal hernias and skin laxity have been reported in some exposed infants. Such adverse outcomes may depend on dose, which should be kept as low as possible while still preventing copper deficiency in the pregnant woman and accounting for the need for fetal copper during development [Członkowska et al 2018]. The dose of D-penicillamine should be maintained at the lowest effective dose during the first and second trimesters of pregnancy. Further reduction in dose may be considered in the third trimester – based on acceptable results of maternal biochemical liver function tests – to account for the increasing copper utilization by the growing fetus.
- Trientine has been used successfully during pregnancy, but the total number of reported individuals is small. Reduction of the dose to the lowest effective dose is recommended using a comparable approach to that for D-penicillamine.

- Zinc has been used effectively in pregnant women and typically does not require a decreased dose during pregnancy. However, changing medical therapy to zinc during pregnancy does not appear to decrease the risk of either miscarriage or adverse fetal outcomes [Członkowska et al 2018].

Lactation. All anti-copper medications appear to pass into breast milk, which can lead to copper deficiency in infants. Therefore, breastfeeding or using expressed maternal breast milk from a mother taking an anti-copper medication is not generally recommended [Członkowska et al 2018].

See MotherToBaby for further information on medication use during pregnancy and lactation.

Therapies Under Investigation

Tetrathiomolybdate (TTM) is an orally administered chelating agent proposed to work by multiple mechanisms [Plitz & Boyling 2019] including:

- Detoxifying non-ceruloplasmin-bound copper by creating a nonreactive tripartite complex with albumin and copper;
- Extracting copper from the endogenous cellular chelator metallothionein (based on its high affinity for copper); and
- Interfering with the intestinal uptake of copper when administered with food.

In a Phase II study [Weiss et al 2017], TTM effectively reduced non-ceruloplasmin-bound copper (corrected for copper-TTM-albumin complex) and improved clinical neurologic findings, without paradoxical neurologic worsening, as demonstrated by an overall improvement in Unified Wilson's Disease Rating Scale scores [Leinweber et al 2008]. Early elevation of serum aminotransferases in approximately 30% of individuals resolved with dose discontinuation or reduction; none developed evidence of drug-induced liver injury. Suitability for treating advanced hepatic Wilson disease requires further investigation. A Phase III trial of bis-choline TTM for Wilson disease is under way.

Search ClinicalTrials.gov in the US and EU Clinical Trials Register in Europe for access to information on clinical studies for a wide range of diseases and conditions.

Genetic Counseling

Genetic counseling is the process of providing individuals and families with information on the nature, mode(s) of inheritance, and implications of genetic disorders to help them make informed medical and personal decisions. The following section deals with genetic risk assessment and the use of family history and genetic testing to clarify genetic status for family members; it is not meant to address all personal, cultural, or ethical issues that may arise or to substitute for consultation with a genetics professional. —ED.

Mode of Inheritance

Wilson disease is inherited in an autosomal recessive manner.

Risk to Family Members

Parents of a proband

- The parents of an affected individual are presumed to be heterozygous for an *ATP7B* pathogenic variant.
- If a molecular diagnosis has been established in the proband, genetic testing is recommended for the parents of a proband to confirm that both parents are heterozygous for an *ATP7B* pathogenic variant and to allow reliable recurrence risk assessment.
- If a pathogenic variant is detected in only one parent and parental identity testing has confirmed biological maternity and paternity, it is possible that one of the pathogenic variants identified in the proband occurred as a *de novo* event in the proband or as a postzygotic *de novo* event in a mosaic parent [Jónsson et al 2017]. If the proband appears to have homozygous pathogenic variants (i.e., the same two pathogenic variants), additional possibilities to consider include:
    - A single- or multiexon deletion in the proband that was not detected by sequence analysis and that resulted in the artifactual appearance of homozygosity;
    - Uniparental isodisomy for the parental chromosome with the pathogenic variant that resulted in homozygosity for the pathogenic variant in the proband.

- Clinical disease is not known to occur in heterozygotes (carriers), although the possibility has not been adequately excluded at older ages. Note: Heterozygotes may have subclinical biochemical findings including low serum ceruloplasmin concentrations, borderline normal urinary copper, elevated urinary copper on provocative testing with D-penicillamine, and/or moderate elevation of hepatic copper (100-250 mg/g dry weight).

Sibs of a proband

- If both parents are known to be heterozygous for an *ATP7B* pathogenic variant, each sib of an affected individual has at conception a 25% chance of being affected, a 50% chance of being heterozygous, and a 25% chance of inheriting neither of the familial pathogenic variants.
- Clinical symptoms may vary between sibs (including monozygotic twins) with untreated Wilson disease. The range of clinical variability observed between sibs with the same biallelic *ATP7B* pathogenic variants and treated Wilson disease depends primarily on the age of diagnosis and treatment initiation, reflecting the period of exposure to copper overload conditions.
- Clinical disease is not known to occur in heterozygotes (carriers), although the possibility has not been adequately excluded at older ages. Note: Heterozygotes may have subclinical biochemical findings including low serum ceruloplasmin concentrations, borderline normal urinary copper, elevated urinary copper on provocative testing with D-penicillamine, and/or moderate elevation of hepatic copper (100-250 mg/g dry weight).

Offspring of a proband

- Unless an affected individual's reproductive partner also has Wilson disease or is a carrier, offspring will be obligate heterozygotes (carriers) for a pathogenic variant in *ATP7B*.
- Given the carrier rate of one in 90 in the general population, the likelihood that an affected individual will have an affected child is one in 180. A higher carrier frequency is observed in some population groups due to founder variants (see Prevalence).
- Because the risk that an individual with Wilson disease will have an affected child is low, testing of serum ceruloplasmin concentration after age one year should be an adequate screening in offspring of a proband, except in populations with a high incidence of Wilson disease and/or a high incidence of consanguinity. In these populations, molecular testing may be useful. If molecular testing is not performed, repeat biochemical testing (including ceruloplasmin and urinary copper excretion) of offspring is strongly encouraged if initial biochemical testing was performed before age three years.

Other family members. Each sib of the proband's parents is at a 50% risk of being a carrier of an *ATP7B* pathogenic variant.

Carrier Detection

Molecular genetic carrier testing for at-risk relatives requires prior identification of the *ATP7B* pathogenic variants in the family.

Heterozygotes may have low serum ceruloplasmin concentrations, borderline normal urinary copper, elevated urinary copper on provocative testing with D-penicillamine, and/or moderate elevation of hepatic copper (100-250 mg/g dry weight), which make these tests unreliable in clarifying carrier status.

Related Genetic Counseling Issues

Predictive testing of adults and children. Because Wilson disease is a treatable condition, it is appropriate to offer predictive testing to asymptomatic at-risk adults and children (see Management, Evaluation of Relatives at Risk).

Family planning

- The optimal time for determination of genetic risk and discussion of the availability of prenatal/preimplantation genetic testing is before pregnancy.
- It is appropriate to offer genetic counseling (including discussion of potential risks to offspring and reproductive options) to young adults who are affected, are carriers, or are at risk of being carriers.
- Carrier testing for the reproductive partners of affected individuals and known carriers should be considered, particularly if consanguinity is likely and/or if both partners are of the same ethnic background. Founder variants have been identified in some populations (see Table 7).

DNA banking. Because it is likely that testing methodology and our understanding of genes, pathogenic mechanisms, and diseases will improve in the future, consideration should be given to banking DNA from probands in whom a molecular diagnosis has not been confirmed (i.e., the causative pathogenic mechanism is unknown). For more information, see Huang et al [2022].

Prenatal Testing and Preimplantation Genetic Testing

Once the *ATP7B* pathogenic variants have been identified in an affected family member, prenatal and preimplantation genetic testing for Wilson disease are possible.

Differences in perspective may exist among medical professionals and within families regarding the use of prenatal testing. While most centers would consider use of prenatal testing to be a personal decision, discussion of these issues may be helpful.

Resources

GeneReviews staff has selected the following disease-specific and/or umbrella support organizations and/or registries for the benefit of individuals with this disorder and their families. GeneReviews is not responsible for the information provided by other organizations. For information on selection criteria, click here.

- **Association Bernard Pépin pour la Maladie de Wilson (ABPWilson)**
  France
  www.abpmaladiewilson.fr
- **Associazone Nazionale Malattida di Wilson**
  Italy
  www.malattiadiwilson.org
- **Deutsche Leberhilfe e.V.**
  Germany
  www.leberhilfe.org/lebererkrankungen/morbus-wilson
- **Morbus Wilson e.V.**
  Germany
  www.morbus-wilson.de/de
- **Wilson Disease Association**
  Phone: 866-961-0533; 414-961-0533
  Email: info@wilsonsdisease.org
  www.wilsonsdisease.org
- **Wilson's Disease Support Group - UK**
  United Kingdom
  www.wilsonsdisease.org.uk
- **American Liver Foundation**
  Phone: 800-465-4837 (HelpLine)
  www.liverfoundation.org
- **Canadian Liver Foundation**
  Canada
  Phone: 800-563-5483
  Email: clf@liver.ca
  www.liver.ca

- **Eurodis**
  Rare Disease Europe
  www.eurordis.org
- **Medline Plus**
  Wilson disease

Molecular Genetics

Information in the Molecular Genetics and OMIM tables may differ from that elsewhere in the GeneReview: tables may contain more recent information. —ED.

Table A.

Wilson Disease: Genes and Databases

Gene	Chromosome Locus	Protein	Locus-Specific Databases	HGMD	ClinVar
ATP7B	13q14.3	Copper-transporting ATPase 2	ATP7B @ LOVD WilsonGen	ATP7B	ATP7B

Data are compiled from the following standard references: gene from HGNC; chromosome locus from OMIM; protein from UniProt. For a description of databases (Locus Specific, HGMD, ClinVar) to which links are provided, click here.

Table B.

OMIM Entries for Wilson Disease (View All in OMIM)

277900	WILSON DISEASE; WND
606882	ATPase, Cu(2+)-TRANSPORTING, BETA POLYPEPTIDE; ATP7B

Molecular Pathogenesis

The product of *ATP7B* is copper-transporting ATPase 2, an intracellular transmembrane copper transporter that is key in incorporating copper into ceruloplasmin and in moving copper out of the hepatocyte into bile. The protein is a P-type ATPase, characterized by cation channel and phosphorylation domains containing a highly conserved Asp-Lys-Thr-Gly-Thr (DKTGT) motif, in which the aspartate residue forms a phosphorylated intermediate during the transport cycle. The gene is expressed mainly in the liver and kidneys.

Tissue damage occurs after excessive copper accumulation resulting from lack of copper transport from the liver. Even when no transporter function is present, accumulation of copper occurs over several years.

Mechanism of disease causation. Various pathogenic variants lead to different impairments in *ATP7B* function.

***ATP7B*-specific laboratory technical considerations.** Comprehensive *ATP7B* testing should include promotor variants, as assessment of exonic sequences only does not rule out the diagnosis of Wilson disease when biochemical and/or clinical features are consistent with the diagnosis.

Table 7.

Notable *ATP7B* Pathogenic Variants

Reference Sequences	DNA Nucleotide Change	Predicted Protein Change	Comment [Reference]
NM_000053.4	c.-436_-422delTGGCCGAGACCGCGG	--	Founder variant common in Sardinia [Loudianos et al 1999]
NM_000053.4 NP_000044.2	c.1934T>G	p.Met645Arg	Founder variants comprising 85% of pathogenic variants in persons of Ashkenazi Jewish ancestry [Shi et al 2017]
	c.3191A>C	p.Glu1064Ala	
	c.3207C>A	p.His1069Gln	
	c.2123T>C	p.Leu708Pro	Founder variant common in Gran Canaria, Canary Islands, Spain [García-Villarreal et al 2000]
	c.3649_3654 delGTTCTG	p.Val1217_ Leu1218del	Founder variant in persons of Druze ancestry [Kalinsky et al 1998]

Variants listed in the table have been provided by the authors. *GeneReviews* staff have not independently verified the classification of variants.

GeneReviews follows the standard naming conventions of the Human Genome Variation Society (varnomen.hgvs.org). See Quick Reference for an explanation of nomenclature.

Chapter Notes

Author Notes

Dr Michael Schilsky's clinical and research interests include transplant hepatology, acute liver failure, and inherited metabolic disorders of the liver, in particular Wilson disease and hemochromatosis. Dr Schilsky co-wrote the AASLD and EASL practice guidelines for Wilson disease and chaired the writing group for the newly released 2022 AASLD practice guidance on Wilson disease. He is author of numerous original manuscripts and reviews on the subject. He is the Principal Investigator on clinical trials of pharmacotherapy and gene therapy for Wilson disease. Dr Schilsky is the organizer and Principal Investigator for the multicenter, multinational registry trial for Wilson disease sponsored by the Wilson Disease Association with data coordinating center at Yale University. He is a member of the NIH-sponsored Acute Liver Failure Study Group. He currently serves as Chair of the Medical Advisory Committee for the Wilson Disease Association.

Dr Karl Heinz Weiss's clinical and research interests include transplant hepatology, Wilson disease, and liver tumors. Dr Weiss co-wrote the 2022 AASLD practice guidance on Wilson disease. He is author of numerous original manuscripts and reviews on the subject.

Drs Weiss and Schilsky are actively involved in clinical research regarding individuals with Wilson disease. They would be happy to communicate with persons who have any questions regarding diagnosis of Wilson disease or other considerations.

Contact Drs Weiss and Schilsky to inquire about review of *ATP7B* variants of uncertain significance. Both authors are also interested in hearing from clinicians treating families affected by Wilson disease in whom no causative variant has been identified.

Acknowledgments

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We would like to acknowledge the Wilson Disease Association for their support of patient care, research, and educational activities.

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Karl Heinz Weiss, MD (2013-present)

Revision History

- 12 January 2023 (bp) Comprehensive update posted live
- 29 July 2016 (bp) Comprehensive update posted live
- 16 May 2013 (me) Comprehensive update posted live
- 24 January 2006 (me) Comprehensive update posted live
- 24 April 2003 (me) Comprehensive update posted live
- 22 October 1999 (me) Review posted live
- 12 May 1999 (dc) Original submission

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WILSON DISEASE ASSOCIATION
Unmasking Strength. Unleashing Promise.

July 23, 2024

Board of Directors
Washington State Department of Health

On behalf of the Wilson Disease Association (WDA), a patient organization dedicated to supporting and educating individuals affected by Wilson disease (WD), I am writing in support of your efforts to include Key Proteo's newborn screening test for newborn screening test (NBS) for Wilson disease in your state's screening panel.

WD is a genetic disorder that is fatal unless detected and treated before serious illness from copper toxicity develops. It affects approximately one in 30,000 people worldwide. The genetic defect causes excess dietary copper to accumulate in the liver or brain. Copper begins to accumulate immediately after birth. Over time, the excess copper harms the liver or brain, resulting in liver, neurologic or psychiatric symptoms. The symptoms usually appear in late adolescence to early adulthood but can occur at any age. WD is fatal if not diagnosed and treated.

Diagnosing WD is challenging for even the most experienced doctors since it can masquerade like many other disorders and is often misdiagnosed, sometimes for many years. Many tests are usually necessary, and sometimes the test results are inconclusive. Early diagnosis and proper treatment are essential to prevent progression of the disease.

We believe an effective newborn screening test would provide better outcomes for patients with Wilson disease so that treatment can be initiated as early as possible. The global Wilson disease community is very excited about the potential for NBS for WD.

We applaud your department for showing leadership in your efforts to have NBS for Wilson disease added to your state's screening panel. We hope that Washington state will be the first jurisdiction to adopt this test, setting the standard for other states to follow.

Thank you for all of your efforts in this area.

Best regards,

Rhonda Rowland

Rhonda Rowland
Vice President, Wilson Disease Association

224 W 35th St Ste 500 # 676 New York, NY 10001 866-961-0533 • 414-961-0533 •www.wilsonsdisease.org

Spanish Association of Relatives and Patients with Wilson Disease
Asociación Española de Familiares y Enfermos de Wilson
Molineta street, 1
04230 Huércal de Almería
Spain

To whom it may concern,

Wilson disease is caused by the accumulation of copper in organs such as the liver and brain. The longer it takes to diagnose and treat, the more copper will accumulate, increasing the probability of symptoms such as liver cirrhosis, speech and walking difficulties, psychiatric symptoms, and even death.

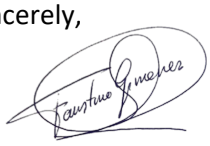
Early diagnosis and treatment of Wilson disease are essential for leading a normal life and avoiding serious complications. Many people remain undiagnosed and suffer from severe neurological symptoms or die from copper accumulation without ever knowing they had Wilson disease or receiving proper treatment.

Implementing newborn screening will improve the quality of life for the boys and girls diagnosed. It will lead to earlier detection, thereby enhancing our knowledge and treatment of the disease. This will also benefit those who are already diagnosed, providing them with more timely and effective care.

The Spanish Association of Relatives and Patients with Wilson Disease (Asociación Española de Familiares y Enfermos de Wilson) wholeheartedly supports the Wilson Disease Association and recognizes the critical importance of implementing newborn screening for Wilson disease in Washington. This initiative will serve as a model, encouraging its adoption in as many states and countries as possible worldwide.

Life has incalculable value, and newborn screening will save countless lives of those affected by Wilson disease.

Sincerely,



Faustino Gimenez Felices
President of
Spanish Association of Relatives and Patients with Wilson Disease
Asociación Española de Familiares y Enfermos de Wilson

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WA State Department of Health

Board of Directors

Heidelberg, 20th July 2024

Dear Washington State Board of Health,

I am writing this letter in support of Dr. Hahn and Key Proteo, Inc. and their petition to include Wilson Disease (WD) on the list of conditions in the WA state newborn screening program.

As a physician and expert in Wilson disease, I have had the privilege of working with Dr. Sihoun Hahn, founder of Key Proteo, Inc., and can attest to their expertise in Wilson Disease and newborn screening.

Wilson Disease (WD) is an autosomal recessive disease of copper metabolism caused by pathogenic mutations in the ATP7B gene, a copper-transporting ATPase. About 1 out of 30,000 individuals are affected by WD. The symptoms typically appear between the ages of 5 and 45 years, and most affected individuals are clinically diagnosed often after they develop significant life-threatening complications, including liver cirrhosis, acute kidney failure, and brain and nerve damage. Early diagnosis and treatment of WD is highly effective in preventing these irreversible damages and can greatly improve the patients' quality of life. Currently there are no efficient and cost-effective screening methods available for early detection of WD. However, the technology developed by Dr. Hahn's laboratory and Key Proteo, Inc. allows early detection of WD prior to clinical manifestation.

Dr. Hahn has over 30 years of experience with WD and has been a pioneer in WD diagnosis. He found that WD patients have very low levels of ATP7B protein, making it a useful biomarker for WD screening. The Immuno-SRM technology developed at Key Proteo, Inc. allows highly precise quantification of ATP7B protein from dried blood spots and can successfully identify WD patients from healthy unaffected individuals. This assay can be readily performed by the WA newborn screening laboratory, since the laboratory already utilizes tandem mass spectrometry on the dried blood spot samples collected from newborn babies. Adopting Key Proteo's technology to screen newborns and detect WD before onset of clinical symptoms can prevent newborns from developing serious permanent complications that require long-term medical care, reduce the financial burden of WD treatment, and greatly improve the long-term clinical outcomes of affected patients.

I wholeheartedly believe that including WD in the WA state newborn screening program can make a lasting impact on thousands of newborns and their families. By testing newborns for WD and providing early intervention to those with WD, we can ensure these children are saved from life-threatening consequences and treated in a timely manner to enjoy healthy lives. Thank you for your consideration.

Sincerely,

Prof. Karl Heinz Weiss
MedicaL Director

Yale School of Medicine

MICHAEL L. SCHILSKY, M.D.
Professor of Medicine

Medical Director, Adult Liver Transplant
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To:
WA State Department of Health
Board of Directors

From:
Michael L. Schilsky MD
Professor of Medicine and Surgery
Director, Center of Excellence for Wilson disease at Yale

Re: Newborn screening for Wilson disease

Dear Board members,

I am Professor of Medicine and Surgery and Director of the Wilson Disease Center of Excellence at Yale, and Chair of the Medical Advisory Committee for the Wilson Disease Association, and currently direct our clinical activities and research program on Wilson disease at Yale. I have over 35 years of experience in working in the field of copper metabolism and Wilson disease with clinical and research expertise in this area. I currently treat approximately over 320 patients with this disorder and direct the Wilson disease patient registry project and an NIH sponsored grant on biomarkers for copper metabolism.

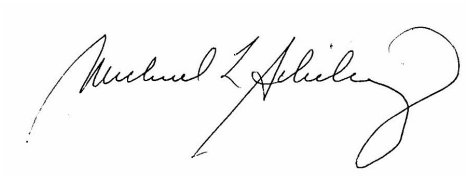
While we are fortunate to have treatments for Wilson disease, there are serious limitations of the available treatment options and disease caused disability due to the late diagnosis of this disorder. For these reasons, the work of Professor Hahn and his colleagues on identifying newborns with Wilson disease is so critically important. When the disorder is identified at an early stage before there is significant liver disease or extrahepatic disease, the patient can then be managed more easily with outcomes for survival comparable to those without the disease. Additionally, the medical and financial burden imposed by disease caused disability due to liver or neuropsychiatric disease can be avoided altogether. This is most desirable.

For these reasons I support the ongoing efforts to utilize the novel testing developed by Professor Hahn to detect this disorder in the earliest stages. We look forward to the successful implementation of the newborn screening program and to the challenge of working with younger patients to prevent disease throughout their lifetime, including the development of new therapeutics that may provide a cure for the disorder. However, all therapeutic efforts are dependent on the

ability to accurately detect the disease. Therefore, the incredible work that has been done by Dr. Hahn in advancing the diagnostics for Wilson disease to where it can be used in newborn screening is extremely important.

I most strongly support the ongoing work of Dr. Hahn and colleagues for the newborn screening project for Wilson disease, and very much appreciate your efforts to date. I look forward to the future universal use of this diagnostic test in clinical practice.

Sincerely,



Michael L. Schilsky MD FAASLD
Professor of Medicine and Surgery
Yale University Medical Center

July 18, 2024

Board of Directors
Washington State Department of Health

I am writing in support of Key Proteo and Dr. Sihoun's Hahn's newborn screening test for Wilson disease. I urge the Washington Department of Health to add Wilson disease to your state's newborn screening panel.

Please indulge me as I share my family's story with Wilson disease.

In December 2022 I had just retired from a 30+ year career in corporate communications and was looking forward to retirement. Our two children (son 23 and daughter 20) were successfully launched. My son had finished university and had a good job. My daughter was in her second year university studying economics and doing well. My husband and I congratulated ourselves on what a great job we had done raising our kids.

Then the walls caved in.

My daughter came home from university feeling very tired, bloated, nauseous and weak. I thought maybe she has irritable bowel syndrome, burnout from studying for exams, the freshman 15 weight gain, her appendix, gall bladder? I thought anything but Wilson disease.

Why would I? I have never heard of Wilson disease.

She suffered through the holidays and slept a ridiculous amount. The fluid was getting more pronounced in her abdomen and she started vomiting at random. A visit to our family doctor who took some blood work and an ultrasound showed fluid in her abdomen and elevated liver enzymes. "Let's monitor and test again in a month," our doctor advised. I realize now that if we had waited, she might not be here.

After the holiday break, my daughter insisted on going back to university to start winter semester. I suggested she stay home so we could arrange for more tests but she was eager to get back to school. She did agree that if the symptoms worsened or didn't improve, she would go to emergency. Two days later, she did just that. Little did we know that she was at end stage liver disease, due to Wilson disease, a condition that she had battled since birth, unbeknownst to any of us.

Fast forward a month later and she was back at home in Toronto Ontario with her new, transplanted liver.

To say we were blindsided is an understatement.

At emergency, doctors quickly diagnosed her with Wilson disease, within a few days. Her liver was so damaged that it couldn't be saved. She needed an emergency liver transplant.

I will never forget the doctor's words. "It's the only lifesaving option at this point." I was frozen and numb. How could this happen to an otherwise healthy young woman who didn't do drugs, didn't drink much, worked out and ate well?

We were lucky. My daughter was put at the top of the provincial transplant list in Ontario and received her new liver within 10 days of her Wilson disease diagnosis. I am forever grateful to her anonymous, deceased donor and their family who made the decision in their unimaginable grief to give back and do some- thing compassionate.

They saved my daughter's life.

While I am thankful for her new liver, I am still haunted with the knowledge that my daughter had a disease since birth that I had no idea about. If we had known that my daughter had Wilson disease as a baby, we could have potentially saved her from needing a liver transplant. She could have been monitored and treated so much earlier before the disease had progressed.

Sadly, my family's story is like many others. A difficult disease to diagnose because it can make other symptoms, people often endure significant liver, neurological and psychiatric symptoms, often irreversible. Left untreated, Wilson idea is fatal.

When I learned about Dr. Sihoun and Key Proteo's work with NBS for Wilson disease, I was very excited. It gives me great hope. If we could diagnose Wilson disease in infants, this could be a game changer and end so many years of suffering and anguish for patients and their families, not to mention significant health savings for the system.

Thank you for your consideration. I hope that Washington State can show leadership and courage and proudly stand as the first jurisdiction to add NBs for Wilson disease to its newborn screening panel.

Sincerely,

Alice Williams

Toronto, Ontario, Canada

To the Washington Board of Medicine

Our family's journey is one that is far too common with Wilson's Disease. Our son Rowdy was 17 years old and preparing to go serve an LDS mission for two years somewhere in the world. As a part of that process to get ready, he went in for his routine physical with our family doctor and friend. Two days after that physical I received an urgent phone call from our family doctor indicating that I needed to get Rowdy to the hospital lab to do additional work to figure out what was going on and that it was a mystery, urgently! As we were discussing things with the doctor, we learned that Rowdy's Platelet blood count was approximately 38 instead of the normal 200, also blood cell shapes and other things were mysteriously wrong. We did as requested and instructed by our doctor and urgently rushed him to the hospital lab to try to find out what was going on with his blood.

Over the course of the next six months with two different oncology specialist doctors we chased every leukemia, bone cancer, and blood cancer that are known. He underwent two separate bone marrow biopsies and was pulled from every physical activity that a normal 17-year-old boy does in fear of bleeding out and possible death. His world was turned upside down. Six months of tests, procedures, countless office visits to doctors and we had no answers. Following his second bone marrow biopsy, under the suggestion of two wonderful doctors at the Anchorage Providence Hospital, we started seeking an internalist to help coordinate all of the care for our son.

We reached out to our family doctor, and inquired who we should be seeing for internal medicine and he made some recommendations, one of the providers I knew. So we made our appointment and began yet another journey of unknowns, additional tests and more doctor visits. Our provider Karen Clements had remembered studying something about these types of mysteries while she was in school, and elected to do a larger blood panel to include many different enzymes and other such things in my son's body. When results came back, there were many red flags of concern as to the balance and health of my son's liver. A few more doctor visits, and our new provider had confirmed that our son had Wilson's Disease.

We had one answer and a whole New World of mysteries. We were referred to Biomedical Genetics at Seattle Primary Children's to begin coordinating care for my son. Additional tests were ordered again and more visits to doctors and hospitals. Ultrasounds, MRIs and CAT scans were ordered to deter,iome the extent of damage caused by the prolonged nature of undiagnosed Wilson's Disease. Our son, now 18-years-old has stage 4 cirrhosis of the liver that will never heal. The cirrhosis of the liver is a direct result of the disease and complications with the copper in his body. The required medication for an individual at this stage of Wilson's Disease is literally $40,000 a month without insurance. He will be on these medications for the next 3 to 5 years, possibly longer. We have now been working with Dr. Hahn and other specialists, geneticists, nutritionists and many others, in our wonderful care team, to treat my son's condition, to prolong the life of his liver, and give him the highest quality of life that he can have at this stage of cirrhosis.

We have a large family and as this is a genetic disorder, genetic tests were ordered for all of our children. This is how we discovered that his younger brother also has the disease. Teancum's journey will be very different from that of Rowdy because his disease was found 5 years earlier in his life. The cirrhosis in his liver is approximately 1.5 on the scale of 5. The

financial burden of the medication is not the same for him as he does not require the mining medication that Rowdy is on. He is able to treat his condition with diet and a therapeutic zinc taken daily, the cost of which is only about $40 a month. His life journey will be very different from Rowdy's because of an earlier diagnosis.

Due to the extent of the damage of Wilson's disease being undiagnosed in his body, there's a very high likelihood that Rowdy will require a liver transplant that could have been avoided with some form of early detection in place. Our family fully supports Dr. Hahn, his team, their research and the desire to make Wilson's Disease testing a functional and easy test at the time of birth. We already conduct many tests through a simple pin prick on the heel of every newborn child in every hospital across the United States to ensure the health safety and quality of life of those children. Wilson's Disease is uncommon and rare. It is debilitating and life altering, and a burden to be born by those who have it. If the State Board will embrace, endorse and approve the testing for newborn children in the hospital, the families of those children who test positive for Wilson's Disease will be able to begin their level of care at birth with low copper diets with high zinc intake. This prevents any damage, liver or the neurological damage, for those children, thus providing them a fullest possible life from beginning to end. Had this testing been available to our family, there's a high likelihood that Rowdy would not have level 4 cirrhosis. He would not be looking at the possibility of a liver transplant; he would be able to engage in regular activities as an 18-year-old young man, all of which is currently being denied to him because of the lack of early testing. If a simple pinprick could've changed my son's life. I would do it 1000 times over. Children should not have to worry about liver failure, Splenomegaly, and the possibility of bleeding to death because they play with their siblings.

A simple test could provide that peace to children. A test that they will never remember, a test that they will never have a memory of feeling,a test that they will never have the memory of conducting and changing their lives for the better. My family supports Dr. Hahn's proposal, and we would hope under the pleading of many many families who face this situation, the State Medical Board will also support this test and support his team in providing this to the newborn children. Once proven in Washington State, this test can be made available in other states, to other children, to other families. We talk about the potential elimination of diseases like polio, mumps, measles and rubella in the United States through simple testing and vaccines. Similarly the long-term debilitating effects of Wilson's Disease is virtually eliminated if care could begin on day one and patients would never feel the effects of the disease that they carry in their genetics, through no fault of their own. Please approve the institution of the heel prick testing of infants for Wilson's Disease.

Sincerely
Christopher and Rachel Johnson
Rowdy and Teancum Johnson
And family

Nora Closser
70 Barons Road
Rochester, NY 14617

July 25, 2024

Washington Department of Health
Board of Directors

To Whom it May Concern,

We are writing this in support of Dr. Sihoun Hahn's newborn screening assay for Wilson's Disease. Our story is like so many others that are diagnosed with a rare disease, it took a very long time to get a diagnosis. The caveat to Wilson's Disease is that unlike many other rare genetic diseases-it is treatable and in most cases manageable. Early diagnosis would have prevented so much disability and heartache that we have endured in the last 23 months of our lives. Our hope is that no family has to go through what we have gone through.

Our 13 year old daughter, Brinley started with abrupt muscle spasms one day at school in October, 2022. These muscle spasms and myoclonus progressed over the course of the next 2 months to balance issues, swallowing issues, extreme fatigue and weakness. I cannot describe the look of terror on her face when she could not control her own body. When we could not get in to see a neurologist or an outpatient MRI of her brain, our former pediatrician recommended taking her to the Emergency Department. The attending neurologist told us that he believed this was a functional disorder and that a brain MRI was not necessary unless we needed it to get on board because in order to treat a functional disorder-formerly known as a conversion disorder, all parties need to be on board. I have been a nurse for over 25 years and in pediatrics for the last 15. I told them we needed the MRI. It was unremarkable. They did not do any additional labs or testing at that 24 hour admission. She was placed on Gabapentin and continued the Hydroxyzine that she was taking. Two months later they added Amantadine because she couldn't focus. She could not focus because she could not control the myoclonus or tremors she was experiencing.

She missed her entire year of 6th grade.
We did home tutoring. The diagnosis of Functional/Conversion disorder followed her until diagnosis.

Her symptoms progressed. She started with severe leg pain in March of 2023. No medications helped her. In May of 2023, she started with severe back pain. Her former pediatrician told me that the only things that would help her would be to get her to sleep, get her to school, get her in a pool and refused to write a continuation letter for home tutoring. The school nurse knew my daughter well and let me call her into school every day. She knew that something was really wrong and urged me to go to a bigger city like Boston and present to their ER. She had been seen by Infectious Disease, Neurology, and Rheumatology. Symptoms progressed. She had

mottling of her hands, Mee's lines on her nails, and looked sickly. I vowed that I would find an answer for her. This was not my daughter. I knew something was very wrong. I researched for hours many nights trying to find an answer. Brinley researched with me. In May of 2023, we changed pediatricians. I had researched all of her new symptoms and Wilson's Disease came up as a differential diagnosis. Her liver function and brain MRI was normal. It wasn't on the radar because of these factors. I needed to talk to a provider who while she didn't know me on a personal level, knew me on a professional level and knew my clinical assessment skills. She immediately wrote the note to continue home tutoring. I asked about Wilson's Disease and she ordered a ceruloplasmin test, which had not been done on this journey. It was low. There was finally something that could explain all of her symptoms. I told Brinley that we may be on the right path to a diagnosis and she said, "Mom, I know this is what it is."

Referral to GI was the next step. She had suffered from unexplained abdominal pain since the age of 10 so we already had a relationship there. The pediatrician sent in the referral the evening the ceruloplasmin level was low. I messaged the GI provider the next morning and she ordered the initial liver screening work up. Everything was still normal except for the ceruloplasmin level. The 24 hour urine screen for copper was normal. We met with the liver team who ordered a referral to Genetics. All of this took time. The positive genetic test did not come back until August 2023. When I told Brinley about the positive test, her response was "I knew it."
No medications helped her symptoms. She tried and failed so many times during this course. She was immediately referred to a Pediatric Hepatologist at Columbia as there were none in our area. We had one in Rochester that left the area 2 days prior to the genetic test results. She was immediately started on Zinc and then did a Penicillamine challenge. She had a seizure with the first dose. When we presented to the ER, we were told by the attending Neurologist that it was likely functional. With one dose, she still had excessive copper in the 24 hour urine. We needed to quantify how much copper was present given the normal liver labs and MRI's. A liver biopsy confirmed high copper content. Columbia wanted us to see specialists at Yale as they are a Center of Excellence. We were seen by Dr. Bamfort at Yale in early December 2023 and then by Dr. Schilsky on December 22, 2024. She started Trientine on December 23, 2023.

Brinley missed her entire year of 7th grade at school.

As a result of her positive test, our 10 year old daughter, Emma was found to be positive for Wilson's disease. She is currently taking trientine. She is asymptomatic and will hopefully remain that way since she was started on treatment earlier. She is diligent about taking her medication and adhering to the dietary restrictions because she saw the hell her sister went through. She never wants to get to that point.

Family testing revealed my nephew was positive and his sisters were carriers.

Brinley took every medication they tried, did all tests ordered, every MRI, every lab draw, every 24 hour urine without a complaint. Never did she say "Why me?". She knew there was an answer, we needed to find it and was willing to do anything to get there.

The impact of a delayed diagnosis took its toll on every aspect of our lives. Physically, mentally, financially….all of it was impacted. No one should have to go through that when a newborn screening tool exists. It would have prevented so much.

In May of 2024, she wrote that she was happy vs. neutral for the first time in over a year. The medication is working.

The retail cost of a 90 day supply of Trientine is $22,700 per child. Thank God we have good insurance. I shudder to think of those that have to choose between paying their electric bill and getting life saving medication.

It has been 7 months since she started chelation. She still has a ways to go, improvements are there. Small things are huge. The hypomimia has improved, I can hear her sing in the car to the radio. I tear up every time. As I write this, we are on our first vacation in 2 years. She is able to participate in so much more these days. She will hopefully get to participate in her last year of Junior High with support.

Carrier status is 1:90. I suspect the incidence of Wilson's Disease is much higher than it is diagnosed. The reality is that it is treatable in most instances. A newborn screening tool would have prevented so much for all of us.

My daughters have exhibited a strength that some adults may struggle with. I cannot express how proud I am of them. We will be advocates for this disease and try to help those navigating it as we navigate it ourselves. Brinley often tells me that she wants her story told.

I thank you for your time and consideration.

Sincerely,

Nora Closser
noratoole@hotmail.com
585-737-5323

Erin Brooks
4907 N Wolcott Avenue, Unit 2B
Chicago, IL 60640

July 25, 2024

Board of Directors
Washington Department of Health
Town Center 2
111 Israel Rd. SE
Tumwater, WA 98501

To Whom It May Concern:

My name is Erin Brooks, and I am writing to urge you to add to add Wilson Disease (WD) to the Washington State newborn screening panel.

My father passed away from Wilson Disease in 2000. At the time, Wilson Disease was a highly unknown disease with minimal resources and options for treatment. It took over a year for my dad to be diagnosed and although there were wonderful doctors overseeing his care, the disease was too much for my dad's body to handle.

Over the past 24 years there have been so many advances made in the diagnosis and treatment for Wilson Disease and if caught early, WD patients and families can live normal and healthy lives. I am so grateful for Dr. Sihoun Hahn's efforts at Seattle Children's Hospital to develop this screening test for WD and lead the pilot project to test this assay on 25,000 newborns in Washington. If this test had been available for my father, his Wilson's would have been discovered long before it took such a serious toll on his body, and he may still be with us today.

So once again, please seriously consider adding Wilson Disease to your newborn screening panel. This is a life-or-death screening for many families.

With deep gratitude,
Erin K Brooks

Dear Dr. Han,

I wasn't diagnosed with Wilson's season 226 mate feeling my second year of medical school at Texas Tech health science Center. I had a Trevor and a grin. Trevor was getting worse and I thought God was punishing me for various events that I thought I was guilty of

It was missed by three expert Neurologist over prior 15 years! It is rarely seen by any neurologist or hepatologist and both kinds of doctors just overlook it. It's inevitable that they will overlook it because I just made you see one case and they're alive If That many?

Hence, with modern medical testing of a newborn DNA would easily diagnose the Wilson's disease gene along with all the other genetic disorders that are diagnosed Postpartum.
Like hemochromatosis ,hemophilia, et al.

Ok ? I feel just Adding Another Fault in a babies DNA the Cost would be in saving and making a life hell of a lot better as well as saving the cost of a liver transplant, which is in the hundreds of thousands of dollars!

In my case it was the cost of the college education and medical school education expenses, while living. Giving up of my dreams of practice medicine and field psychiatry where we know psychiatrist are all over the country. I took disability insurance while I had it. Dammit, That was when I was 40.

Thank You,

Dr Kirk Vestal MD

Date: July 25, 2024

To: WA State Department of Health, Board of Directors

re: My personal story with Wilson Disease

I first started experiencing symptoms of Wilson Disease when I was 20 years old while attending the University of Washington. My symptoms included tremors in my hands and arms, difficulty walking, and slowed speech. My symptoms became so bad that I had to drop out of the University and move back in with my parents.

My parents and I went to see a doctor so that we could find out what was causing my symptoms. After undergoing several medical tests, the doctor misdiagnosed me as having multiple sclerosis. For approximately five years, I lived with this misdiagnosis, while my symptoms kept getting worse, especially the tremors. During this five year period, in order to help correct the tremors, I underwent a type of brain surgery, called a thalamotomy. The surgery was a success, as it definitely helped lessen the tremors in my arms and hands. After the surgery, I did have to go through lengthy physical and occupational therapy to regain my body's full motor skills, in what I referred to at the time as "learning how to walk again".

After the five years of my misdiagnosis of multiple sclerosis, and while undergoing a routine eye exam, I found that I had a brown colored ring around my cornea. The eye doctor informed me that this was unusual and suggested I get it checked out by my doctor. With this new information, I went to see my neurologist. After undergoing some medical tests, I was correctly diagnosed as having Wilson Disease. I then was prescribed the correct medication, cuprimine, to help manage the excess amount of copper in my body. Within about six months of treatment most of my original symptoms of Wilson Disease greatly improved.

I was not correctly diagnosed with Wilson Disease until I was 25 years old (I am currently 61), so my life could have benefited from an earlier detection of the disease. My story shows how important it is to have Wilson Disease as part of mandatory newborn screenings.

Sincerely,

Thomas Sandall

Marilee & Gary Wolter
7324 Lantana Way
Naples, FL 34119

To: Washington Department of Health, Board of Directors,

My name is Marilee Wolter, my husband and I are writing to you to join in the advocating of newborn screening for Wilson disease (WD).

Our daughter Rhonda Rowland was diagnosed at the age of 21 with Wilson disease in Madison, WI by a new gastroenterologist. Looking back, we know she was incredibly lucky that this young physician thought of WD.

Six months prior to her diagnosis, she developed sudden, severe abdominal pain that kept her in bed so she couldn't attend her college classes. We brought her to our family internist in Milwaukee who attributed her abdominal pain to drinking bad water during a recent trip to Florida. Because of abdominal pain and fatigue, she ended her college junior year on academic probation. That summer she had extensive fatigue, experienced moodiness and made careless mistakes at her summer job. By September she developed jaundice, swelling, ascites and cirrhosis of the liver. Fortunately after receiving the correct diagnosis, she responded well to WD medication and has lived a normal life.

In 2016, my husband and I joined Rhonda at a Wilson Disease Association conference. We were shocked and saddened to see the devastating effects of WD. There were people in wheelchairs or walked with difficulty; many people couldn't speak clearly or control their verbal outbursts. Some needed assistance with eating. I couldn't believe they had the same disease as our daughter and we realized just how lucky we were.

Fortunately her younger sister and brother do not have Wilsons. Rhonda is in good health, and has dedicated the last 41 years of her life reporting medical information and raising awareness of this rare disease. We are proud of her work as vice president of the Wilsons Disease Association.

We strongly encourage the Washington Dept. of Health, Board of Directors, to consider and include WD in its newborn screening panel. While life-threatening if not detected, it's no longer a "rare" disease to those who experience it!

Thank-you for your support.

Best Regards,
Marilee & Gary Wolter

Sirs:

I read today that you have developed a screening test for WD. You plan to present letters from patients, caregivers and supporters reinforcing the importance of adding WD to Washington's newborn screening panel.

We live in Highland Park, IL. Our daughter, Hilary, was diagnosed with WD at age 25.

Before she was diagnosed, since she was little, she suffered from stomach pains, illnesses that could not be explained, and general discomfort. WD was not suggested by any doctor. We are not surprised; it is so rare. Her doctors attributed it all to stomach and intestinal issues. Hilary managed to do everything (school, activities, summer camp, college, graduate school) but too frequently with great discomfort. After graduating with a master's degree from NYU, she stayed in New York.

She became extremely sick, exhausted and incapacitated while living in NYC. Doctors gave her disparate diagnoses; one even thought she could be cured with marijuana. Unable to work, she was terrified at the unknown nature of her problems. We dropped everything to join her to try to find out what was going on.

We were led to the wonderful Dr. Dietrich and his PA Helen Adams at Mt Sinai Liver Institute. They diagnosed WD and saved her life. We learned to our shock that we are carriers of recessive genes for a disease we had never heard of. Much damage had already been done to Hilary's liver as a result of copper she could not eliminate. Thankfully, she did not need a liver transplant, but it was discussed. This all could have been avoided had there been a screening test at birth.

Hilary has been successfully treated for 5 years now and we have nothing but optimism for her future.

How much easier her life would have been had she been diagnosed at birth. Please add WD to the newborn screening panel.

Maxine and Michael Bonn

350 North Deere Park Drive West

Highland Park, IL. 60035

mmbonn@gmail.com

To: Washington Department of Health, Board of Directors

I am writing in support of adding the screening test for Wilson's Disease to the newborn screening. Our son was diagnosed with Wilson's Disease on his 24th birthday. He is now 26 and is fully chelated and healthier than ever. That is the good news. The bad news is all of the years that he (and we) suffered with his symptoms, mostly psychological and behavioral. Again, those symptoms are greatly improved now that he is chelated.

When he was a teenager, he had elevated liver enzymes in his bloodwork results. Physicians assumed that it was caused by the medicines he was on because of his psychiatric issues, and they did not assess for other possible reasons. No one considered Wilson's Disease, and we had never heard of it. Just think how different his life (and ours) would have been if he had been diagnosed (and treated) beginning at birth. It would have saved years of heartache for all of us, and he would not have to deal with cirrhosis of the liver for the rest of his life.

Please begin testing all children at birth for this serious disease so that no one else has to experience the sometimes permanent damage caused by Wilson's disease. Thank you for your consideration.

Janet Laubgross, PhD

Clinical Psychologist

July 24, 2024

Board of Director
Washington State Department of Health

I'm writing in support of Key Proteo and Dr. Sihoun's Hahn's newborn screening test for Wilson disease. I urge the Washington Department of Health to add Wilson disease to your state's newborn screening panel.

Please indulge me as I share situation of people with Wilson disease in the place where I live.

My family's journey with Wilson's disease began in 2011. My older daughter, then 9 years old, often complained of abdominal pains. At the beginning we thought this was related to the stress of starting primary school in 2009 and the arrival of her younger sister at the same time. The recurring complaints prompted our physician to extend the diagnostics, which showed high levels of liver tests. For the next six months, doctors observed and monitored my child. Unfortunately, there was liver damage during this period, so we decided to transfer our daughter to another hospital in December 2010. Fortunately, experts in Wilson's disease were working there and in January 2010 we had a diagnosis and treatment began. In February 2011, my younger daughter, who was then 2.5 years old, was also diagnosed.

I run a support group on Facebook for people with Wilson's disease, which currently has 270 patients or relatives as members. A year ago, a dad of a 16-year-old girl who had been diagnosed with Wilson's disease four days earlier wrote in the Facebook support group. The girl required an urgent liver transplant. She had brain swelling and was put into a pharmacological coma. Her liver was transplanted but unfortunately this child's life could not be saved. There is also another student. She is her mother's second child, the first died undiagnosed. There are several more similar stories...

I would like to emphasize that in Wilson's disease, the key issue is the earliest possible diagnosis. Unfortunately, knowledge of the disease among people and physicians is still very low. The introduction of screening could save many lives and certainly dramatically improve diagnosis and treatment and thus prevent side effects and improve the quality of life of those suffering from this disease.

I am in the process of registering a patients' association, of which I will be president. I am in contact with patients from many countries and I intend to take any action that improves the quality of life of people affected by this disease.

As patients and their families, we look forward to organized early diagnosis, to screening. We look to the future with hope. We have high hopes that Washington State will show leadership and courage and stand the first jurisdiction to add NBs for Wilson disease to its newborn screening panel and become in the future an example for other states and countries.

Best regards,
Anna Aniol
Wroclaw, Poland

Board of Directors

Washington State Department of Health

P.O. Box 47890

Olympia, WA 98504-7890

29th July 2024

Dear Board Members,

I am writing to express my strong support for Dr. Sihoun Hahn's newborn screening test for Wilson Disease (WD) and to urge you to consider adding this critical test to your screening panel. Including Wilson Disease in the newborn screening programme could spare many families the devastating consequences of a late WD diagnosis, as my family has endured.

Please allow me to share my daughter Sophie's story, which underscores the urgent need for early detection. Sophie was a happy, (seemingly) healthy child and teenager who excelled in her studies, and she was a talented pianist.

At the age of 15, Sophie experienced a sudden and dramatic personality change accompanied by severe psychiatric symptoms. Like many WD patients, her diagnosis was delayed by about a year from the onset of symptoms and she deteriorated significantly during this time. Sophie is now 19 years old and has spent the last four years in mental health institutions or acute healthcare settings. She hasn't spent a Christmas day at home since she was 14. Sophie's condition has worsened over time. In addition to her initial psychiatric symptoms, she now suffers from hepatic and neurological problems.

Sadly, she has also developed cognitive deficits due to the WD brain damage she has suffered, and it is nothing short of a tragedy that she was unable to complete her education, receiving no qualifications despite being a straight A student. She had always dreamed of becoming a paediatric nurse. Albeit minor in the scheme of things but of significant magnitude to Sophie, sadly, she can no longer play the piano.

Sophie now requires 24-hour care and is under a Deprivation of Liberty order. The once incredibly witty, smart, vibrant, and talented girl I knew has been stripped of everything that brought her joy or function in life. She now has an extremely poor quality of life, and despite four years of treatment with penicillamine, there has been no improvement in her condition. I fear she may never recover, but I will never lose hope.

In Sophie's words: "Mum, it just isn't living."

During a recent visit to our local emergency department, a physician greeted her with, "*I don't know how you are still alive!"* This stark comment underscores the severity of her condition. This is our reality now - a reality I am forced to accept, but if she had been diagnosed as a baby, *oh, how different things would be*!

I would give anything to have known she had WD as a baby. This profound loss and suffering could have been prevented. There are other children out there right now who have WD but don't know it yet, and devastating consequences are looming. This thought keeps me awake at night.

You have the power to prevent other children in your state from suffering the profound, relentless, enduring suffering and loss that we do, every single day. You have the power to save lives.

Thank you for kindly reading about Sophie and for your consideration of this critical matter.

Yours sincerely,

Claire Stapleton

Mother of Sophie, WD Patient

England, UK

E: claire.stapleton1@outlook.com

T: +44 7917 823241

Petition for Rulemaking: Newborn Screening

Chapter 246-650 WAC

Wilson's Disease

Kelly Kramer, Policy Advisor - August 7, 2024

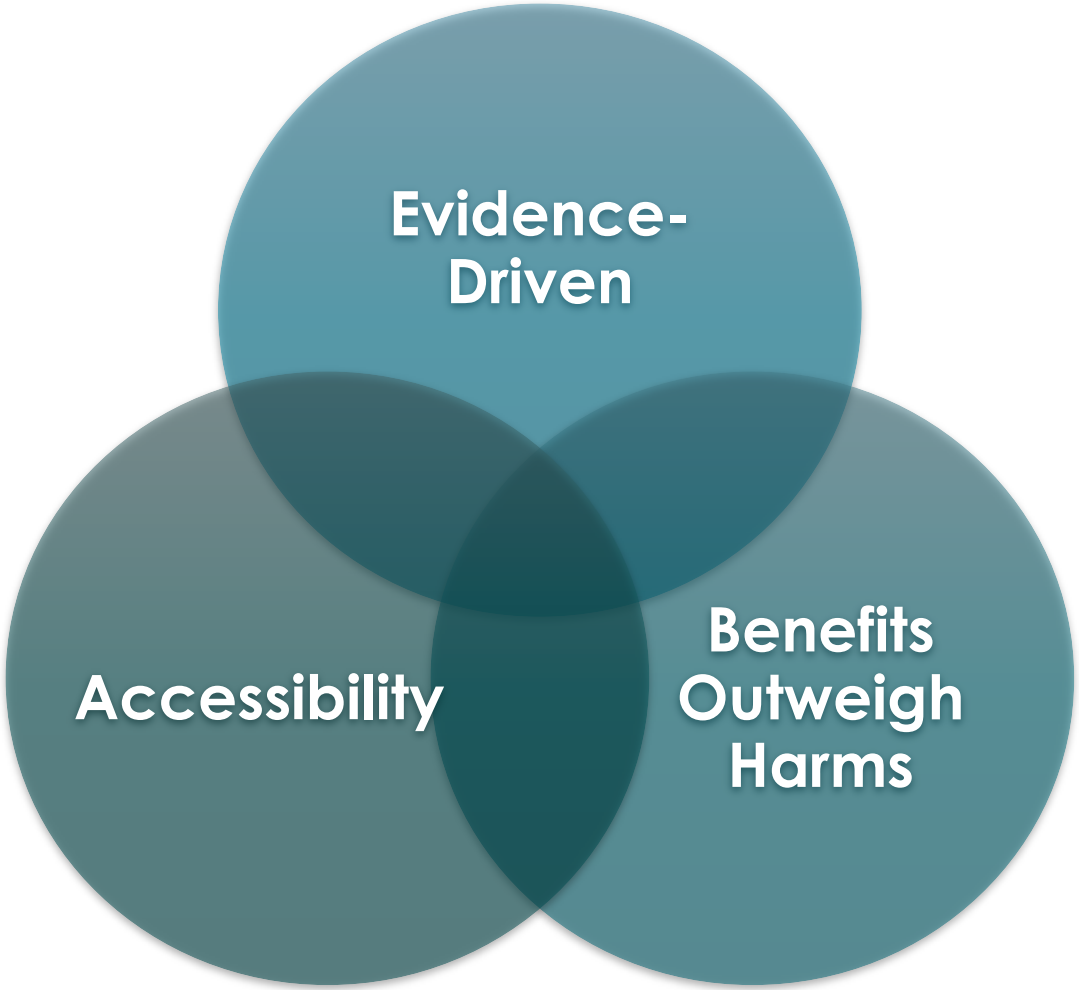
WASHINGTON STATE BOARD OF HEALTH

Kelly Kramer, MPH
Policy Advisor, State Board of Health (Board)

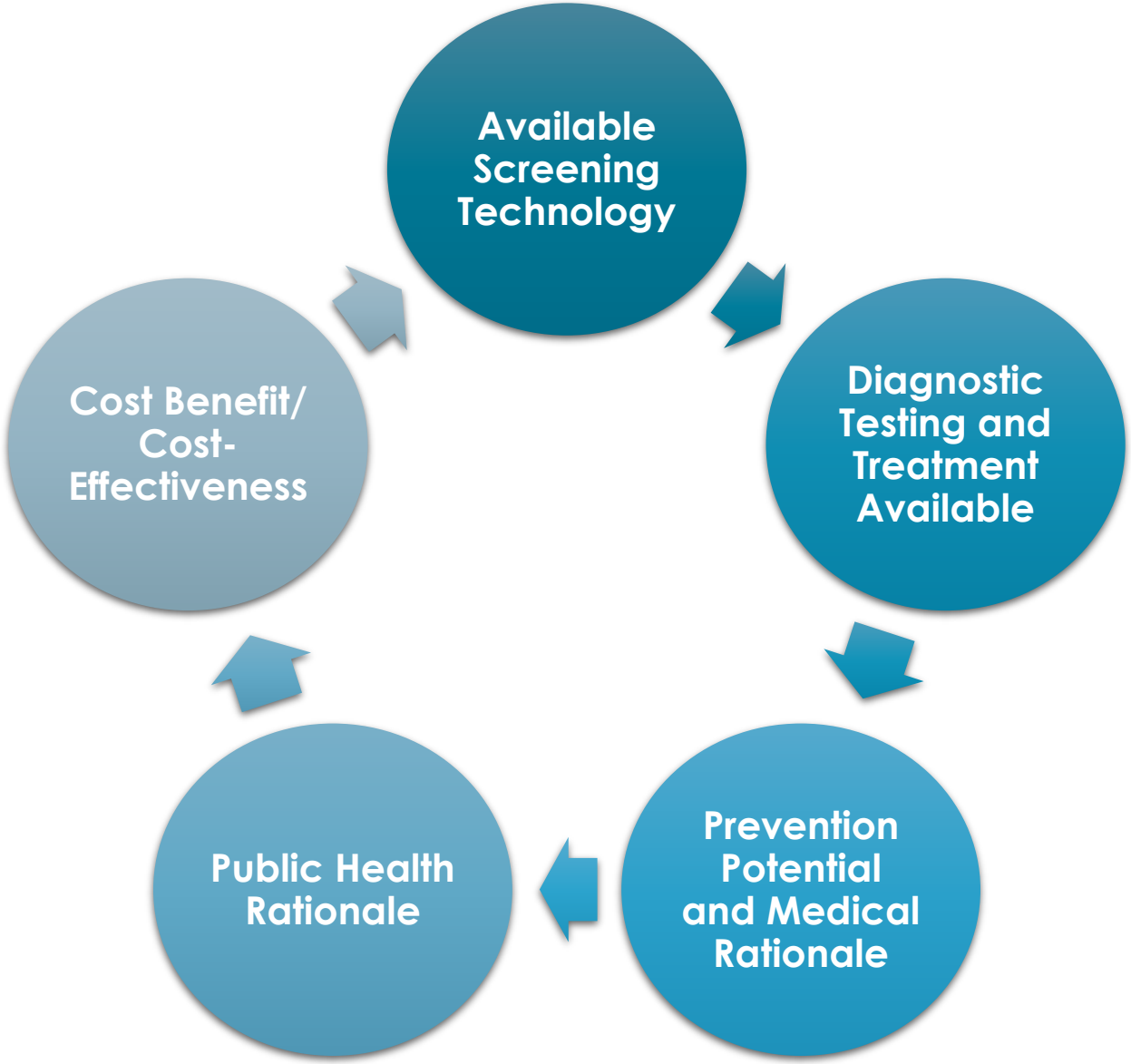
John Thompson, PhD, MPH, MPA
Director, Department of Health (Department) Newborn Screening Program

Board Policy for Newborn Screening

Three guiding principles govern all aspects of the evaluation of a candidate condition for possible inclusion in Washington's Newborn Screening panel:



Five Newborn Screening Criteria



Petition for Rulemaking

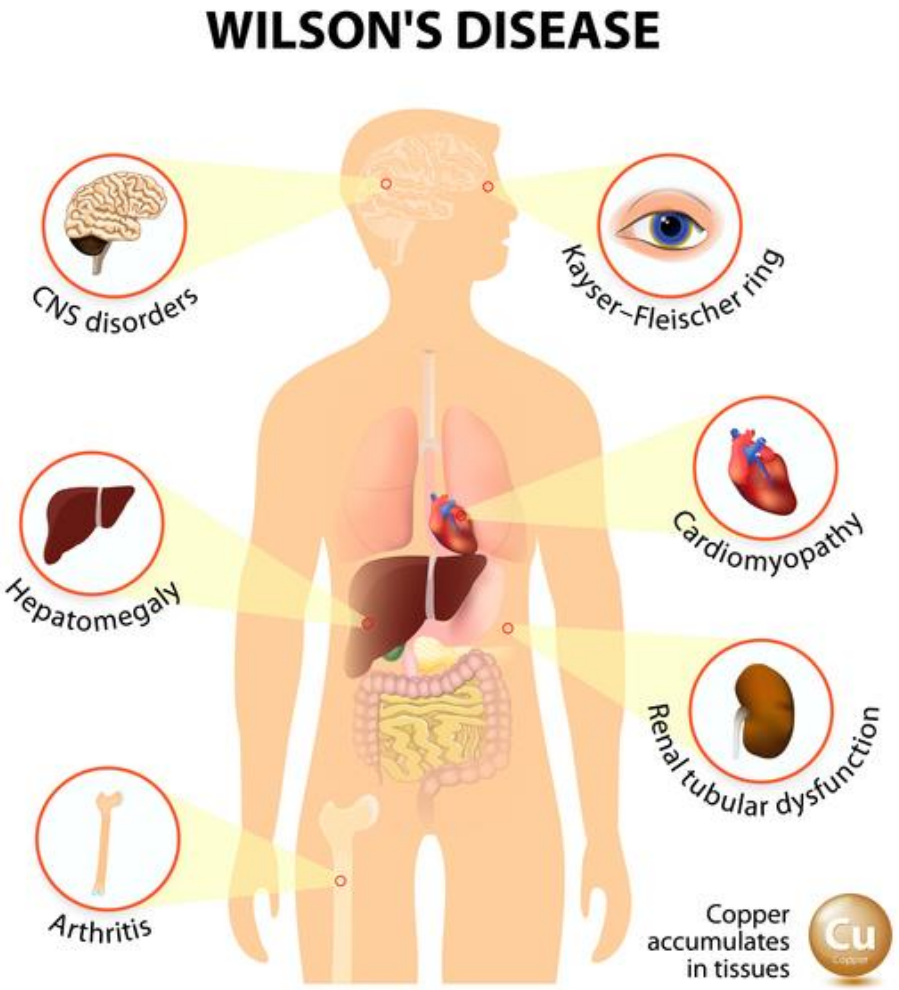
- On July 26, 2024, the Board received a petition request to amend chapter 246-650 WAC to add Wilson's Disease as a mandatory condition on the state's newborn screening panel.

- Early identification of an individual affected with Wilson's Disease would allow for early treatment and prevent tissue damage to the liver or nervous system.

What is Wilson's Disease

- Rare, inherited metabolic disorder
  - Autosomal recessive inheritance pattern
- Prevents body from eliminating excess copper
  - Genetic mutation prevents properly expelling copper
  - Copper builds up in tissues
  - Too much copper is toxic to body
- Symptoms
  - Can occur from age 3-70
  - Jaundice, fatigue, loss of appetite, swelling, difficulty with speech
  - Significant nervous system impairment
  - Death

[Source: NIH, National Library of Medicine; National Organization for Rare Disorders, Mayo Clinic]



Source: Medlineplus.gov/genetics/condition/Wilson-disease

Screening, Diagnostics, Treatment

- Available Screening Technology
  - Proteomic mass spectrometry
  - Key Proteo developed newborn screening kit, piloted at Washington Newborn Screening program

- Diagnostic Testing
  - Low ceruloplasmin in blood
  - High copper in urine
  - Sometimes, liver biopsy and/or brain imaging
  - Molecular testing

- Treatment
  - Copper chelation therapy
  - Liver transplant

[Source: Key Proteo, NIH, National Library of Medicine; National Organization for Rare Disorders]

Prevention Potential and Medical Rationale

- Literature recommends diagnosing Wilson's Disease as early as possible.
- Early connection to treatment prevents permanent neurological damage and liver disease.
  - Treatment is lifelong.
- Available treatments only resolve some complications related to Wilson's Disease.
- Damage to liver and brain is irreversible.

[Source: NIH, National Library of Medicine; GeneReviews; Mayo Clinic]

Public Health Rationale

- Autosomal recessive inheritance pattern
  - Prevalence: 1:32,400
  - Approximately 1 in 90 carry Wilson's Disease gene
  - If both parents are carriers, there's a 1 in 4 chance their child will have Wilson's Disease
  - If parents have a child with Wilson's Disease, they still have a 1 in 4 chance of having another child with Wilson's Disease, and chances stay the same for future children

- Wilson's Disease can impact all people equally
  - No differences based on sex, race, or ethnicity

[Source: National Organization for Rare Disorders]

Considerations

- No state in the US is screening for Wilson's Disease.
- Wilson's Disease is not on the Recommended Uniform Screening Panel (RUSP).
- The Washington Newborn Screening program is running a pilot project for Wilson's Disease screening.
- The petitioner and Washington Newborn Screening program have been working for over 15 years to develop newborn screening tests for Wilson's Disease.

For Board Member Discussion

- Would the Board consider accepting or denying this petition? Why or why not?
- Do Board Members want to direct staff to conduct a preliminary review of the condition and return to the Board at an upcoming meeting? Or proceed to a technical advisory committee?
- Discussion and justification for the Board's decision will be included in the Board's determination letter to the petitioner.

WASHINGTON STATE BOARD OF HEALTH

THANK YOU

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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
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RCW 70.83.020

Screening tests of newborn infants.

(1) It shall be the duty of the department of health to require screening tests of all newborn infants born in any setting. Each hospital or health care provider attending a birth outside of a hospital shall collect and submit a sample blood specimen for all newborns no more than forty-eight hours following birth. The department of health shall conduct screening tests of samples for the detection of phenylketonuria and other heritable or metabolic disorders leading to intellectual disabilities or physical defects as defined by the state board of health: PROVIDED, That no such tests shall be given to any newborn infant whose parents or guardian object thereto on the grounds that such tests conflict with their religious tenets and practices.

(2) The sample required in subsection (1) of this section must be received by the department [of health] within seventy-two hours of the collection of the sample, excluding any day that the Washington state public health laboratory is closed.

[**2014 c 18 § 1**; **2010 c 94 § 18**; **1991 c 3 § 348**; 1975-'76 2nd ex.s. c 27 § 1; **1967 c 82 § 2**.]

RCW 70.83.030

Report of positive test to department of health.

Laboratories, attending physicians, hospital administrators, or other persons performing or requesting the performance of tests for phenylketonuria shall report to the department of health all positive tests. The state board of health by rule shall, when it deems appropriate, require that positive tests for other heritable and metabolic disorders covered by this chapter be reported to the state department of health by such persons or agencies requesting or performing such tests.

[**1991 c 3 § 349; 1979 c 141 § 113; 1967 c 82 § 3.**]

RCW 70.83.050

Rules and regulations to be adopted by state board of health.

The state board of health shall adopt rules and regulations necessary to carry out the intent of this chapter.

[**1967 c 82 § 5.**]

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.

WASHINGTON STATE BOARD OF HEALTH

Notice of Public Meeting

School Environmental Health and Safety Rule Project
Technical Advisory Committee
Thursday, August 1, 2024, 8:30 a.m. – 2:30 p.m.
Meeting location:
Confluence Technology Center
285 Technology Center Way
Wenatchee, WA 98801
Meeting Rooms: Okanogan & Entiat
Language interpretation available

Agenda

Time	Agenda Item	Speaker
8:30 a.m.	Call to Order	Andrew Kamali, Project Manager
8:35 a.m.	1. Welcome Video	Patty Hayes, TAC Chair
8:40 a.m.	2. Meeting Objectives	Karen Langehough, Facilitator
8:50 a.m.	3. Introductions/Ice Breaker	Karen Langehough, Facilitator
10:00 a.m.	4. Role of SBOH & Project History	Andrew Kamali, Project Manager
10:20 a.m.	5. Proviso & Timeline	Andrew Kamali, Project Manager
10:40 a.m.	Break	
10:50 a.m.	6. Charter Agreement	Karen Langehough, Facilitator
11:20 a.m.	7. Decision-Making Options	Karen Langehough, Facilitator
11:40 a.m.	8. Proposed Meeting Dates and Locations	Andrew Kamali, Project Manager
12:00 p.m.	Lunch	
1:00 p.m.	9. Open Discussion/Questions	Karen Langehough, Facilitator
2:00 p.m.	10. Next Steps	Andrew Kamali, Project Manager
2:10 p.m.	Adjournment	

WASHINGTON STATE BOARD OF HEALTH

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- **To access the meeting online and to register:**
  https://us02web.zoom.us/webinar/register/WN_tDNCXI-HSV2o5eoYzQmNhQ

- **You can also dial-in using your phone for listen-only mode:**
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+1 646 558 8656	+1 646 931 3860	+1 689 278 1000
+1 301 715 8592	+1 305 224 1968	+1 309 205 3325
+1 312 626 6799	+1 360 209 5623	

Webinar ID: 847 8644 6696
Passcode: 620348

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 written public comment:

- Please visit the Board's Public Comment webpage for details.

WASHINGTON STATE BOARD OF HEALTH

School Environmental Health and Safety Rule Project 2024 - 2025

TAC Membership

MEMBER	ALTERNATE	REPRESENTING
Patty Hayes WSBOH Chair		Washington State Board of Health
Tyler Muench Director of Advocacy & External Affairs	**Randy Newman** Director of School Facilities & Organization	Washington State Office of Superintendent of Public Instruction
Steve Main Division Director, School Safety Lead	**Sandy Phillips** School Health and Safety Program Technical Advisor	Spokane Regional Health District
Gina Yonts Associate Director	**Roz Thompson** Director of Government Relations	Association of Washington School Principals
Geoff Lawson Operations Coordinator	**Jeff Rogers** Manager or Environmental Health & Safety	Washington Association of Maintenance and Operation Administrators & Auburn School District
Tammy Bigelow Board Director – Region 121	**Nicole Roel** WASBO Board of Directors, Olympia ESD 114	Washington Association of School Business Officials
David Hammond School Construction Committee Chair	**Dan Steele** Assistant Executive Director, Government Relations	Washington Association of School Administrators
Suzanne Hanson Executive Director	**Sharon Ricci** Community Relations	Washington Federation of Independent Schools
Kate Espy Board Member and Legislative Representative		South Kitsap School District
Erin Hockaday Senior Manager, Surveillance & Investigation		Benton-Franklin Health District

School Environmental Health and Safety Rule Project 2024 - 2025

TAC Membership

MEMBER	ALTERNATE	REPRESENTING
Laurette Rasmussen School EH Specialist	**Jamie Bodden** WSALPHO Managing Director	Whatcom County Health & Community Services
Lauren Jenks Assistant Secretary, Environmental Public Health	**Kelly Cooper** Director, Policy and Legislative Relations	Washington State Department of Health
Kevin Jacka Executive Director	**Richard Conley** Consultant	The Rural Alliance
Samantha Fogg Co-President Seattle Council PTSA		Seattle Council PTSA
Devon Kellogg Volunteer WSPTA, Advocacy Committee	**Susan Baird-Joshi** Volunteer WSPTA	Washington State PTA
Laura Peterson Volunteer/Appointed Role WSPTA		Washington State PTA
Brook Wilkerson Director of Operational Supports	**Anders Lindgren** President	School Ops
Preet Singh Director of Health Services	**Jessica Sankey** Chief Operations Officer	Bellingham Public Schools
Brian Buck Executive Director of Support Services	**Kenny Johnson** Director of Maintenance & Operations	Lake Washington School District
Kellie Lacey Assistant Director of Human Resource	**Kelsey Greenough** Records Specialist	Richland School District
Nicole Daltoso Senior Director of Capital Facilities	**Martin (Marty) Madarieta** Director of Maintenance	Evergreen Public Schools

TAC Membership

MEMBER	ALTERNATE	REPRESENTING
Brian Freeman Superintendent		Inchelium School District
Rebecca Doughty Executive Director of School Support Services (Operations)		Spokane Public Schools
Jared Mason-Gere Government Relations Staff	**Julie Salvi** Lobbyist/Government Relations	Washington Education Association
Jake Cook Public Advocate	N/A	Public
Pam Schwartz Assistant Superintendent	**Doug Rich** Superintendent	Washington State Catholic Conference

School Rule Project Staff

Andrew Kamali
School Rule Project Manager

Nina Helpling
Policy Advisor

Mary Baechler
Community Engagement Coordinator

Marcus DeHart
Communications Consultant

Crystal Ogle
Administrative Assistant

WASHINGTON STATE BOARD OF HEALTH

School Environmental Health and Safety Rule

What: Updates to K-12 School Environmental Health and Safety Standards

The Washington State Board of Health (Board) is working to develop new proposed standards for K–12 school environmental health and safety. The Board plans to develop and propose new language to the legislature by June 2025.

Why:

The current standards are over 50 years old, and the Legislature considers them outdated. Legislative restrictions delayed updating the standards, but now the Legislature has directed the Board to propose a new school environmental health and safety rule. As part of that process the Board is inviting all interested parties (parents, teachers, administrators, the general public) to provide comments and make recommendations.

History:

Chapter 246-366[1] of the Washington Administrative Code (WAC) sets the current standards for K–12 school environmental health and safety for over one million students. In 2004, the Board initiated rulemaking to update this outdated rule and spent the next five years creating and adopting chapter 246-366A[2] WAC.

In the 2009–2011 biennium, the Legislature directed the Department of Health (Department) and the Board not to implement any new or amended rules related to these school facility standards due to concerns about the cost of implementation. Every budget since 2010 has included the proviso. In response, the Board has continued to extend the effective date of Chapter 246-366A.

In 2016, Governor Inslee directed the Department to continue providing technical assistance and guidance for school districts to conduct voluntary water quality tests[3]. The Legislature appropriated over 7.4 million dollars to the Department and the Office of the Superintendent of Public Instruction (OSPI) during the 2019–2021 and 2021–2023 biennium budgets to support lead testing and remediation in schools.

During the 2024 legislative session, the Legislature included funds for the Board to review chapter 246-366 and 246-366A WACs and to propose updated environmental health and safety standards for K–12 schools in Washington state.

[1] https://apps.leg.wa.gov/WAC/default.aspx?cite=246-366&full=true&pdf=true
[2] https://apps.leg.wa.gov/wac/default.aspx?cite=246-366A&full=true&pdf=true
[3] https://governor.wa.gov/sites/default/files/directive/dir_16-06.pdf

School Environmental Health and Safety Rule Project 2024 - 2025

The Legislature directed the Board to:

- Convene a technical advisory committee (TAC) consisting of various school associations, school districts, and OSPI to propose updated requirements.
- Collaborate with OSPI to develop a fiscal analysis.
- Assist the Department in completing an environmental justice assessment[4] on any proposed rule.
- Work with the Department, OSPI, the TAC, and local health jurisdictions to provide a report to the Office of the Governor and appropriate committees of the Legislature by June 30, 2025.

The Board's Timeline:

DATE	MILESTONE/ACTION	PURPOSE
May 2024	Invite TAC Members	In addition to the members that the proviso required, the Board will include additional members such as Parent-Teacher Organizations, Teachers Unions, Students, and Private Schools.
June 20, 2024	Filed CR-101 Pre-proposal Statement of Inquiry	The Board formally filed WSR 24-13-117[5] to announce the intent to create rule language.
Aug 2024 – Nov 2024	TAC Meetings	The Board rulemaking team works with TAC members to draft rule language and discuss implementation.
Nov 2024	Initial Draft Rule Complete	Interested parties and members of the public review the draft rule language.
Dec 2024	Focus Groups	The Board rulemaking team holds virtual and in-person meetings to discuss the preliminary draft language and make informed decisions about the finalized draft rule language. These meetings will take place across Washington state.
Dec 2024	Informal Comment Period	The Board rulemaking team invites all interested parties to review and share feedback on the draft rule language.

[4] https://doh.wa.gov/community-and-environment/health-equity/environmental-justice/assessments
[5] https://sboh.wa.gov/sites/default/files/2024-06/WSR 24-13-117.pdf

School Environmental Health and Safety Rule Project 2024 - 2025

The Board's Timeline:

DATE	MILESTONE/ACTION	PURPOSE
March 12, 2025	Preliminary Review by the Board	Board Members preliminary review of the draft proposed rule language, Environmental Justice Assessment, and Fiscal Analysis.
April 9, 2025	TAC Provides Recommendations to the Board	TAC members address the Board to provide comments and make recommendations.
May 2025	Final Draft Rule Proposal	After considering all comments and recommendations, the Board will finalize the draft rule.
June 11, 2025	Board Approves Report	The Board approves the final draft rule documents and recommendations and will submit them to the Governor's office and legislative committees.
June 30, 2025	Report to the Governor	The Board will submit the final draft rule language, Environmental Justice Assessment, and Fiscal Analysis to the Governor's office and legislative committees.

You can subscribe at schoolehs@sboh.wa.gov to receive notifications about this rule update or participate in our TAC meetings, focus groups, or provide informal comments.

For more information, please visit our website[6].

Do you have questions about this rule or the environmental justice assessment? If so, please contact us at schoolehs@sboh.wa.gov.

[6] https://sboh.wa.gov/rulemaking/agency-rules-and-activity/2024-2025-school-rule-review-project

WASHINGTON STATE BOARD OF HEALTH

Date: August 7, 2024

To: Washington State Board of Health Members

From: Paj Nandi, Board Member

Subject: Pro-Equity Anti-Racism Plan and Playbook Briefing

Background and Summary:

The COVID-19 pandemic highlighted disparities that impact Washington State communities in different ways, often leading to inequitable outcomes. The Governor's Executive Order 22-04 implements the Washington State Pro-Equity Anti-Racism Plan and Playbook (PEAR). It requires that all state agencies, including boards and commissions, implement a PEAR Strategic Action Plan to drive systemic change, work towards dismantling oppressive systems, and promote equity across all of society.

This year, the Board will need to complete their initial PEAR Strategic Action Plan. The PEAR Strategic Action Plan must be within our sphere of influence, capacity, and authority. Today's presentation and materials will include general background information on the PEAR Plan, current Board staff progress, a draft PEAR Strategic Action Plan, and an updated timeline. We are asking the Board to discuss goals 1 and 2 of the draft PEAR Strategic Action Plan and recommend changes to reduce disparities and bridge gaps with communities.

Staff

Ashley Bell

PO Box 47990 • Olympia, WA 98504-7990
360-236-4110 • wsboh@sboh.wa.gov • sboh.wa.gov

PRO-EQUITY ANTI-RACISM PLANNING

Ashley Bell, MPA
Equity and Engagement Manager
Washington State Board of Health
August 7, 2024

WASHINGTON STATE BOARD OF HEALTH

Agenda

- Brief Overview
- Progress Update
- Draft Discussion

PEAR Progress

Thoughts, Feedback, and Questions

WASHINGTON STATE
BOARD OF HEALTH

Pro-Equity Anti-Racism (PEAR)

- The Office of Equity recognizes that systems of oppression are the upstream sources of all inequities
  - Directs state agencies, Boards, and Commissions to implement a PEAR Strategic Action Plan

- The PEAR Strategic Action Plan works to:
  - Drive systemic change
  - Dismantle oppressive systems
  - Promote equity in all facets of society

- We want Washingtonians to:
  - Be involved in decision-making
  - Deliver services that meet their needs
  - Trust state government

Pro-Equity Anti-Racism (PEAR)

- With the PEAR Plan we can:
  - Bridge opportunity gaps and reducing disparities statewide and across state government
  - Invest where the needs are the greatest to addresses upstream, root cause, issues that perpetuate systemic inequities
  - Create meaningful impact to the determinants of equity

- We can invest in intentional and meaningful change in how we do our work by embedding equity into decision making. This can:
  - Reduce disparities in key business areas
  - Improve outcomes that benefit all tribes, communities, and employees

PEAR Progress

Thoughts, Feedback, and Questions

WASHINGTON STATE
BOARD OF HEALTH

PEAR Progress

- ✓ Develop a PEAR Team (April 2024)
- ✓ Completed an Equity Impact Assessment of the Board’s scope of work (June 2024)
- Develop and Implement a PEAR Strategic Action Plan (Ongoing through 2024)
- Track and Report Performance (End of year 2025)

✓ PEAR Team Creation

- Internal team was self-selected, and includes:
  - Board Sponsor
  - Executive Director
  - Equity and Engagement Manager
  - 8 additional staff

- We have been engaging with a few community-based organizations to provide recommendations on:
  - Metrics
  - Objectives
  - Additional community partners

- We have been meeting bi-weekly to collectively work on the PEAR Plan

✓ Equity Impact Assessment

- Completed by the PEAR Team, in consultation with individuals representing CBO’s and supported the State Health Report development
- Reviewed what the Board does, and how that work is done
- Looked at equity-focused tasks that are currently being implemented
- Found equity gaps in activities or areas that need more work
- Grouped gaps into buckets, which informed goals and objectives

Drafting PEAR Plan

- The PEAR Team pulled Goals and Objectives from the equity impact assessment
    - Proposed actions for each step were reviewed

- Draft is a living document, with members of the team contributing to the language
    - Discussions occur during the PEAR Team meeting

- Hoping for feedback
    - Community feedback in August and September
    - Board feedback at anytime

PEAR Themes

- Increase community access to Board meetings
- Create pathways for equitable policy and rule development
- Build and maintain community and Tribal relationships
- Enhance opportunities for DEI training and professional development

DRAFT PLAN DISCUSSION

Thoughts, Feedback, and Questions

WASHINGTON STATE BOARD OF HEALTH

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WASHINGTON STATE BOARD OF HEALTH

DRAFT

Washington State Board of Health

Pro-Equity Anti-Racism

Strategic Action Plan

October 2024

Report Authors

Paj Nandi, Sponsor, Board of Health

Ashley Bell, Equity and Engagement Manager, Board Staff

Pro-Equity Anti-Racism Plan

Table of Contents

Washington State Board of Health

Statement on Pro-Equity, Anti-Racism

In 2022, Governor Jay Inslee issued Executive Order 22-04, which directs state agencies to implement the Washington State Pro-Equity Anti-Racism Plan and Playbook. The PEAR strategic plan intends to drive systemic change and promote equity by investing where needs are the greatest to address upstream, root cause issues that perpetuate systemic inequities.

The Washington State Board of Health (Board) wants to enable all people in Washington to flourish and thrive. This plan lays out actions the State Board of Health will take to create meaningful impact to the determinants of equity. By investing in action steps and goals, we can reduce health inequities in the State of Washington. Health inequities are differences in health outcomes that are unfair, unjust, and avoidable. Most differences in health are due to inequities, a result of a lack of access to resources so that all can meet their full health potential. These health inequities are a result of laws and policies that perpetuated health inequities.

This strategic action plan exists to guide our work and create change for communities who are disproportionately affected by systemic inequities. It embeds equity into our decision-making, policy development, and public meetings. This strategic action plan is an evolving document, that is reviewed every year to ensure that we are following through with our commitments, continuing to assess our equity impact, making informed investments, being transparent and accountable, and shifting practices as necessary.

Place holder for statement the Board develops.

All Board members can sign the statement at the time of plan approval

Contact Information

Ashley Bell

Equity and Engagement Manager

Washington State Board of Health

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360-800-7481

Website| Facebook |"X" *formerly Twitter*

Accessibility and the Americans With Disabilities Act (ADA)

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For more information or additional copies of this report, contact Ashley Bell, or Board of Health Staff at wsboh@sboh.wa.gov.

Pro-Equity Anti-Racism

Team Members

Agency Executive Leadership

Patty Hayes, Board Chair, Board of Health

Michelle Davis, Executive Director, Board of Health

Board Members

Paj Nandi, Sponsor and Board Member, Board of Health

Agency Equity Officer

Ashley Bell, Equity and Engagement Manager, Board of Health Staff

Agency Employees

Shay Bauman, Policy Advisor, Board of Health Staff

Heather Carawan, Communications, Board of Health Staff

Molly Dinardo, Policy Advisor, Board of Health Staff

Hannah Haag, Community Outreach Coordinator, Board of Health Staff

Melanie Hisaw, Executive Secretary, Board of Health Staff

Jo-Ann Huynh, Administrative Assistant, Board of Health Staff

LinhPhụng Huỳnh, Health Disparities Council Manager, Board of Health Staff

Cait Lang-Perez, Health Policy Analyst, Board of Health Staff

Michelle Larson, Communications Manager, Board of Health Staff

External Partners

Amanda Shi, Research and Evaluation Manager, Tubman Center for Health & Freedom

AyeNay Abye, Tubman Center for Health & Freedom

Danisha Jefferson-Abye, Tubman Center for Health & Freedom

Tiara Ranson, Tubman Center for Health & Freedom

Johnny Buck, Tubman Center for Health & Freedom

Agency Partners

Washington State Department of Health

Office of Equity

Business Line Experts

Patty Hayes, Board Chair, Board of Health

Paj Nandi, Sponsor and Board Member, Board of Health

Michelle Davis, Executive Director, Board of Health

Ashley Bell, Equity and Engagement Manager, Board of Health Staff

Baseline Equity Impact Review

The Washington State Board of Health (Board) has completed the Baseline Equity Review of our agency's key business lines to determine where the needs are the greatest.

The PEAR Team reviewed the work that the Board completes and took an inventory of the equity work currently being done. A review of the gaps in equitable service were analyzed and put into buckets. Those buckets then informed our goals and objectives. The Baseline Equity Impact Review is available upon request.

Readiness Checklist

Agency leader and all PEAR Team members watched the recordings of the PEAR Team Orientation Session hosted by the Office of Equity.

- Dr. Johnson Welcome Video
- 2022 PEAR Team Orientation Recorded Session

Agency leader and PEAR Team members meet to debrief the PEAR Team orientation session and discuss next steps in May 2024.

The Board of Health has created a Board Pro-Equity Anti-Racism (PEAR) Statement. This has been signed by the Board of Health Chair, Board Members, and the Executive Director. This signals the Board's commitment to Pro-Equity Anti-Racism strategies.

The Board of Health Executive Director and PEAR team have created a video sharing information about the Board's PEAR statement. In addition, there is a stand-alone agency policy that outlines the Board's roles and responsibilities for implementing Executive Order 22-04.

Goal 1 Summary (Office of Equity Template)

Agency Key Business Line

Communications and Engagement

PEAR Service Line (where needs are greatest)

Engagement & Community Partnerships

Public Communications & Education

Description (Summarize the policies, processes, practices, and procedures related to this investment.

Will require policies and procedures related to access to meetings, materials, and addressing language needs.

What disparities do you seek to decrease or eliminate with this investment?

Access to government practices, information, and participation.

Which people groups and/or places, with the greatest need, does this investment focus on?

Communities at a disadvantage such as disability, education, geographic location, and language/literacy.

Did your agency consult with Tribal governments and Recognized American Indian Organizations? How does this investment address the consultation they provided?

No

What did impacted communities/employees/other interested parties identify as the root causes of the disparities? How will your investment address root causes?

Individuals feel that it is difficult to overcome barriers to participating in government forums and policy decisions. This is due to barriers, such as language access, meeting access challenges, and engagement on policy decisions. Investments in this area will focus on language access, meeting in community-based organizations spaces, and improving inclusiveness for non-regulated parties. This promotes equity in local and state practices. Systems have been in place that place barriers to participation for groups that continue to be at a disadvantage.

How does your investment address concerns and priorities identified by impacted communities/employees/other interested parties.

We will address concerns and priorities identified by ensuring that language access is present and consistent with all our written and spoken work. This will ensure our documents are presented in a way that can be better understood and increase engagement in Board activities. We will also make sure that our meeting spaces are reflective of the topics we are engaged in and the communities who may be directly impacted by our work. This will create spaces for individuals to attend our meetings and engage with our work, instead of expecting those communities to travel towards us. Finally, we will ensure that our public activities are proactively inclusive for all to attend, by focusing on providing compensation, having inclusive presentation standards, and creating space for broader public input.

PEAR Determinants of Equity supported by this investment.

Equity in State & Local Practices

PEAR Determinants of Equity Group(s) and (Community support systems (trunk), Family support systems (branches), Community infrastructure (Root system), Government Practices (Soil & nutrients) supported by this investment.

Soil & Nutrients

Root System

PEAR Habit(s) needed to achieve desired PEAR Outcomes

Making meetings, documents, and education accessible so that everyone can participate in government activities.

PEAR Service Line Investment Lead or Team (Who is responsible for leading the action.

Equity and engagement team

Collaboration needs – Who does the agency need to collaborate with to make the investment?

Collaboration with communities to determine if investment is working. Collaboration with Department of Health to meet Board accessibility standards at Board meetings. Collaboration with presenters from other agencies and organizations to meet Board accessibility standards at Board meetings.

What are the potential barriers, challenges, and/or risks of this investment?

We lack the capacity to complete a CLAS assessment. Failure to achieve these objectives can lead to barriers for community participation.

Solution(s) identified to address resource needs and barriers/challenges/risks.

Request additional funding to hire an outside contractor or consultant to complete a CLAS assessment and provide any recommendations that are needed.

PEAR Service Line Investment Start Date

10/1/2024

PEAR Service Line Investment Target End Dates

1/1/2027

PEAR Performance Measure(s) – What measures will be used to determine effectiveness of investments? Were the measures informed by impacted communities/employees/interested parties? What outcome measure was used to evaluate the effectiveness of the investment in achieving the desired PEAR outcome. What process measure used to evaluate whether investment activities have been accomplished.

CLAS assessment completion and future compliance with CLAS recommendations will be used to measure success. Guidance around plain talked presentations, documents, websites, and summaries will be created in collaboration with the executive director, equity and engagement manager, and communications manager. We will also use the amount of community compensation provided during the fiscal year, as well as increased use of compensation across all types of compensation, including reimbursements and gift cards.

The internal PEAR Team has consulted with a community-based organization on the effectiveness of this plan and its positive impact for communities and desired PEAR outcomes.

What data sources will be used to measure success? Consider data sources created by those impacted if available.

We will be using a community relationship management system to track CLAS recommendations and scores, manage community compensation, and provide data on demographics and meeting

spaces. We will also be sending responsiveness surveys to community members who participate in Board activities.

Goal 1

Create avenues for communities to participate and inform Board activities.

Objective 1

- **Ensure that language access is present and consistent in all our written and spoken work by January 2027**.
    - Complete a CLAS assessment on our public-facing communications by the end of 2025, by an outside consultant.
    - All staff will ensure that translations of primary and secondary documents are accurate and culturally appropriate according to CLAS procedures identified in our CLAS assessment, to maintain 85% or higher compliance prior to January 2027.
    - Communications will plain talk all our external-facing, public communications, such as presentations, documents, websites, and summaries, by using internal guidance documents, created by the equity and engagement team, laying out language access standards for Board work, prior to January 2026

Objective 2

- **Ensure that our meeting spaces reflect the topics we work on and communities who may be directly impacted by our work by January 2026**.
    - The equity and engagement team establish, implement, and consistently use meeting scoping procedure to ensure we are meeting in community spaces that removes barriers and promotes equity.
    - Admin will incorporate this topic into all our internal staff meeting pre and post meeting evaluation conversations, by creating a form that allows evaluation of Board meeting spaces during briefings and debriefings.
    - Outreach coordinator will support opportunities for Board members and staff to be more visible and accessible in communities, by the equity and engagement team creating guidance documents prior to January 2026.

Objective 3

- **Ensure that all public activities are proactively inclusive of impacted, non-regulated parties by January 2025**.
    - The equity and engagement manager will ensure that the community compensation process is standardized and applied broadly across all Board work by creating internal tools and providing staff training prior to January 2025.
    - The equity and engagement team will create and implement accessibility and equity standards for presenters in our meetings, such as verbal delivery and presentation standards at our Board meetings prior to January 2025.
    - The equity and engagement manager will review and make recommendations to the Board for access to public comment period and rulemaking processes, including expanded timelines to incorporate Disability Justice practices into the Board's public activities prior to July 2025.

Goal 2 Summary (Office of Equity Template)

Agency Key Business Line

Communications, Engagement, and Policy

PEAR Service Line (where needs are greatest)

Engagement & Community Partnerships

Plans, Policies, and Budgets

Policy Agenda

Public Communications & Education

Tribal Government Relations

Description (Summarize the policies, processes, practices, and procedures related to this investment.

Will require policies and procedures for community engagement, Tribal Engagement, Government to Government work, and equitable rulemaking.

What disparities do you seek to decrease or eliminate with this investment?

Decrease disparities in policy and rule development.

Which people groups and/or places, with the greatest need, does this investment focus on?

Communities at a disadvantage such as age, disability, education, experience in/with the criminal legal system, gender identity/social orientation, geographic location, housing, language/literacy, national origin, race/ethnicity, and socio-economic status.

Did your agency consult with Tribal governments and Recognized American Indian Organizations? How does this investment address the consultation they provided?

No, but will work in collaboration with Tribes will completing objectives and for future strategic plan iterations.

What did impacted communities/employees/other interested parties identify as the root causes of the disparities? How will your investment address root causes?

Community members shared the importance of connecting with the Board and the work that the Board does. We lack visibility in communities and technical communications do not help bridge the gap, although we are a public forum. By ensuring that we build and maintain relationships, are inclusive in rulemaking, and are proactive in meeting Tribes and communities where they are, we can strengthen relationships.

How does your investment address concerns and priorities identified by impacted communities/employees/other interested parties.

Our investments will bring community voice to the table in our rulemaking process. Because of our focus on developing new and on-going relationships, we will be able to better identify community groups that may want to be present at our Board activities. Having a Tribal engagement and procedure guide will improve our connections with Tribes and facilitate the meaningful information reaching Tribal Leaders.

PEAR Determinants of Equity supported by this investment.

Community & Public Safety

Equity in State & Local Practices

Healthy Built & Natural Environments

Health & Human Services

Housing & Home Ownership

Parks, Recreation & Natural Resources

PEAR Determinants of Equity Group(s) and (Community support systems (trunk), Family support systems (branches), Community infrastructure (Root system), Government Practices (Soil & nutrients) supported by this investment.

Soil & Nutrients

Trunk

Branches

PEAR Habit(s) needed to achieve desired PEAR Outcomes

Foster and maintain relationships so that Tribes and communities have a louder voice in rule and policy development.

PEAR Service Line Investment Lead or Team (Who is responsible for leading the action.

Equity and engagement team

Executive Director

Collaboration needs – Who does the agency need to collaborate with to make the investment?

Collaboration with communities to determine if investment is working.

Collaboration with Department of Health for rules process.

What are the potential barriers, challenges, and/or risks of this investment?

Rules follow the APA standards, which may limit community co-creation, which is what the community wants to maintain positive relationships.

Solution(s) identified to address resource needs and barriers/challenges/risks.

Find creative ways to create equitable policy and rulemaking that can maintain relationships with Tribes and communities. A responsiveness feedback survey can help with this.

PEAR Service Line Investment Start Date

10/1/2024

PEAR Service Line Investment Target End Dates

1/1/2027

PEAR Performance Measure(s) – What measures will be used to determine effectiveness of investments? Were the measures informed by impacted communities/employees/interested parties? What outcome measure was used to evaluate the effectiveness of the investment in achieving the desired PEAR outcome. What process measure used to evaluate whether investment activities have been accomplished.

The Board will use the completion of guidance documents as part of the performance indicators. Without a foundation, additional investments cannot be made. During our technical advisory groups (TAC), we will be able to review the demographic data of our participants, ensuring that we have included key messengers in communities in our TACs. We will also monitor our engagement database, and track new relationships, looking for a percentage increase in relationships each year.

Additionally, during our update of our PEAR plan, we will be able to listen to community feedback on changes in engagement, participation, and outreach.

What data sources will be used to measure success? Consider data sources created by those impacted if available.

We will be using a community relationship management system to receive data on outreach, participation, and engagement. Social media can also provide us with post engagement information. We will also be sending responsiveness surveys to community members who participate in Board activities.

Goal 2

Build relationships with Tribes, community-based organizations, and impacted community members.

Objective 1

- **Center community partnerships during rule development by July 2025**.
    - Board staff will review rulemaking policies and procedures with an equity lens to ensure they are creating equitable, accessible opportunities for participation.
    - The equity and engagement team will develop a review tool in partnership with impacted communities to assess draft rules language for likely equity impacts.
    - Coordinate with community engagement staff to ensure people with direct lived experience are included equitably on our Technical Advisory Committees (TACs) and other rulemaking activities.

Objective 2

- **Develop new and ongoing relationships with communities who are currently, and have been historically marginalized and oppressed, by January 2026**.
    - The equity and engagement team will create and maintain a community engagement database to coordinate engagement with community across all Board staff by January 2025.
    - All Board staff will engage with community-based organizations and other trusted messengers prior to all Board activities, through the use of social media, emails, community events, and other avenues. Guidance will be provided to staff by the outreach coordinator by July 2026.
    - The equity and engagement team will create opportunities for Board members to interact with and build relationships with communities, including community panels at Board meetings, and document a process by January 2027.

Objective 3

- **Build stronger ties with sovereign Tribes, Tribal organizations, and Tribal communities by July 2025**.
    - The Tribal Liaison will create a Tribal engagement plan, which centers Tribal sovereignty and government-to-government relationship, for the Board by July 2025.
    - The Tribal Liaison will provide guidance to staff and Board members around the Board's Tribal engagement procedures and processes by July 2025.
    - Board staff will provide quarterly updates to Tribal partners that are intentional and meaningful, as identified by the Tribes, by July 2025.

Goal 3 Summary (Office of Equity Template)

Agency Key Business Line

Human Resources and Professional Development

PEAR Service Line (where needs are greatest)

Leadership, Operations, & Services

Workforce Equity

Capacity Building

Description (Summarize the policies, processes, practices, and procedures related to this investment.

Invest in professional development for staff and Board members. Will require a review of internal hiring practice that includes recommendations for additional job postings and outreach.

What disparities do you seek to decrease or eliminate with this investment?

Decrease workforce equity disparities and increase engagement knowledge, skills, and abilities among all staff.

Which people groups and/or places, with the greatest need, does this investment focus on?

Potential workforce, Board Members, and Board Staff.

Did your agency consult with Tribal governments and Recognized American Indian Organizations? How does this investment address the consultation they provided?

No

What did impacted communities/employees/other interested parties identify as the root causes of the disparities? How will your investment address root causes?

Board staff were consulted, and they stated that they lack training and professional development that centers equity. Many of the inequities that persist are due to lack of training and better methodologies for completing work. Communities shared that engaging with Board staff can be a scary process. By investing in community relationship training, Board members can be present in the community in ways that do not perpetuate harm.

How does your investment address concerns and priorities identified by impacted communities/employees/other interested parties.

By providing education and training around equity and engagement type activities, staff will be better prepared to work with groups that are currently and have been historically marginalized. By providing engagement training to Board members, they will be able to connect with community and Tribes in ways that do not perpetuate harm. Guidance for hiring practices will increase the number of applicants with diverse backgrounds.

PEAR Determinants of Equity supported by this investment.

Equity in Jobs & Job Training

PEAR Determinants of Equity Group(s) and (Community support systems (trunk), Family support systems (branches), Community infrastructure (Root system), Government Practices (Soil & nutrients) supported by this investment.

Root System

PEAR Habit(s) needed to achieve desired PEAR Outcomes

Provide training opportunities to staff and Board Members. Reevaluate hiring practices.

PEAR Service Line Investment Lead or Team (Who is responsible for leading the action.

Equity and Engagement Manager

Executive Director

Collaboration needs – Who does the agency need to collaborate with to make the investment?

Collaboration with communities to determine if investment is working.

Collaboration with Department of Health for human resources guidance and policies.

What are the potential barriers, challenges, and/or risks of this investment?

The Board of Health has a memorandum of understanding with the Department of Health for recruitment, hiring, and other human resource needs.

Solution(s) identified to address resource needs and barriers/challenges/risks.

Look at recruitment needs for the Board of Health and add additional recruitment requirements. Provide training in recruitment and other training as needed.

PEAR Service Line Investment Start Date

10/1/2024

PEAR Service Line Investment Target End Dates

1/1/2026

PEAR Performance Measure(s) – What measures will be used to determine effectiveness of investments? Were the measures informed by impacted communities/employees/interested parties? What outcome measure was used to evaluate the effectiveness of the investment in achieving the desired PEAR outcome. What process measure used to evaluate whether investment activities have been accomplished.

The Board will use the completion of guidance documents as part of the performance indicators. Without a foundation, additional investments cannot be made.

What data sources will be used to measure success? Consider data sources created by those impacted if available.

We will provide learning and growth surveys at the end of training and professional development opportunities for both Board members and Board staff.

Goal 3

Ensure that hiring and professional development activities increase Board and Board staff understanding of equity principles by January 2027.

Objective 1

- **Provide opportunities for candidates from diverse backgrounds to have the same opportunities to obtain work at the Board of Health by January 2027.**
    - The Executive Director, or designee, will document recruitment processes that include employment outreach beyond traditional avenues prior to January 2027
    - The Executive Director, or designee, will document will write guidance around hiring processes that work to remove biases, including intersectionality on the hiring panel, by January 2027

Objective 2

- **Invest in Board staff professional development by providing equity-centered education and training by January 2027.**
    - The equity and engagement manager will provide or arrange quarterly training on anti-bias, cultural humility, pro-equity and anti-racism by
    - The equity and engagement team will ensure that Board members and staff are visible in communities in ways that do not perpetuate harm, by providing training and support to both Board members and staff.

Desired Outcomes

Write what the overall desired outcomes are for this plan and the key service lines presented.

WASHINGTON STATE BOARD OF HEALTH

Date: August 7, 2024

To: Washington State Board of Health Members

From: Kelly Oshiro, Board Member

Subject: Briefing – Newborn Screening Rulemaking for Ornithine Transcarbamylase Deficiency (OTCD), Guanidinoacetate Methyltransferase Deficiency (GAMT), and Arginase 1 Deficiency (ARG1-D) – Chapter 246-650 WAC

Background and Summary:

The Washington State Board of Health (Board) has the authority under RCW 70.82.050 to define and adopt rules for screening of Washington-born infants for hereditary conditions. WAC 246-650-010 defines the conditions and WAC 246-650-020 lists conditions for which all Washington-born newborns are to be screened. The Board convenes a technical advisory committee (TAC) in order to determine which conditions to include in the newborn screen (NBS) panel. The TAC evaluates candidate conditions using an established set of criteria. The Board directed Board and the Department of Health (Department) staff to convene technical advisory committees (TAC) to evaluate three conditions for possible inclusion in the NBS panel: Ornithine Transcarbamylase Deficiency (OTCD), Guanidinoacetate methyltransferase (GAMT) Deficiency, and Arginase 1 Deficiency (ARG1-D). The TACs are tasked with evaluating the conditions against the Board's five screening criteria.

The OTCD TAC convened June 16 and July 7, 2021and voted to recommend the inclusion of OTCD in the NBS panel, and the Board approved this recommendation at its October 2021 meeting. Board staff filed a CR-101 on February 4, 2022, to initiate the rulemaking process.

On September 8, 2023, the TACs met and evaluated GAMT and ARG1-D. The TACs voted to recommend the inclusion of GAMT and ARG1-D in the NBS panel. On October 9, 2023, staff presented the findings and recommendation of the TAC to the Board, at which time the Board approved a motion to initiate rulemaking for the inclusion of both GAMT and ARG1-D to the NBS panel. Board staff filed a CR-101 on November 28, 2023, to initiate the rulemaking process.

The inclusion of OTCD, GAMT, and ARG1-D in the NBS panel, as indicated in the Department's cost-benefit analysis, will require an increase to the NBS fee. This fee is collected for each infant in Washington State and covers the costs of the screening, follow-up services, and coordination with specialty services.

(continued on the next page)

I have invited Dr. John Thompson, Director of the Newborn Screening Program at the Department of Health, and Kelly Kramer, Policy Advisor to the Board, to provide an update on the current progress of the rule update ahead of an anticipated rules hearing in October.

Staff

Kelly Kramer

Hearing Briefing: Newborn Screening

Chapter 246-650 WAC

Adding OTCD, GAMT, and ARG1-D

Kelly Kramer, Policy Advisor - August 7, 2024

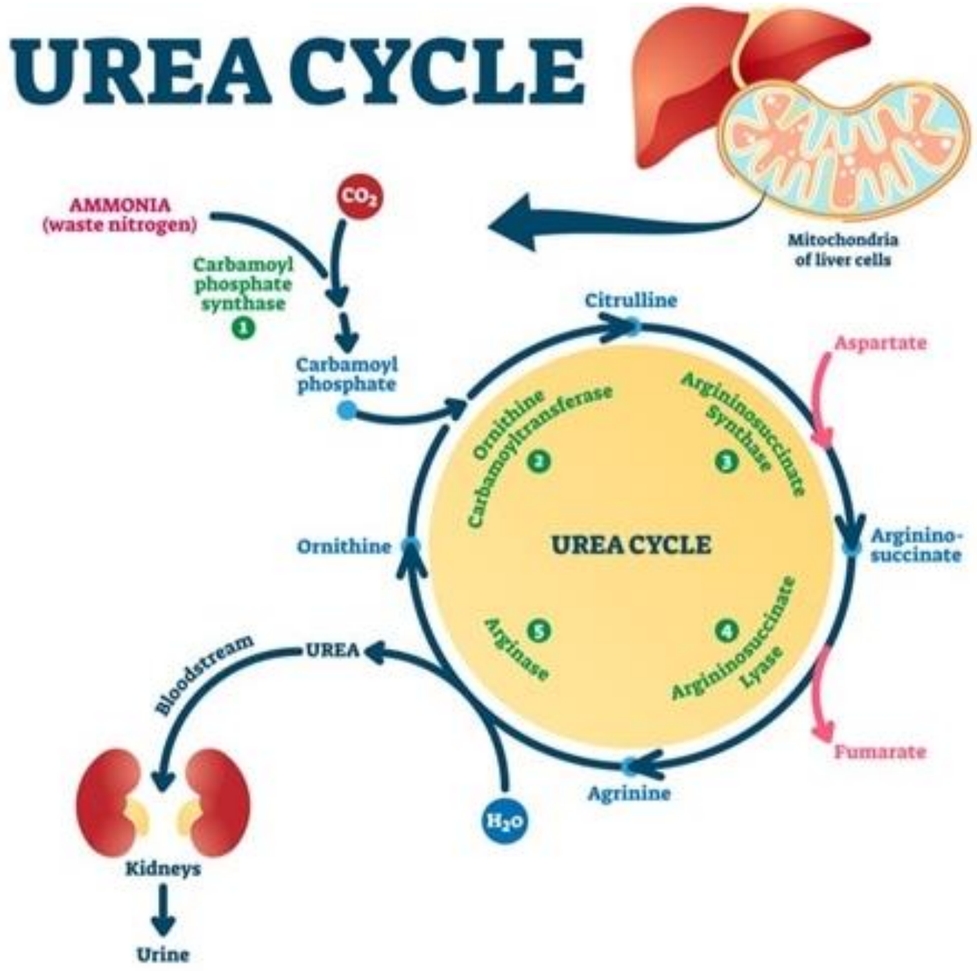
WASHINGTON STATE BOARD OF HEALTH

Briefing Agenda

- Condition Overviews
- Rulemaking Progress
- Proposed Rule Changes
- Next Steps and Timeline

Ornithine Transcarbamylase Deficiency (OTCD)

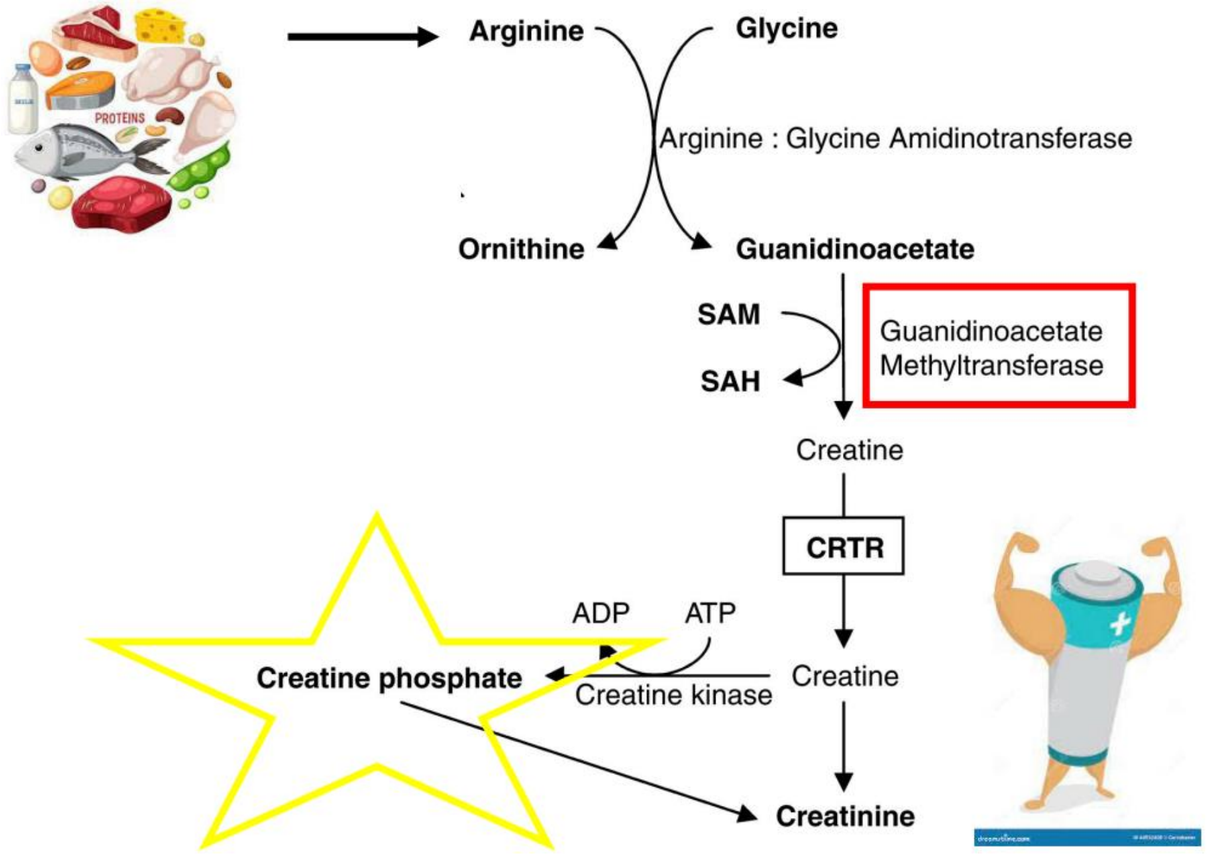
- Inherited urea cycle disorder.
- X-linked recessive inheritance pattern (more likely to affect male babies).
- Prevalence: ~1:56,000.
- Interferes with the body's ability to process ammonia (or nitrogen waste).
- High levels of ammonia are toxic to the brain and nervous system, and can lead to:
  - Tiredness/sluggishness
  - Sepsis-like symptoms
  - Comas
    - Developmental delays
    - Death



Source: News Medical, The Urea Cycle Step-by-Step

Guanidinoacetate Methyltransferase (GAMT) Deficiency

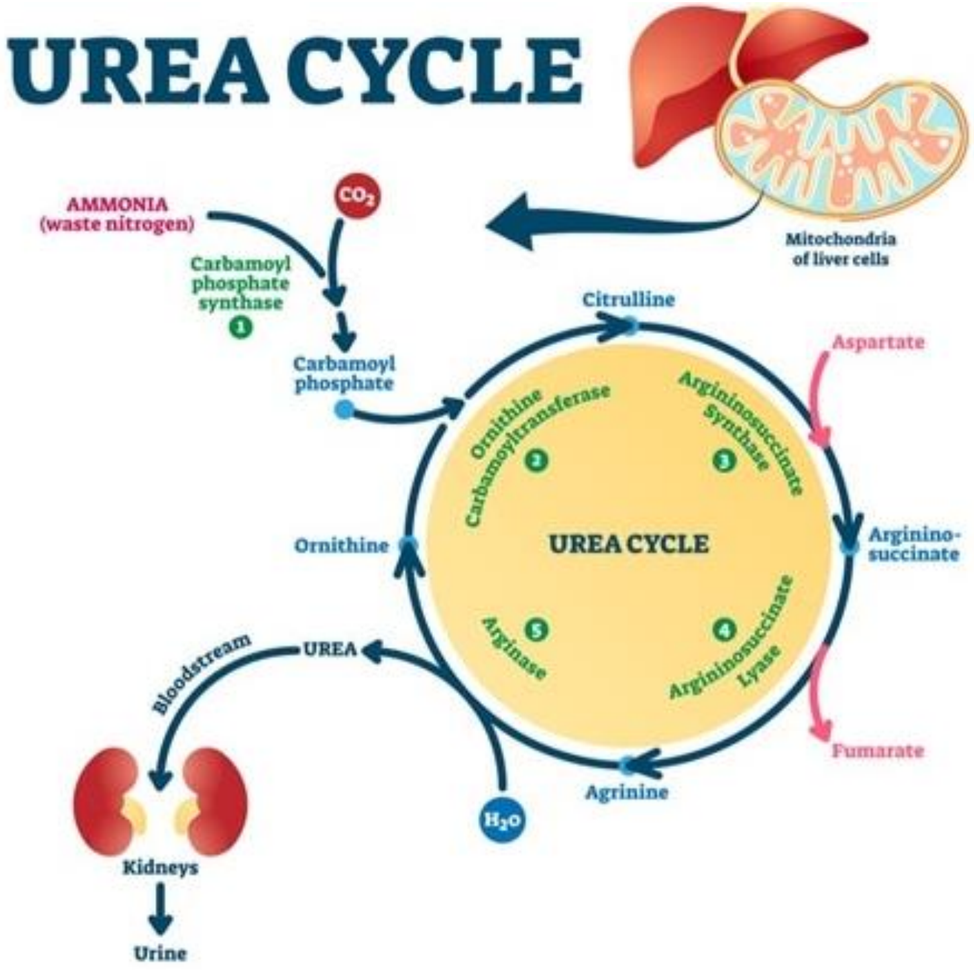
- Inherited disorder that prevents the body from metabolizing and transporting creatine.
- Autosomal recessive inheritance pattern (affects male and female babies equally).
- Prevalence: About 130 individuals diagnosed worldwide.
- Signs and symptoms of the condition vary; can start anywhere from 3 months to 2 years of age.
- If untreated, GAMT Deficiency leads to developmental delays and cognitive impairments (can be moderate or severe).



Source: Shelkowitz, E. GAMT Deficiency: Natural History, Diagnostic Testing and Treatment. SBOH NBS TAC Meeting. September 2023.

Arginase 1 Deficiency (ARG1-D)

- Inherited urea cycle disorder that causes the amino acid arginine and ammonia to accumulate in the blood.
- Autosomal recessive inheritance pattern (affects male and female babies equally).
- Prevalence: Less than 260 individuals diagnosed worldwide.
- Symptoms start off subtle and do not become apparent until early childhood (1-3 years of age). May include:
  - Spasticity in lower extremities (most common, affects 80 -90% of those diagnosed)
  - Slowed cognitive development
  - Seizures
  - Stunted growth
  - Challenges with eating
  - Liver problems



Source: News Medical, The Urea Cycle Step-by-Step

Screening and Treatment Available

Screening

- All three conditions can be screened using tandem mass spectrometry technology (MS/MS).
  - Uses one 1/8” hole punch from a dried blood spot to test for 19 congenital disorders at the same time.
  - Technology used in the WA Newborn Screening Program since 2004.

OTCD Treatment

- Emergency treatment of high blood ammonia levels
- Dietary modifications
- Liver transplant

GAMT Treatment

- Dietary modifications and oral supplements (creatine, sodium benzoate, and/or ornithine).
- Physical, occupational, speech, and behavioral therapy.

ARG1-D Treatment

- Dietary modifications and oral supplements.
- Ammonia diversion therapy: nitrogen scavenging medications (sodium benzoate, sodium phenylbutyrate).
- Referrals to neurology and physical, occupational, and/or speech therapy.

Rulemaking Progress - OTCD

- In 2020, the Board approved the Department of Health (Department) to form a Technical Advisory Committee (TAC).

- June and July 2021, the TAC convened to review the condition.
  - The TAC voted to recommend the inclusion of OTCD to the NBS panel.

- October 2021, Board staff presented TAC recommendations to the Board.
  - Approved motion to initiate rulemaking

- February 2022, CR-101 filed.

- Legislature did not fund the Department's requests in the 2022 and 2023 sessions for funding to increase the NBS fee.
  - Rulemaking to add OTCD has been put on hold since.

Rulemaking Progress – GAMT and ARG1-D

- March and April 2023 Board meetings, the Board approved Board staff and the Department to convene a TAC.
- September 2023, the TAC convened to review the conditions for inclusion on the newborn screening panel.
  - The Technical Advisory Committee voted to recommend the inclusion of GAMT and ARG1-D to the panel.
- October 2023, Board staff presented Technical Advisory Committee recommendations to the Board.
  - The Board approved a motion to initiate rulemaking for the inclusion of GAMT and ARG1-D to the panel.
- November 2023, Board staff filed CR-101.

Newborn Screening Fee Increase

- Estimate for all three conditions is **$1.77** per baby.
- Fee increase includes salaries/benefits for lab and follow-up, cost of reagents, and biochemical genetics consulting.

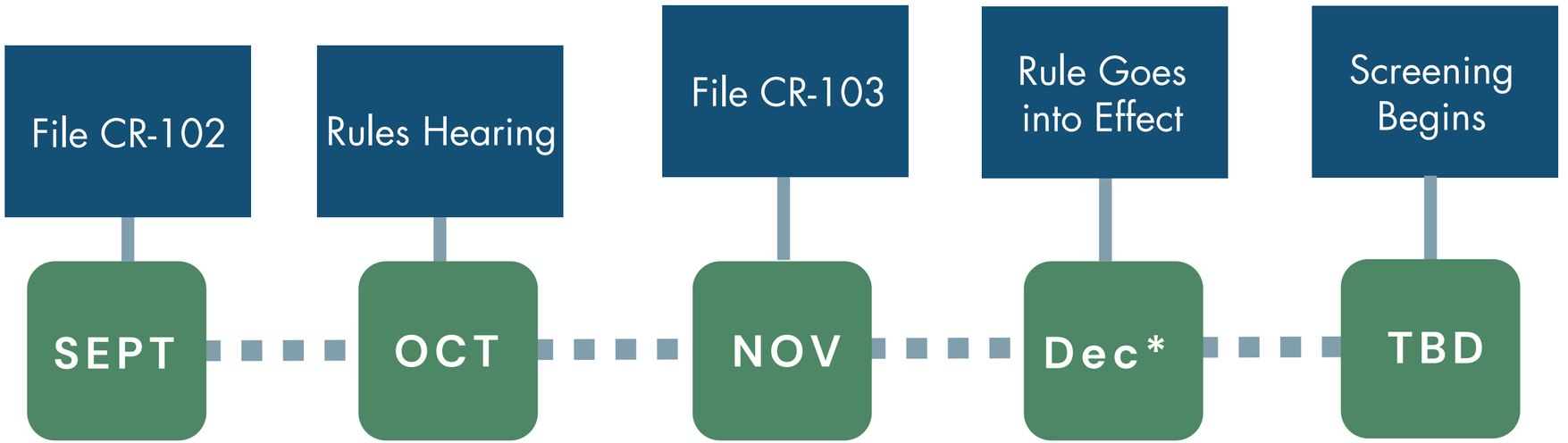
Proposed Changes:
WAC 246-650-010, Definitions

- Adding a definition Ornithine Transcarbamylase Deficiency (OTCD).
- Adding a definition Guanidinoacetate Methyltransferase Deficiency (GAMT).
- Adding a definition Arginase 1 Deficiency (ARG1-D).

Proposed Changes: WAC 246-650-020, Performance of Screening Tests

- Amending the list of newborn screenings to be performed from a dried blood specimen to include OTCD, GAMT, and ARG1-D.
- Renumber to retain alphabetical nature of the list.

Next Steps



*December or a date otherwise specified by the Department's newborn screening team. Update on screening start by the October rules hearing.

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

Washington State Department of HEALTH

Arginase 1 Deficiency (ARG1-D) Overview

ABOUT THE CONDITION

- ARG1-D is a rare and inherited metabolic disease that prevents the body from properly breaking down the amino acid arginine, an enzyme in the blood.[1,2]
- Arginase is one of six enzymes responsible for breaking down arginine and is part of an essential process in the body called the urea cycle.
- The urea cycle helps remove ammonia (or nitrogen) from the body, a waste product used to process protein.
- If the arginase enzyme isn't working properly, the body can't break down arginine and get rid of ammonia through the urea cycle.
- Irregularities in the urea cycle may cause levels of ammonia in the blood to increase.
- When ammonia levels become too high, it has toxic effects and can cause serious damage to the nervous system and other parts of the body.

SIGNS & SYMPTOMS

- Signs of ARG1-D can vary widely and may appear anytime from infancy to early childhood.
- Symptoms of ARG1-D include seizures, muscle tightness or stiffness, difficulty eating, vomiting, and trouble breathing.
- People with ARG1-D might also experience delays in both physical and cognitive development, loss of developmental milestones, and intellectual disabilities.

DIAGNOSIS

- ARG1-D can be detected through a newborn screening blood spot using tandem mass spectrometry.
- Diagnostic tests include testing for ammonia levels, amino acids, and urine organic acids (specifically orotic acid) after a positive newborn screening test.

TREATMENT

- May include a diet low in protein, special foods or formulas, eating regularly and avoiding missing meals, and medications to get rid of extra arginine and ammonia in the body.

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 at wsboh@sboh.wa.gov.

PO Box 47990, Olympia, WA 98504-7990
(360) 236-4110 • wsboh@sboh.wa.gov • sboh.wa.gov

1. Morales A, Sticco KL. Arginase Deficiency - NIH Bookshelf. In: *StatPearls*. StatPearls Publishing; 2023. Accessed August 25, 2023. http://www.ncbi.nlm.nih.gov/books/NBK482365/

2. Health Resources and Services Administration. Arginase deficiency | Newborn Screening. Updated June 2023. Accessed August 25, 2023. https://newbornscreening.hrsa.gov/conditions/arginase-deficiency

WASHINGTON STATE BOARD OF HEALTH

Washington State Department of HEALTH

Guanidinoacetate methyltransferase (GAMT) Deficiency Overview

ABOUT THE CONDITION

- GAMT Deficiency is a rare inherited amino acid disorder that prevents the body from properly producing creatine, which helps your organs store and use energy.[1,2]
- Without enough creatine, the body's organs do not get enough energy to support its vital functions, which can cause damage.
- This damage primarily affects the brain and muscles, as these organs need the most energy.
- Without early treatment, GAMT deficiency can cause serious cognitive and neurological impairments.
- GAMT deficiency is caused by changes in the GAMT gene.

SIGNS & SYMPTOMS

- People with GAMT Deficiency may begin showing symptoms from early infancy to age three.
- Signs and symptoms can vary but may include mild to severe intellectual and developmental disabilities, delayed sitting or walking, delayed or limited speech ability, muscle weakness or low muscle tone, behavioral issues (anxiety, aggression, self-injury, hyperactivity), seizures, and uncontrollable movements.

DIAGNOSIS

- GAMT Deficiency can be detected through a newborn screening blood spot using tandem mass spectrometry.
- Diagnostic tests include testing for guanidinoacetate (GUAC) and low creatine in the blood after a positive newborn screening test.
- Molecular testing can also be helpful.

TREATMENT

- May include creatine and ornithine supplements, sodium benzoate (a medication that can reduce levels of an amino acid called glycine), medications to treat seizures, a lifelong diet low in protein, and speech, occupational, and behavior therapy.

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 at wsboh@sboh.wa.gov.

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Ornithine Transcarbamylase Deficiency (OTCD) Overview

ABOUT THE CONDITION

- OTCD is an inherited metabolic disorder that prevents the body from properly removing ammonia waste.[1,2]
- OTCD is an X-linked recessive condition, meaning it affects males more than females.
- OTC is an enzyme that is part of an essential process in the body called the urea cycle. The urea cycle removes ammonia (or nitrogen) from the body.
- People with OTCD do not make enough of the OTC enzyme.
- If the OTC enzyme isn't working properly, the body can't break down proteins during the urea cycle.
- Irregularities in the urea cycle may cause levels of ammonia in the blood to increase.
- When ammonia levels become too high, it has toxic effects and can cause serious damage to the nervous system and other parts of the body.

SIGNS & SYMPTOMS

- Signs of OTCD can vary widely and may appear anytime from infancy through adulthood.
- Symptoms of OTCD include seizures, floppy arms and legs (hypotonia), lack of energy, difficulty eating, vomiting, and trouble breathing.
- People with OTCD might also experience neurological and behavioral symptoms, intellectual disabilities, coma, or even death.

DIAGNOSIS

- OTCD can be detected through a newborn screening blood spot using tandem mass spectrometry.
- Diagnostic tests include testing for ammonia levels, amino acids, and urine organic acids (specifically orotic acid) after a positive newborn screening test.

TREATMENT

- May include a diet low in protein, special foods or formulas, amino acid supplements, medications and dialysis to get rid of ammonia in the body, and liver transplant.

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RCW 70.83.020

Screening tests of newborn infants.

(1) It shall be the duty of the department of health to require screening tests of all newborn infants born in any setting. Each hospital or health care provider attending a birth outside of a hospital shall collect and submit a sample blood specimen for all newborns no more than forty-eight hours following birth. The department of health shall conduct screening tests of samples for the detection of phenylketonuria and other heritable or metabolic disorders leading to intellectual disabilities or physical defects as defined by the state board of health: PROVIDED, That no such tests shall be given to any newborn infant whose parents or guardian object thereto on the grounds that such tests conflict with their religious tenets and practices.

(2) The sample required in subsection (1) of this section must be received by the department [of health] within seventy-two hours of the collection of the sample, excluding any day that the Washington state public health laboratory is closed.

[**2014 c 18 § 1**; **2010 c 94 § 18**; **1991 c 3 § 348**; 1975-'76 2nd ex.s. c 27 § 1; **1967 c 82 § 2**.]

RCW 70.83.030

Report of positive test to department of health.

Laboratories, attending physicians, hospital administrators, or other persons performing or requesting the performance of tests for phenylketonuria shall report to the department of health all positive tests. The state board of health by rule shall, when it deems appropriate, require that positive tests for other heritable and metabolic disorders covered by this chapter be reported to the state department of health by such persons or agencies requesting or performing such tests.

[**1991 c 3 § 349; 1979 c 141 § 113; 1967 c 82 § 3.**]

RCW 70.83.050

Rules and regulations to be adopted by state board of health.

The state board of health shall adopt rules and regulations necessary to carry out the intent of this chapter.

[**1967 c 82 § 5.**]