Health Consultation

(AMENDED) WAITES WHARF DEVELOPMENT SITE:
POLYCYCLIC AROMATIC HYDROCARBONS AND HEAVY METALS IN SOIL

NEWPORT, NEWPORT COUNTY, RHODE ISLAND

Prepared by the
Rhode Island Department of Health

OCTOBER 18, 2021

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Office of Community Health and Hazard Assessment
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

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Newport, Newport County, Rhode Island

Prepared By:

Environmental Health Risk Assessment Program
Rhode Island Department of Health
Under Cooperative Agreement with
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

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SUMMARY

INTRODUCTION	The Rhode Island Department of Health (RIDOH) Environmental Health Risk Assessment Program (EHRAP) received a request from the Rhode Island Department of Environmental Management (DEM) to evaluate potential public health concerns related to the Waites Wharf demolition project in Newport, Rhode Island. DEM requested that EHRAP review levels of polycyclic aromatic hydrocarbons (PAHs), heavy metals, volatile organic compounds (VOCs), polychlorinated biphenyls (PCBs), and cyanide in soil samples and determine whether the levels pose a health hazard to occupational workers or nearby residents. The site of concern is the Waites Wharf development project, a planned 150-room hotel to be constructed between West Extension Street and Coddington Wharf in Newport, Rhode Island. The property is comprised of five individual lots, which were developed pre-1884 for various purposes. The lots were used for oil industry, restaurants, automotive repair, blacksmithing, and personal residences. Currently, the lots are used for a marina, a restaurant, and storage. Historic oil and gasoline spills from above- and underground storage tanks have resulted in multiple site investigations and remedial activities (Table 1). The most recent site investigation report found levels of arsenic and benzo(a)pyrene (e.g., PAH) above DEM's Industrial/Commercial Direct Exposure Criteria for soil. This health consultation addresses DEM's and the community's concerns about contaminant exposures and risks to public health at the Waites Wharf development project.
CONCLUSION 1	Accidentally swallowing or touching contaminants in Waites Wharf soil from the western property lots with historic manufacturing posed a minor public health hazard to past (1990s) occupational workers, nearby adult residents, and nearby child residents (50^{th} and 95^{th} percentile exposure scenarios; abbrev: median, max).
BASIS FOR CONCLUSION 1	In the past (1990s), people had direct contact with surface soil (0-3 inches depth) at Waites Wharf. For the western lots (155 and 268), the available data was limited to the 1993 site investigation report, which combined soil from 0-10 feet in depth into single samples. This was a significant limitation because EHRAP could not determine whether the contaminants were in surface or subsurface soil. Subsequent reports and letters suggested that the contaminants in surface soil were only present at low levels, but this statement could not be

verified using the currently available data. Surface soil is more likely to have higher lead levels because of dust deposits from lead-based paint. Furthermore, because Lots 267, 272, and 248 (eastern lots) had surface soil levels higher than subsurface soil, it was reasonable to assume this trend was consistent with Lots 155 and 268 (western lots). EHRAP assumed that contaminants were present in surface soil and that humans in the past would have been exposed to these contaminants.

The soil in the western lots (155 and 268) contained elevated levels of PAHs and lead, but not elevated levels of VOCs and PCBs. Because lead is a known environmental health hazard, these exposure scenarios also suggested increased risks for non-cancer lead-related health effects.

On-site indoor occupational workers would have had a low-level excess lifetime cancer risk from exposures to PAHs and lead at Lot 268 only (median, max exposure scenarios). At both lots, nearby adult residents would have had a low-level excess lifetime cancer risk from exposures to PAHs and lead (median, max). Low-level risk is defined as between one in one million ($1*10^{-6}$) and one in ten thousand ($1*10^{-4}$), but the EPA's Superfund guidance (OSWER Directive 9355.0-30) states that "Where cumulative carcinogenic site risk to an individual… is less than $1.0*10^{-4}$, … action generally is not warranted unless there are adverse environmental impacts."

At Lot 155, nearby child residents would have had a low-level excess lifetime cancer risk from exposures to PAHs and lead (median, max). At Lot 268, nearby child residents would have had a low-level excess lifetime cancer risk from lead-related exposures but a moderate-level risk from PAH-related exposures (median, max). Moderate-level risk is defined as between one in ten thousand ($1*10^{-4}$) and one in one thousand ($1*10^{-3}$). However, the risk was no greater than two in ten thousand ($2*10^{-4}$) at Lot 268. According to EPA Superfund guidance (OSWER Directive 9355.0-30), this meant that cleanup action may or may not be warranted and adverse health effects need to be considered alongside environmental standards (e.g., RIDEM I/C DEC soil standards).

NEXT STEPS

- Assuming contaminant levels have not decreased since the 1990s, EHRAP recommends that occupational workers and nearby residents take precautions to minimize contact with contaminated soil at the above listed Waites Wharf lots.

- EHRAP recommends at least one of the following options:
  - Additional sampling at Lots 155 and 268 (western lots) to isolate surface soil for more accurate and current exposure dose calculations, and/or

	▪ Soil encapsulation (i.e., ≥6 inches clean soil and ≥4 inches asphalt) and institutional controls as the remedial alternative, consistent with the 2020 site investigation report recommendations for Lots 267, 272, and 248 (eastern lots).
CONCLUSION 2	Accidentally swallowing or touching contaminants in Waites Wharf surface soil from the eastern property lots with historic manufacturing currently poses a minor public health hazard to past and present occupational workers, nearby adult residents, and nearby child residents (median, max).
BASIS FOR CONCLUSION 2	People who currently have direct contact with contaminated surface soil (0-2 inches depth) at Waites Wharf. At the eastern lots (267, 272, and 248), the surface soil contained elevated levels of PAHs, arsenic, lead, and chromium, but not elevated levels of VOCs and cyanide. Because lead is a known environmental health hazard, these exposure scenarios also suggested increased risks for non-cancer lead-related health effects. On-site indoor occupational workers would have a low-level excess lifetime cancer risk from exposures to PAHs, arsenic, and chromium at Lot 267 only (median, max). Nearby adult residents would have a low-level excess lifetime cancer risk from exposures to PAHs (Lots 267, 272, and 248), arsenic (Lots 267, 272, 248), and chromium (Lot 267) (median, max). Low-level risk is defined as between one in one million ($1*10^{-6}$) and one in ten thousand ($1*10^{-4}$). Again, EPA states that "Where cumulative carcinogenic site risk to an individual… is less than $1.0*10^{-4}$, … action generally is not warranted unless there are adverse environmental impacts." At Lots 272 and 248, nearby child residents would have a low-level excess lifetime cancer risk from exposures to PAHs, arsenic, lead, and chromium (median, max). At Lot 267, nearby child residents would have had a low-level excess lifetime cancer risk from arsenic- and lead-related exposures (median, max) but a moderate-level risk from PAH- and chromium-related exposures (max only). Moderate-level risk is defined as between one in ten thousand ($1*10^{-4}$) and one in one thousand ($1*10^{-3}$). However, the risk was no greater than two in ten thousand ($2*10^{-4}$) at Lot 267. According to EPA Superfund guidance (OSWER Directive 9355.0-30), this meant that cleanup action may or may not be warranted and adverse health effects need to be considered alongside environmental standards (e.g., RIDEM I/C DEC soil standards).

NEXT STEPS	○ EHRAP recommends that occupational workers and nearby residents take precautions to minimize contact with contaminated soil at the above listed Waites Wharf lots. ○ EHRAP agrees with the 2020 site investigation report in recommending soil encapsulation (i.e., ≥6 inches clean soil and ≥4 inches asphalt) and institutional controls as the remedial alternative.
FOR MORE INFORMATION	If you have concerns about your health, you should contact your local health care provider. You may also contact EHRAP at carolyn.poutasse@health.ri.gov and ask about lead and PAH exposures.

BACKGROUND

History

The Rhode Island Department of Health (RIDOH) Environmental Health Risk Assessment Program (EHRAP) received a request from the Rhode Island Department of Environmental Management (DEM) to evaluate potential public health concerns related to the Waites Wharf development project, a planned 150-room hotel to be constructed between West Extension Street and Coddington Wharf in Newport, Rhode Island (the site). DEM requested that EHRAP review levels of polycyclic aromatic hydrocarbons (PAHs), heavy metals, volatile organic compounds (VOCs), polychlorinated biphenyls (PCBs), and cyanide in soil samples and determine whether the levels pose a health hazard to occupational workers or nearby residents. This health consultation was based on information provided by DEM, Sage Environmental, Inc., and various public records.

The site is located at the northwestern corner of Newport's 5th Ward at Tax Assessor's plat 32 for five parcels of property: Lots 155, 268, 267, 272, and 248 (Figure 1). Lots 155 and 268 (western lots) are located closer to the Atlantic Ocean. Lots 267, 272, and 248 (eastern lots) are located closer inland towards Thames Street (SAGE Environmental 2020). In general, the lots were developed before 1884 for various purposes, including oil industry, restaurants, automotive repair, blacksmithing, and personal residences (Table 1). The current owner of the five lots acquired the western properties in the 1980s from a real estate development company, Newport Coastal Partners.

In 2020, the owner began petitioning the City of Newport to demolish current buildings on the properties in favor of building a 150-room hotel. In May 2021, the demolition permit was approved by Newport's Planning Board (Friedrichs 2021). A separate building permit application for Waites Wharf will be required to build the proposed hotel and DEM will likely be involved in the approval process because of the previous environmental site investigations. For this health consultation, previous site investigation data was evaluated by individual lot.

Site Investigations

Western Lots

In 1993 and 1994, RI Analytical Laboratory (Triangle Environmental Division) conducted a full site investigation of Lots 155 and 268, at the request of an attorney (Letter from October 25, 1993). The report noted previous concerns about underground storage tanks (USTs) and a fuel oil spill in 1984. Investigators sampled soil up to 17 feet below ground surface to explore the area for petroleum-type material (e.g., petroleum hydrocarbons) and other contaminants (Appendix A). Although soil-gas and groundwater samples were also analyzed in 1993, this health consultation focused on contaminants in soil only (see ***Contaminant Levels in Soil***).

The majority of detected contaminants were found in the southwestern edge of the site (e.g., western edge of Lot 268) (RI Analytical Laboratories 1993). The report further determined that the adjacent industries (Newport Gas Company coal gasification plants) were not major contributors of petroleum-related contamination because of the westerly (west to east) and southerly (south to north) groundwater flow (RI Analytical Laboratories 1993).[1]

[1] The groundwater flow direction varied due to tidal influences.

A subsequent Remediation Site Investigation Report (Environmental Science Services 1994) led to various historical remedial activities (1994-1996): removing above- and underground storage tanks, emptying and sealing two tanks under the current marina building, and instituting a deed restriction. The 1994 report noted elevated levels of PAHs and lead, but not elevated levels of VOCs and PCBs, and assumed contamination was buried deep enough to prevent human exposure (see ***Contaminant Levels in Soil***). Because DEM's remedial standards of the early 1990s were limited to PCBs, the authors did not recommend removing all contaminated soil. Documents related to the tank closure and removal process can be found here (DEM Site ID 2236-LS). DEM issued a Letter of Compliance in 1996, provided the site was only used for industrial/commercial (I/C) purposes.

Eastern Lots

In 2019, Sage Environmental conducted two limited site investigations of Lots 248, 267, and 272. The investigators sampled soil (n=6) up to 7 feet below ground surface and screened soil borings for total volatile organic vapors, or soil-gas (n=6). Coal ash, previously used as urban fill material, was detected at a depth of 7-8 feet. At all three lots, PAHs and select heavy metals were detected above the DEM Direct Exposure Criteria (DEC)[2] for soil in I/C locations[3] (Table 2). The site investigation report recommended soil encapsulation (i.e., ≥6 inches clean soil and ≥4 inches asphalt) and institutional controls as the remedial alternative.

Community Health Concerns

Local community groups, such as the Newport Waterfront Alliance, have opposed the Waites Wharf development project due to concerns of traffic, noise, lack of historic preservation, and environmental contamination. Because Waites Wharf is located in the storm surge zone (Rhode Island StormTools), nearby residents worry that disturbing the coal ash and contaminated soil may lead to negative health effects during a major flooding event. Some community members question whether a soil cap would adequately prevent contaminants from being disturbed, which could subsequently wash over beaches, into Newport Harbor, and into drinking water. Others note climate change and rising sea levels could similarly disturb soil contaminants, and they cite these concerns in their requests for additional site investigations into Lots 155 and 268 (western parcels).

[2] DEM Direct Exposure Criteria (DEC) soil standards for individual contaminants were calculated from animal studies with cancer endpoints (chronic exposure), where uncertainty factors accounted for animal-to-human differences and human-to-human differences (e.g. children versus adults) (ATSDR 1995). In general, higher contaminant levels are needed for non-cancer health effects to happen compared to cancer health effects, such that it was more conservative for DEM to set regulations based on cancer endpoints. DEM DEC soil standards are separate for locations specific to Industrial/Commercial (I/C) purposes and Residential (Res) purposes.

[3] Waites Wharf currently classifies as an industrial/commercial property, and the DEM I/C DEC soil standards apply, rather than the Residential DEC soil standards. Although the residential soil standards are stricter, the I/C DEC standards are considered sufficiently protective of human health. Exposure to contaminant levels above the DEM I/C DEC soil standards does not necessarily mean that adverse health effects will occur. However, such contaminant levels would indicate that remedial actions or further investigation to quantify actual exposures may be necessary.

ATSDR EVALUATION PROCESS

The Agency for Toxic Substances and Disease Registry (ATSDR) uses a detailed process to assess the potential for adverse health effects caused by exposure to site-specific contaminants (ATSDR 2005). This process involves two separate evaluations, one for exposure and one for health effects.

The ATSDR *exposure evaluation* process has two steps: determine what hazards are at the site (*environmental data screen*) and evaluate how people may contact these hazards (*exposure pathway analysis*). ATSDR identifies contaminants of concern by comparing site-specific levels (e.g., concentration) to health-based *comparison values* (CVs) (Table 2). An ATSDR CV is a contaminant level at which adverse health effects are not expected (ATSDR 2005), based on animal studies and human epidemiological studies. Adverse health effects include both cancer and non-cancer endpoints. If a contaminant of concern is present at levels higher than the corresponding CV, then the contaminant is included in the exposure pathway analysis. However, even if a contaminant level was higher than the CV, an adverse health effect may not occur.

For the exposure pathway analysis, the following five elements must all be present for an exposure to occur (i.e., completed exposure pathway):

- Contaminant source (e.g., hazardous waste site)
- Environmental medium (e.g., soil), which the contaminant moves through
- Exposure point (e.g., soil in outdoor paved play areas), where people come in contact with a contaminated medium
- Exposure route (e.g., swallowing soil), or how people come into contact with the chemical (ingestion/swallowing, inhalation/breathing, dermal contact/touching)
- Potentially exposed population

Even if all five elements are present, an adverse health effect may not necessarily occur because the chemical concentration and the amount of contact a human has with the chemical must both be high enough for harm to occur (ATSDR 2005). If data for one or more element is unknown, then it is considered a potential exposure pathway. If one or more element is missing, then it is considered an eliminated exposure pathway.

If the initial evaluation indicated that exposure may occur, then a more in-depth analysis is conducted to consider possible public health impacts. The ATSDR *health effects evaluation* has two steps: identify site-specific exposure dose estimates and determine public health implications for contaminants of concern. This evaluation calculates whether a public health hazard exists, depending on the site-specific contaminant levels. This evaluation also calculates whether people contact highly contaminated environmental media for long enough time periods to potentially contribute to cancer health risks.

Contact with a contaminant does not necessarily result in harmful health effects. Some important factors that influence whether contact with a contaminant result in adverse health effects include:

- Dose, or how much contaminant a person is exposed to
- Duration, or how long a person is exposed to a contaminant
- Frequency, or how often a person is exposed to a contaminant
- Toxicity, or what type of damage a contaminant can cause to a person

Furthermore, different people or groups of people may respond differently to contaminant exposures. When exposed to the same concentration of a contaminant in the environment, children, the elderly, and people with weakened immune responses may have larger responses and more severe health outcomes compared to members of the general population.

All these factors are included when calculating an *exposure dose*. An exposure dose estimates the contaminant level that a person may come into contact with over time. To protect public health, ATSDR and the US Environmental Protection Agency (EPA) assume a worst-case scenario to conservatively calculate exposure doses. Estimated exposure doses were compared with ATSDR *minimum risk levels* and EPA *cancer slope factors*.

A minimum risk level (MRL) estimates the daily exposure to a contaminant below which non-cancer health outcomes are unlikely to occur (ATSDR 2005). When the calculated exposure dose is divided by a contaminant's MRL, the resulting *hazard quotient* (HQ) describes the risk of non-cancer health effects. Generally, an HQ less than 1.0 means that it is unlikely an exposed person would experience adverse non-cancer health effects, while an HQ equal to or greater than 1.0 means an increased likelihood.

A cancer slope factor (CSF) estimates the increased cancer risk from a lifetime (t=78 years) of exposure to a contaminant by ingestion or inhalation (ATSDR 2005). When the cancer-specific exposure dose is multiplied by a contaminant's CSF, the resulting *excess lifetime cancer risk* (ELCR) describes the risk of cancer health effects in excess of the "background" risk. The ELCR does not estimate the number of expected cancers. Instead, the ELCR measures the probability that a person may develop cancer sometime in their lifetime following exposure to a particular contaminant.

An ELCR below $1.0*10^{-6}$ (one in one million) is "very low" or "negligible" risk, while an ELCR between $1.0*10^{-6}$ and $1.0*10^{-4}$ (one in ten thousand) is "low" risk, and between $1.0*10^{-4}$ and $1.0*10^{-3}$ (one in one thousand) is "moderate" risk (NY Department of Health 2010). Generally, an ELCR of less than one in one million is not considered a significant public health concern. Furthermore, the EPA's Superfund guidance (OSWER Directive 9355.0-30) states that "Where cumulative carcinogenic site risk to an individual… is less than $1.0*10^{-4}$, and the non-carcinogenic hazard quotient is less than 1.0, action generally is not warranted unless there are adverse environmental impacts. The decision whether a specific risk estimate for a site/pathway around 10^{-4} (e.g., $4.0*10^{-4}$) is considered acceptable is based on site-specific conditions, including any remaining uncertainties on the nature and extent of contamination and associated risks. Therefore, EPA may consider risk estimates slightly above the $1*10^{-4}$ level to be protective."

EXPOSURE PATHWAY ANALYSIS

Contaminant Levels in Soil

Western Lots

Only the 1993-1994 pre-remediation data at Lots 155 and 268 (RI Analytical Laboratories 1993) were available for EHRAP to assess. Between the two western lots, Triangle Environmental

Division took 35 soil boring samples (RI Analytical Laboratories 1993). For each lot, the soil sample with the highest contaminant level was used for the respective CV comparison (Table 2). Groundwater and soil-gas samples were also taken, but the on-site groundwater was not expected to impact drinking water supplies and the soil-gas results reported total volatile organic vapors, rather than individual contaminants. This limited the EHRAP health consultation to examining soil samples only for potential adverse health outcomes.

The soil boring samples were taken from depths ranging between 0 and 17 feet (RI Analytical Laboratories 1993). Of the 35 samples, all were analyzed for VOCs and PCBs; nine were analyzed for PAHs; and five were analyzed for total lead.[4] At Lots 155 and 268, VOC concentrations were below the relevant CVs and not evaluated further. Unusually, the soil borings were mixed (e.g., composited) between 0-10 feet to create a single sample for the total lead and PAH analyses. As a result, elevated contaminant concentrations in composite samples cannot be attributed to either surface (0-3 inches) or subsurface (>3 inches) soil.[5] This was a major limitation because EHRAP was not able to determine contaminant depth based on the composited samples (RI Analytical Laboratories 1993).

Subsequent DEM communications with the Waites Wharf owner and legal representatives suggested that surface soil samples from Lots 155 and 268 had low to negligible concentrations of heavy metals and total petroleum hydrocarbons (Martella 1995; O'Connor 1996), but this statement could not be verified using data currently available (RI Analytical Laboratories 1993). In fact, surface soil is more likely to have higher lead levels because of dust deposits from lead-based paint (Mielkel and Reagan 1998; ATSDR 2007b; California OEHHA, n.d.). Furthermore, because Lots 267, 272, and 248 (eastern lots) had surface soil contaminant levels higher than subsurface soil, it was reasonable to assume this trend was similar in Lots 155 and 268 (western lots). As such, EHRAP assumed that the contaminants were concentrated only in surface soil and that humans in the past would have been exposed to these contaminants.

Soil samples from Lot 155 (Table 2) contained maximum concentrations of total lead (n=3; 1220 mg/kg) and phenanthrene as a PAH (n=2; 10.0 mg/kg). Soil samples from Lot 268 (Table 2) contained maximum concentrations of total lead (n=2; 1994 mg/kg) and fluoranthene as a PAH (n=7; 36.0 mg/kg). The majority of contaminants in Lot 268 were located on the eastern side, adjacent to Lot 272. Exposure doses (see ***Exposure Scenarios***) were calculated for these two contaminants, with a mixture approach taken for phenanthrene and fluoranthene as PAHs (see *Polycyclic Aromatic Hydrocarbons*).

Because of the composited samples and because soil encapsulation did not occur as a remedial activity in the 1990s, data specific to surface soil would be needed to evaluate exposure pathways and potential health effects more accurately at the western lots. Alternatively, Sage Environmental could assume surface soil contamination is still occurring and consider remediation strategies without additional sampling. Soil encapsulation (i.e., ≥6 inches clean soil

[4] Total lead, rather than lead from the toxicity characteristic leaching procedure (TCLP), is typically used for ATSDR exposure dose calculations.

[5] Humans are not likely to come into contact with soils at depths greater than 3 in.

and ≥4 inches asphalt) and institutional controls are recommended at the eastern lots (SAGE Environmental 2020), and these strategies could also be applied to the western lots.

Eastern Lots
Lots 267, 272, and 248 have not undergone remediation, but as previously noted, institutional and engineered controls are currently recommended (SAGE Environmental 2020). For each lot, the soil sample with the highest contaminant level was used for the respective CV comparison (Table 2).

Between the three lots, Sage Environmental took 12 soil boring samples at depths ranging from 0-2 feet (SAGE Environmental 2020). Of the soil samples, three were exclusively analyzed for cyanide (all non-detects). The remaining nine were analyzed for heavy metals, but only six for PAHs, VOCs, and total petroleum hydrocarbons.[6] At Lots 272 and 248, concentrations of cyanide and VOCs were below the relevant CVs and not evaluated further. Subsurface soil samples (2-7 feet) were also taken, but PAH and heavy metal concentrations were similar to or lower than the corresponding surface soil sample. For this reason, only the surface soil samples were assessed for this health consultation.

Surface soil samples from Lot 267 (n=5) contained maximum concentrations of arsenic (23.1 mg/kg), lead (763 mg/kg), benzo(a)pyrene as a PAH (BaP; 12.4 mg/kg), and 1,2-dibromoethane (0.19 mg/kg) above the corresponding soil CV (Table 2). Total chromium (18.0 mg/kg) does not have an ATSDR CV, but the detected maximum concentration was larger than the EPA regional screening level (RSL) for soil (0.30 mg/kg). Exposure doses (see ***Exposure Scenarios***) were calculated for these five contaminants, with a mixture approach taken for BaP as a PAH (see *Polycyclic Aromatic Hydrocarbons*). Levels of barium, cadmium, inorganic mercury, and VOCs, with the exception of 1,2-dibromoethane, were below the appropriate CV or RSL, and not analyzed further.

Surface soil samples from Lot 272 (n=4) contained maximum concentrations of arsenic (12.5 mg/kg), lead (284 mg/kg), chromium (11.4 mg/kg), and BaP (2.87 mg/kg) above the CV or RSL (Table 2), and exposure doses were calculated for these contaminants. Levels of barium, inorganic mercury, and VOCs were below the appropriate CV or RSL, and not analyzed further.

Surface soil samples from Lot 248 (n=3) contained maximum concentrations of arsenic (8.62 mg/kg), lead (246 mg/kg), chromium (11.6 mg/kg), and BaP (2.55 mg/kg) above the CV or RSL (Table 2), and exposure doses were calculated for these contaminants. Like Lot 272, levels of barium, inorganic mercury, and VOCs were below the appropriate CV or RSL and not analyzed further.

Contaminants of Concern
Polycyclic Aromatic Hydrocarbons
PAHs are a group of chemicals that commonly occur in the environment, and dozens of different PAHs are regularly detected in soil, air, water, food, and animals. PAHs are naturally found in petroleum but are also frequently formed by burning materials, such as wood, coal, and gasoline (ATSDR 1995). As a chemical group, PAHs are generally considered to be *semi-volatile organic*

[6] Total petroleum hydrocarbons do not have an ATSDR CV because they are a mixture of many different chemicals.

chemicals, meaning that PAH levels are often higher in soil than in air. Because burning coal and gasoline produce PAHs, soil in cities often contain many different PAHs (Bradley, Magee, and Allen 1994; Kim et al. 2019). These PAH levels are known as *background* and are often the result of human-related activities (e.g., anthropogenic sources). At Waites Wharf, the detected PAH levels were above New England background concentrations (Appendix B).

Humans can be exposed to PAHs through breathing (inhalation), eating (ingestion), and touching (dermal contact) different environmental media (i.e., air, water, food, soil). Most people are exposed by breathing air with PAHs (tobacco smoke) or eating foods with PAHs (grilling or charring). Smoke and food typically contribute more to human PAH exposures than soil ingestion (Klaassen 2013). If a person breathes in PAHs from air for long periods of time (i.e., years), PAHs are known to cause heart attacks and lung diseases, such as emphysema and asthma, among other health effects (ATSDR 1995).

The EPA has classified several PAHs as "probable human carcinogens" (ATSDR 1995). Long-term exposure to high PAH levels, especially in tobacco smoke and food, is known to cause lung cancer, colon cancer, bladder cancer, and a variety of other cancers.

Of the detected PAHs, BaP is the most studied. When studying adverse health endpoints, other PAHs are often compared to BaP for relative toxicity (Nisbet and Lagoy 1992). Because multiple PAHs were detected in Waites Wharf soil, EHRAP evaluated the overall toxicity of the PAH mixture. Each PAH concentration was multiplied by a BaP Toxic Equivalency Factor (Table 3), which determined what the equivalent PAH level would be if it were BaP (Nisbet and Lagoy 1992). The sum of all BaP equivalents at each lot (Table 3; $155_{totalPAHs}$=5.65 mg/kg; $268_{totalPAHs}$=31.8 mg/kg; $267_{totalPAHs}$=20.4 mg/kg; $272_{totalPAHs}$=4.43 mg/kg; $248_{totalPAHs}$=4.04 mg/kg) was used to assess the potential health risks of the PAH mixtures.

Lead

Lead is a naturally occurring metal in soil, air, water, and other environmental media. Lead has been used as an alloy with other metals in pipes, automotive batteries, weights, ammunition, cable covers, and radiation shields (Klaassen 2013). Since with 1970s, lead has been banned for most uses (e.g., paints, caulking, ceramic), although US leaded gasoline was not phased out until the 1980s and only banned in 1996. The presence of lead in soil likely reflects past use in paint and gasoline because lead does not break down and remains in the environment following use (ATSDR 2007b; Klaassen 2013).

Lead exposures most commonly occur by inhaling dust, drinking contaminated water, or eating contaminated food, paint chips, or soil (Mielkel and Reagan 1998). Touching lead-containing products is not a major exposure route because lead is not easily absorbed through the skin (ATSDR 2007b). After eating contaminated materials, children absorb more lead into their bodies compared to adults (Klaassen 2013), putting them at greater risk for developing adverse health effects.

Environmental lead exposures are a public health hazard, with known effects on the nervous system, kidneys, liver, cardiovascular system, gastrointestinal system, reproductive system, and red blood cells (ATSDR 2007b). Among children, very low blood lead levels have been shown

to permanently affect IQ, memory, speech, and hearing (ATSDR 2007b; Klaassen 2013). Among adults, similarly low blood lead levels have been associated with high blood pressure and kidney damage (Klaassen 2013). As a result, no safe blood lead level has been identified.

Lead is expected to be a probable human carcinogen, with potential links to cancers of the kidney, lung, and brain (Klaassen 2013; ATSDR 2007b). The California EPA's Office of Environmental Health Hazard Assessment published a cancer oral slope factor of $8.5*10^{-3}$ mg/kg/day for lead compounds (California OEHHA, n.d.), which was used for calculations in this health consultation. However, scientists are more concerned about non-cancer than cancer health effects from lead because no safe blood lead level has ever been identified for neurological effects (ATSDR 2007b; Klaassen 2013). EHRAP considered non-cancer health effects from lead to be cause for concern at all five Waites Wharf lots.

Arsenic

Arsenic is a metalloid that naturally occurs in the environment and is widely distributed in soil, air, water, and other environmental media. Historically, arsenic compounds were used in wood preservatives and commercial pesticides (ATSDR 2007a), and arsenic is regularly detected in Rhode Island soil because of those uses (O'Connor 1998). EPA has classified arsenic as a probable human carcinogen because studies indicate that exposure to arsenic in drinking water was associated with increased risk of lung, skin, and bladder cancer (ATSDR 2007a). Long-term arsenic exposures have also been associated with skin darkening (hyperpigmentation), nerve damage (peripheral neuropathy, encephalopathy), high blood pressure, and cardiovascular disease (ATSDR 2007a). Because soil levels were above DEM's I/C DEC regulations (7.0 mg/kg) at Lots 267, 272, and 248, arsenic was included in the exposure pathway assessment. Soil from Lots 155 and 268 were not assessed for arsenic.

Chromium

Chromium is a common metal found in soil, water, plants, and animals. Chromium has been used for wood preservatives, leather tanning, stainless steel cookware, and metal-on-metal hip replacements (Wilbur 2000). Of the different valence states, chromium(VI), or hexavalent chromium, is more toxic than chromium(III), trivalent chromium. Chromium(VI) is most commonly linked to industrial sources, and there is strong evidence that occupational exposures are associated with lung cancer. Other associated cancers include stomach and kidney cancer (Wilbur 2000). Non-cancer gastrointestinal tract effects, such as oral ulcers, stomach pain, and diarrhea, are also associated with chromium(VI) exposures. To be conservative with the exposure dose calculations, EHRAP used the ATSDR MRL and EPA CSF for chromium(VI) in this health consultation. Because soil levels were above EPA RSL at Lots 267, 272, and 248, chromium was included in the exposure pathway assessment and EHRAP assumed that all detected chromium was chromium(VI). Soil from Lots 155 and 268 were not assessed for chromium.

1,2-Dibromoethane

1,2-dibromoethane is a colorless, man-made liquid that evaporates easily at room temperature. This VOC has historically been used as an insecticide and a leaded gasoline additive, and most applications were banned by the EPA in 1984. However, 1,2-dibromoethane can persist in groundwater and soil for years. 1,2-dibromoethane inhalation exposures have been associated

with decreased body weight and damage to the respiratory, gastrointestinal, and reproductive systems, including various cancer outcomes (e.g. probable carcinogen) (ATSDR 2018). Because soil levels were above EPA RSL at Lots 267, 1,2-dibromoethane was included in the exposure pathway assessment.

Completed Exposure Pathways

For the Waites Wharf soil samples, EHRAP considered the exposure pathways for PAHs, arsenic, lead, chromium, and 1,2-dibromoethane to be complete: contaminated surface soil (source), soil (media and exposure point), accidental ingestion (exposure route), and occupational workers (population). A second complete exposure pathway was also considered: contaminated surface soil (source), soil (media), dust blown into outdoor paved play areas (exposure point), accidental ingestion (exposure route), and nearby residents (population).

HEALTH EFFECTS EVALUATION

EHRAP quantitatively evaluated three exposure scenarios: occupational worker, nearby adult resident, and nearby child resident. The most important exposure route at Waites Wharf was accidental soil ingestion (swallowing) and accidental dermal contact (touching).

For soil ingestion, all exposure scenarios include two sets of calculations based on how much soil might be swallowed: the *central tendency exposure* (CTE, or 50th percentile exposure scenario as median soil ingestion) and *reasonable maximum exposure* (RME, or 95th percentile exposure scenario as maximum soil ingestion).

For dermal contact, the exposure dose input was the same between the CTE and RME scenarios. Inputs to the dose calculations are reported in Table 4. Health guideline values and cancer slope factors are reported in Table 5.

Exposure Scenarios

Occupational Worker

For the occupational worker exposure scenario, job tenure can vary widely by age, sex, ethnicity, and education level, with a median of 6.6 years for the working population (US EPA 2011). This exposure scenario assumed that all workers (80 kg adult) followed all personal protective equipment requirements, stayed predominantly indoors, and worked for 5 days/wk over 50 wk/y (ATSDR 2005; US EPA 2011). Based on ATSDR and EPA guidance, this health consultation used the 30 mg/day and 80 mg/day soil ingestion assumptions for the CTE (median) and RME (maximum) estimates, respectively. The estimated daily exposure doses for contaminants of concern are organized by lot, CTE, and RME in Table 6.

Nearby Resident

For evaluating the risk of health endpoints among nearby adult residents, a 50th percentile residential occupancy period of 12 years was used (US EPA 2011). The CTE exposure scenario (median) assumed that nearby adult residents (80 kg) ingested 10 mg/day for 7 days/wk for 52 weeks over 12 years (US EPA 2017). The 10 mg/day was an overestimate because nearby adults would have accidentally ingested a combination of soil from the Waites Wharf site and from their outdoor property areas. The soil ingestion may have been closer to 5 mg/day. However, based on ATSDR and EPA guidance, this health consultation used the 10 mg/day and 50 mg/day

soil ingestion assumptions as conservative CTE (median) and RME (maximum) estimates. The estimated daily exposure doses are organized by lot, CTE, and RME in Table 7.

Childhood Health Considerations
EHRAP recognizes that infants and children are at greater risk than adults from certain exposures to contamination of their water, soil, air, or food. At residential communities adjacent to Waites Wharf, children were more likely than adult residents to be exposed to higher doses: they play outdoors more frequently, have developing bodies, are smaller than adults (breathe closer to the ground), and weigh less (higher doses of chemical exposure per body weight). Children also depend on adults to identify risks, make housing decisions, and access medical care.

The CTE exposure scenario assumed that nearby babies (7.8 kg; birth to <1 y) ingested 25 mg soil per day for 7 days/wk for 52 weeks (US EPA 2017). The 25 mg/day was an overestimate because nearby babies would have accidentally ingested a combination of soil from the Waites Wharf site and from their outdoor play areas. The soil ingestion may have been closer to 10-15 mg/day for a baby. However, based on ATSDR and EPA guidance, this health consultation used the 25 mg/day and 70 mg/day soil ingestion assumptions as conservative CTE (median) and RME (maximum) estimates. The cancer-specific exposure dose assumed children stayed at a single residence from birth to 12 years old (US EPA 2017). The estimated daily exposure doses are organized by lot, CTE, and RME in Table 8.

Non-Cancer Health Effects
Using the three exposure scenarios, EHRAP calculated the risk of non-cancer health effects, or the likelihood of a health outcome other than cancer. As previously discussed (see ***Contaminants of Concern: Lead***), environmental lead exposures are known to be a public health hazard. Because no safe blood lead level has been identified, MRLs have not been developed for lead and HQs cannot be calculated. For these reasons, EHRAP conservatively concluded that increased risks for non-cancer health effects related to lead exposures at all five lots were present for occupational workers, nearby adult residents, and nearby child residents. People who accidentally swallow Waites Wharf soil from the eastern lots may be at risk of neurological damage, kidney damage, and high blood pressure (ATSDR 2007b; Klaassen 2013).

<u>CTE or Median Exposure Scenario for Non-Lead Contaminants</u>
For occupational workers, no increased risks for non-cancer health effects were found at any of the five lots (HQ<<1.0, Table 6).

For nearby adult residents, no increased risks for non-cancer health effects were found at any of the five lots (HQ<<1.0, Table 7).

For nearby child residents, no increased risks for non-cancer health effects were found at any of the five lots (HQ<1.0, Table 8).

<u>RME or Maximum Exposure Scenario for Non-Lead Contaminants</u>
For occupational workers, no increased risks for non-cancer health effects were found at any of the five lots (HQ<<1.0, Table 6).

For nearby adult residents, no increased risks for non-cancer health effects were found at any of the five lots (HQ<<1.0, Table 7).

For nearby child residents, no increased risks for non-cancer health effects were found at Lots 155, 272, and 248 (HQ<1.0, Table 8). However, at Lots 268 and 267 under with maximum soil ingestion exposure scenario (70 mg/day), HQs greater than 1.0 were calculated for total PAHs with chronic exposures (365 days or longer), indicating an increased risk for non-cancer health effects among children. The PAH MRL is based on the non-cancer endpoint of developmental toxicity (ATSDR 1995), such that children who ingest excessive amounts of Lot 268's and 267's surface soil may experience delayed developmental milestones. The remaining contaminants at Lot 267 did not demonstrate increased non-cancer health risks (HQ<1.0, Table 8).

Cancer Health Effects

Again, the *excess lifetime cancer risk* (ELCR) describes the risk of cancer health effects in excess of the "background" risk. The ELCR does not estimate the number of expected cancers in an exposed population. Rather, the ELCR measures the probability that a person may develop cancer sometime in their lifetime following an exposure. Notably, most probable carcinogens do not have a *threshold level*, or a level below which there is no risk of getting cancer. Unless data says otherwise (e.g., chloroform), every exposure to a carcinogen is assumed to be associated with some increased risk. However, EPA has stated that an increased lifetime cancer risk of one in one million or less ($<1.0*10^{-6}$) is not considered a significant public health concern (NY Department of Health 2010).

Again, the EPA's Superfund guidance (OSWER Directive 9355.0-30) states that "Where cumulative carcinogenic site risk to an individual… is less than $1.0*10^{-4}$, and the non-carcinogenic hazard quotient is less than 1.0, action generally is not warranted unless there are adverse environmental impacts. The decision whether a specific risk estimate for a site/pathway around 10^{-4} (e.g., $4.0*10^{-4}$) is considered acceptable is based on site-specific conditions, including any remaining uncertainties on the nature and extent of contamination and associated risks." Therefore, the EPA considers the $1*10^{-4}$ risk boundary to be a flexible cutoff for warranting action.

<u>CTE or Median Exposure Scenario</u>

For occupational workers, no increased risks for cancer were found at Lots 155, 267, 272, and 248 ($ELCR<1.0*10^{-6}$, Table 6). At Lot 268, there was a "low" level of increased cancer risk ($1.0*10^{-6}<ELCR<1.0*10^{-4}$) from total PAH exposures, but not from lead exposures.

For nearby adult residents, no increased risks for cancer were found at Lot 155 ($ELCR<1.0*10^{-6}$, Table 7). At Lot 268, there was a "low" level of increased cancer risk ($1.0*10^{-6}<ELCR<1.0*10^{-4}$) from total PAH exposures, but not from lead exposures. At Lot 267, there was a low level of increased cancer risk from total PAH, arsenic, and chromium exposures, but not from lead or dibromoethane exposures. At Lots 272 and 248, there was a low level of increased cancer risk from chromium exposures, but not from arsenic, lead, or total PAH exposures.

For nearby child residents, there was a "low" level of increased cancer risk ($1.0*10^{-6}<ELCR<1.0*10^{-4}$) from total PAH and lead exposures at Lot 155 (Table 8). At Lot 268, there

was a low level of increased cancer risk for lead exposures, but a "moderate" level of increased cancer risk ($1.0*10^{-4}$<ELCR<$1.0*10^{-3}$) for total PAH exposures of no more than $1.5*10^{-4}$. At Lot 267, there was a low level of increased cancer risk from total PAH, arsenic, lead, and chromium exposures, but not from dibromoethane exposures. At Lots 272 and 248, there was a low level of increased cancer risk from total PAH, arsenic, and chromium exposures, but not from lead exposures.

RME or Maximum Exposure Scenario

For occupational workers, no increased risks for cancer were found at Lots 155, 272, and 248 (ELCR<$1.0*10^{-6}$, Table 6). At Lot 268, there was a "low" level of increased cancer risk ($1.0*10^{-6}$<ELCR<$1.0*10^{-4}$) from total PAH and lead exposures. At Lot 267, there was a low level of increased cancer risk from total PAH, arsenic, and chromium exposures.

For nearby adult residents, there was a "low" level of increased cancer risk ($1.0*10^{-6}$<ELCR<$1.0*10^{-4}$, Table 7) at Lots 155 and 268 from total PAH and lead exposures. At Lot 267, there was a low level of increased cancer risk from total PAH, arsenic, and chromium exposures, but not from lead or dibromoethane exposures. At Lots 272 and 248, there was a low level of increased cancer risk from arsenic and chromium exposures, but not from lead or total PAH exposures.

For nearby child residents, there was a "low" level of increased cancer risk ($1.0*10^{-6}$<ELCR<$1.0*10^{-4}$) from total PAH and lead exposures at Lot 155 (Table 8). At Lot 268, there was a low level of increased cancer risk for lead exposures, but a "moderate" level of increased cancer risk ($1.0*10^{-4}$<ELCR<$1.0*10^{-3}$) for total PAH exposures of no more than $2.0*10^{-4}$. At Lot 267, there was a low level of increased cancer risk from arsenic and lead, a moderate level of increased risk from chromium and total PAH exposures of no more than $1.5*10^{-4}$, and no increased risk from dibromoethane exposures. At Lots 272 and 248, there was a low level of increased cancer risk from total PAH, arsenic, lead, and chromium exposures.

For these three scenarios, the level of increased cancer risk ranged from "negligible" to "moderate." Although specific risk level differed by the exposure scenario and quantity of soil ingested, the ELCRs did not exceed $2.0*10^{-4}$. According to EPA Superfund guidance (OSWER Directive 9355.0-30), this meant that cleanup action may or may not be warranted and adverse health effects need to be considered alongside environmental standards (e.g., RIDEM I/C DEC soil standards). On the conservative perspective, surface soil remediation at all five lots could reduce exposure to carcinogens. For these contaminants, specific cancers could include lung, bladder, colon (PAHs); lung, skin, and bladder (arsenic); stomach and kidney (chromium); and lung, brain, and kidney (lead) (ATSDR 1995; 2007a; Wilbur 2000; ATSDR 2007b). Remediation may be particularly useful at Lots 268 and 267, in the interest of childhood health.

Assessment of Community Concerns

Sea Level Rise

Local community members are concerned about whether sea level rise and climate change may disturb subsurface soil at Waites Wharf, even if the remedial activities include encapsulation (i.e., ≥6 inches clean soil and ≥4 inches asphalt). People have referenced the subsurface coal ash detected in Lots 267, 272, and 248 (eastern parcels) and the 1984 oil spill in Lots 155 and 268

(western parcels). To address these concerns, EHRAP qualitatively considered potential contaminant movement during a major flooding event at Waites Wharf. However, ATSDR does not typically conduct climate change sensitivity analyses, and a single qualitative analysis is not sufficient to evaluate defensible remedial alternatives. DEM may choose to request a formal sensitivity analysis before remedial activities begin.

While soil stability is site-specific, major storm events and flooding can lead to scouring[7] and soil erosion (Ziegler 2002; Heise and Förstner 2007; US EPA 2020). Scouring around buildings, bridges, and piers has been the most studied (Coulbourne 2010; Nadal et al. 2010), although there are limited methodologies capable of estimating local soil scour at a given location. Past mathematical models have typically overestimated how much soil will be swept away during a given event (Coulbourne 2010; Nadal et al. 2010). When scouring does occur, the top soil layer (i.e., younger) has to be swept away before the older soil layers can become resuspended in the floodwaters (Ziegler 2002; Heise and Förstner 2007). Previous research has shown that a rare storm event may not necessarily result in re-exposure to buried soil contaminants (Ziegler 2002).

At Waites Wharf, no surface soil remediation has occurred to date, such that a flooding event could resuspend subsurface soil contaminants and elevate surface soil contaminant concentrations. If soil encapsulation occurs, then a flooding event would be less likely to resuspend subsurface soil contaminants because this remedial alternative includes additional layers of clean soil and an asphalt cover to decrease floodwater access to subsurface soil.

EHRAP also assumed that flooding from the Atlantic Ocean would dilute on-site contaminant levels, even if subsurface soils were resuspended. In the eastern lots (267, 272, and 248), contaminant levels were relatively low (SAGE Environmental 2020), such that dilution would not be expected to increase health effect risks. This would likely be true for the western lots (155 and 268), although additional information on surface soil versus subsurface coal ash could support this conclusion.

Should DEM conduct a formal sensitivity analysis, several key questions will help define the potential for soil instability at Waites Wharf during a storm event (Ziegler 2002):

- What scour depths will be caused by the storm?
- Where will the scouring and deposition likely occur?
- What effects will scouring and resuspension have on the availability of buried contaminants?
- If the effects will be significant, what controls could result in appropriate mitigation?

Soil Inhalation During Development

Due to community concerns surrounding the hotel development project, EHRAP also evaluated potential exposures from soil inhalation. A person can be exposed by breathing in PAH-contaminated soils at a construction site, but this is less likely to lead to negative health effects than breathing in tobacco smoke (Ramírez et al. 2011). The soil inhalation health evaluation was qualitative, rather than quantitative, for several reasons: air samples were not taken at the Waites

[7] To clean or brighten the surface (of something) by rubbing it hard, typically with an abrasive, detergent, or swift-flowing water.

Wharf lots, and ATSDR does not currently have guidance for calculating health risks from soil inhalation.

Because soil inhalation is not considered a major exposure route for PAHs and heavy metals, most US states do not have regulatory standards, known as Particulate Soil Inhalation Criteria (PSIC). For this qualitative assessment, RIDOH used PSIC values from Michigan as the references (Michigan Department of Community Health 2005). All relevant values are listed in Appendix C.

For total PAHs, arsenic, chromium, and lead, the Michigan PSICs for both industrial sites and residential communities were at least 10 times larger than the maximum level detected in Waites Wharf soil. EHRAP did not expect inhalation of Waites Wharf soil to result in adverse health effects from PAHs or heavy metals among occupational workers or nearby residents.

LIMITATIONS

Several assumptions were included in this health consultation. First, the exposure scenarios are conservative estimates of accidental soil ingestion based on previous research (ATSDR 2005; US EAP 2011). These estimates rely on consistent and predictable human behavior in occupational and residential situations, but human activities can have large variability and chemical exposures are unique to each individual person. Therefore, it was most conservative to focus on the median and maximum exposure estimates. Second, soil samples were not taken at residential properties adjacent to Waites Wharf, such that the residential soil ingestion rates used in this health consultation were overestimates. Soil samples taken from outdoor play areas would be more applicable for estimating nearby residential exposures.

As previously mentioned, a major challenge was that the soil dataset (1993) from the western lots (Lots 155 and 268) were mixed between 0-10 feet to create a single sample for the total lead and PAH analyses. Because the elevated contaminant concentrations in composite samples could not be attributed to either surface (0-3 inches) or subsurface (>3 inches) soil, EHRAP was not able to determine contaminant depth and assumed that contaminants were primarily present in surface soil. Future datasets should avoid mixing soil samples from multiple depths in the interest of exposure scenario calculations.

CONCLUSIONS

At Waites Wharf, DEM asked EHRAP to evaluate data on contaminated soil for exposure potential to occupational workers and nearby residents. The combination of a long timeframe for exposures (>365 days) and multiple exposure pathways put nearby populations at risk for health effects associated with soil ingestion of PAHs and heavy metals. Based on the surface soil samples and exposure scenarios evaluated, EHRAP came to the following conclusions:

1. Accidentally swallowing or touching contaminants in Waites Wharf soil from the western property lots with historic manufacturing posed a minor public health hazard to past (1990s) occupational workers, nearby adult residents, and nearby child residents.

2. Accidentally swallowing or touching contaminants in Waites Wharf surface soil from the eastern property lots with historic manufacturing currently poses a minor public health hazard to past and present occupational workers, nearby adult residents, and nearby child residents.

RECOMMENDATIONS

Because the Waites Wharf site may soon be under development, EHRAP provided the following recommendations to DEM:

1. EHRAP recommends that occupational workers and nearby residents take precautions to minimize contact with contaminated soil at the above listed Waites Wharf lots.

2. EHRAP agrees with the 2020 site investigation report in recommending soil encapsulation (i.e., ≥6 inches clean soil and ≥4 inches asphalt) and institutional controls as the remedial alternative at Lots 267, 272, and 248 (eastern lots).

3. EHRAP recommends at least one of the following options for the western property lots:
    a. Additional soil sampling at Lots 155 and 268 (western lots) to isolate surface soil for more accurate exposure dose calculations, and/or
    b. Soil encapsulation (i.e., ≥6 inches clean soil and ≥4 inches asphalt) and institutional controls as the remedial alternative, consistent with the 2020 site investigation report recommendations for Lots 267, 272, and 248 (eastern lots).

PUBLIC HEALTH ACTION PLAN

At this time, EHRAP's public health action plan include the following items to protect the populations involved:

1. EHRAP will distribute a frequently-asked-questions sheet describing this health consultation for the Waites Wharf and what the public can do to minimize contaminant exposures.

2. Upon request, EHRAP is available to assess additional exposure scenarios and health endpoints of interest beyond what is discussed in this health consultation.

3. Should new environmental data become available for the site, EHRAP will further evaluate opportunities for human exposure.

Individuals with additional information or questions regarding this health consultation should contact the Environmental Health Risk Assessment Program, Division of Environmental Health, RIDOH at carolyn.poutasse@health.ri.gov.

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Author

Carolyn Poutasse, PhD
Environmental Health Risk Assessment Toxicologist
Division of Environmental Health
Rhode Island Department of Health

Rhode Island Department of Health Reviewers

Michael Byrns, PhD
Principal Environmental Health Risk Assessment Toxicologist
Division of Environmental Health

Seema Dixit
Director – Division of Environmental Health
Rhode Island Department of Health

Caroline Hoffman, MPH
Senior Public Health Promotion Specialist
Division of Environmental Health

Melissa Orpen-Tuz
Assistant Health Program Administrator
Division of Environmental Health

Robert Suscy, MPH
Environmental Health Risk Assessment Epidemiologist
Division of Environmental Health

Rhode Island Department of Environmental Management Reviewers

Joseph Martella II
Environmental Engineer III
Office of Land Revitalization and Sustainable Materials Management

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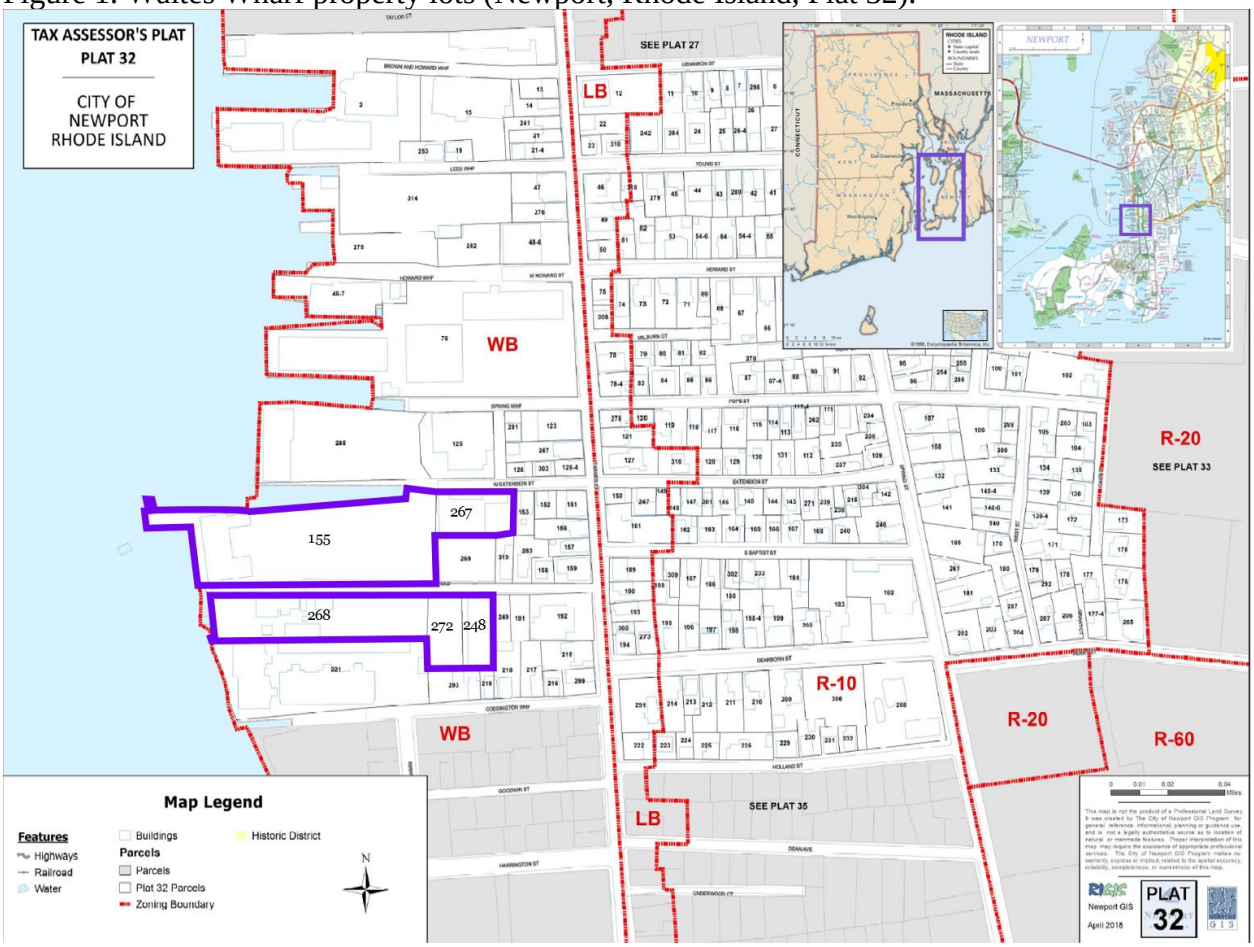
FIGURES

Figure 1. Waites Wharf property lots (Newport, Rhode Island, Plat 32).



TABLES

Table 1. Waites Wharf lot history.

Lot	Portion	Relevant History	Previous Site Investigation	Remedial Activities*
155	West	○ Pre-1883: Unknown ○ 1890s: Liquor sales, livery stables ○ 1920s: Realty office ○ 1990s: Restaurant ○ 1996: Underground storage tanks drained ○ Present Day: Marina	1993	Yes
268	West	○ Pre-1883: Storage ○ 1900s: Coal and wood yard ○ 1910s: Standard Oil of New York ○ 1916: Gasoline spill ○ 1920s: Mobil Oil (underground storage tanks present) ○ 1984: Gasoline spill ○ 1996: Underground storage tanks drained ○ Present Day: Restaurant, storage	1993	Yes
267	East	○ Pre-1883: Personal residences ○ 1950s: Automotive repairs ○ 1990s: Parking lot ○ Present Day: Storage	2020	No
272	East	○ Pre-1883: Personal residences ○ 1950s: Blacksmith/welding/metal fabricating shop, automotive repairs, and personal residences ○ Present Day: Storage	2020	No
248	East	○ Pre-1883: Personal residences ○ 1950s: Wooden Case Assembling and Painting ○ Present Day: Storage	2020	No

* DEM remedial regulations of the early 1990s were limited to PCBs, which were not detected at elevated levels in soil.

Table 2. Contaminants of concern at Waites Wharf, organized by lot number.

Contaminant*	Lot Number	Sampling Year	Sample Depth (ft) of Maximum Concentration	Maximum Concentration (mg/kg)	ATSDR Comparison Values	
					Value	Source
Metals						
Arsenic	155	1993	-	-	0.26 mg/kg	CREG^
	268	1993	-	-		
	267	2020	0-2	23.1		
	272	2020	0-2	12.5		
	248	2020	0-2	8.62		
Chromium	155	1993	-	-	0.30 mg/kg	EPA RSL^{+}
	268	1993	-	-		
	267	2020	0-2	18.0		
	272	2020	0-2	11.4		
	248	2020	0-2	11.6		
Lead	155	1993	0-10	1220	2.6 mg/kg	EPA RSL^{+}
	268	1993	0-10	1994		
	267	2020	0-2	763		
	272	2020	0-2	284		
	248	2020	0-2	246		
Mercury	155	1993	-	-	6.53 mg/kg	EPA RSL^{+}
	268	1993	-	-		
	267	2020	0-2	1.46		
	272	2020	0-2	2.80		
	248	2020	0-2	0.35		
PAHs						
2-Methylnaphthalene	155	1993	0-14	ND	*210 mg/kg*	*RMEG Child*
	268	1993	0-10	ND		
	267	2020	0-2	0.962		
	272	2020	0-2	ND		
	248	2020	0-2	ND		
Acenaphthene	155	1993	0-14	ND	*3100 mg/kg*	*RMEG Child*

	268	1993	0-10	ND		
	267	2020	0-2	3.18		
	272	2020	0-2	ND		
	248	2020	0-2	ND		
Acenaphthylene	155	1993	0-14	ND	NC	NC
	268	1993	0-10	ND		
	267	2020	0-2	0.367		
	272	2020	0-2	ND		
	248	2020	0-2	ND		
Anthracene	155	1993	0-14	ND	*16,000 mg/kg*	*RMEG Child*
	268	1993	0-10	ND		
	267	2020	0-2	6.49		
	272	2020	0-2	0.773		
	248	2020	0-2	1.44		
Benzo(a)anthracene	155	1993	0-14	ND	NC	NC
	268	1993	0-10	18		
	267	2020	0-2	16.4		
	272	2020	0-2	2.74		
	248	2020	0-2	3.27		
Benzo(a)pyrene	155	1993	0-14	ND	0.11 mg/kg	CREG
	268	1993	0-10	22		
	267	2020	0-2	12.4		
	272	2020	0-2	2.87		
	248	2020	0-2	2.55		
Benzo(b)fluoranthene	155	1993	0-14	ND	NC	NC
	268	1993	0-10	22		
	267	2020	0-2	13.3		
	272	2020	0-2	2.61		
	248	2020	0-2	2.20		
Benzo(ghi)perylene	155	1993	0-14	ND	NC	NC
	268	1993	0-10	18		
	267	2020	0-2	5.87		

	272	2020	0-2	1.79		
	248	2020	0-2	1.34		
Benzo(k)fluoranthene	155	1993	0-14	ND	NC	NC
	268	1993	0-10	21		
	267	2020	0-2	9.75		
	272	2020	0-2	2.01		
	248	2020	0-2	2.12		
Chrysene	155	1993	0-14	4.6	NC	NC
	268	1993	0-10	21		
	267	2020	0-2	15.4		
	272	2020	0-2	2.61		
	248	2020	0-2	2.91		
Dibenzo(a,h)anthracene	155	1993	0-14	ND	NC	NC
	268	1993	0-10	ND		
	267	2020	0-2	3.07		
	272	2020	0-2	0.583		
	248	2020	0-2	0.531		
Fluoranthene	155	1993	0-14	9.0	*2100 mg/kg*	*RMEG Child*
	268	1993	0-10	36		
	267	2020	0-2	35.1		
	272	2020	0-2	5.06		
	248	2020	0-2	6.32		
Fluorene	155	1993	0-14	ND	*2100 mg/kg*	*RMEG Child*
	268	1993	0-10	ND		
	267	2020	0-2	3.23		
	272	2020	0-2	0.782		
	248	2020	0-2	ND		
Indeno(123-cd)pyrene	155	1993	0-14	ND	NC	NC
	268	1993	0-10	11		
	267	2020	0-2	5.68		
	272	2020	0-2	1.72		
	248	2020	0-2	1.32		

Naphthalene	155	1993	0-14	ND	*1000 mg/kg*	*RMEG Child*
	268	1993	0-10	6.4		
	267	2020	0-2	1.45		
	272	2020	0-2	0.460		
	248	2020	0-2	ND		
Phenanthrene	155	1993	0-14	10	NC	NC
	268	1993	0-10	8.4		
	267	2020	0-2	31.1		
	272	2020	0-2	2.86		
	248	2020	0-2	3.73		
Pyrene	155	1993	0-14	7.0	*1000 mg/kg*	*RMEG Child*
	268	1993	0-10	30		
	267	2020	0-2	27.9		
	272	2020	0-2	3.96		
	248	2020	0-2	4.50		
VOCs						
1,2-Dibromoethane	155	1993	4-10	ND	0.19 mg/kg	CREG
	268	1993	4-10	ND		
	267	2020	0-2	0.19		
	272	2020	0-2	ND		
	248	2020	0-2	ND		

-: Not analyzed
ND: Not detected
*Contaminants of concern that exceeded or did not have comparison values are listed. All detected PAHs were included because they were subsequently used for the BaP TEQ calculations (Table 3).
^CREG: cancer risk evaluation guide
$^{+}$EPA RSL: EPA Regional Screening Level for carcinogenic endpoints

Table 3. PAH soil levels (mg/kg) and toxic equivalency factors.

PAH	TEF	RIDEM I/C DEC	Lot 155		Lot 268		Lot 267		Lot 272		Lot 248	
			Max	BaP_{eq}	Max	BaP_{eq}	Max	BaP_{eq}	Max	BaP_{eq}	Max	BaP_{eq}
Benzo(a)pyrene	1.0	0.8	2.3	2.3	**22**	**22**	**12.4**	**12.4**	**2.87**	**2.87**	**2.55**	**2.55**
2-Methylnaphthalene	0.001	10,000	2.3	$2.3*10^{-3}$	2.2	$2.2*10^{-3}$	**0.962**	$\mathbf{9.6*10^{-4}}$	0.208	$2.1*10^{-4}$	0.208	$2.1*10^{-4}$
Acenaphthene	0.001	10,000	2.3	$2.3*10^{-3}$	2.2	$2.2*10^{-3}$	**3.18**	$\mathbf{3.2*10^{-3}}$	0.208	$2.1*10^{-4}$	0.208	$2.1*10^{-4}$
Acenaphthylene	0.001	10,000	2.3	$2.3*10^{-3}$	2.2	$2.2*10^{-3}$	**0.367**	$\mathbf{3.7*10^{-4}}$	0.208	$2.1*10^{-4}$	0.208	$2.1*10^{-4}$
Anthracene	0.01	10,000	2.3	0.023	2.2	0.022	**6.49**	**0.065**	**0.773**	$\mathbf{7.7*10^{-3}}$	**1.44**	**0.014**
Benzo(a)anthracene	0.1	7.8	2.3	0.230	**18**	**1.8**	**16.4**	**1.64**	**2.74**	**0.274**	**3.27**	**0.327**
Benzo(b)fluoranthene	0.1	7.8	2.3	0.230	**21**	**2.1**	**13.3**	**1.33**	**2.61**	**0.261**	**2.20**	**0.220**
Benzo(ghi)perylene	0.01	10,000	2.3	0.023	**18**	**0.18**	**5.87**	**0.059**	**1.79**	**0.018**	**1.34**	**0.013**
Benzo(k)fluoranthene	0.1	78	2.3	0.230	**21**	**2.1**	**9.75**	**0.975**	**2.01**	**0.201**	**2.12**	**0.212**
Chrysene	0.01	780	**4.6**	**0.046**	**21**	**0.21**	**15.4**	**0.15**	**2.61**	**0.030**	**2.91**	**0.030**
Dibenzo(ah)anthracene	1.0	0.8	2.3	2.3	2.2	**4.4**	**3.07**	**3.07**	**0.583**	**0.583**	**0.531**	**0.531**
Fluoranthene	0.001	10,000	**9.0**	$\mathbf{9.0*10^{-3}}$	**36**	**0.036**	**35.1**	**0.035**	**5.06**	$\mathbf{5.1*10^{-3}}$	**6.32**	$\mathbf{6.3*10^{-3}}$
Fluorene	0.001	10,000	2.3	$2.3*10^{-3}$	2.2	$2.2*10^{-3}$	**3.23**	$\mathbf{3.2*10^{-3}}$	**0.782**	$\mathbf{7.8*10^{-4}}$	0.208	$2.1*10^{-4}$
Indeno(1,2,3-cd)pyrene	0.1	7.8	2.3	0.230	**11**	**1.1**	**5.68**	**0.568**	**1.72**	**0.172**	**1.32**	**0.132**
Naphthalene	0.001	10,000	2.3	$2.3*10^{-3}$	**6.4**	$\mathbf{6.4*10^{-3}}$	**1.45**	$\mathbf{1.5*10^{-3}}$	**0.460**	$\mathbf{4.6*10^{-4}}$	0.208	$2.1*10^{-4}$
Phenanthrene	0.001	10,000	**10**	**0.010**	**8.4**	$\mathbf{8.4*10^{-3}}$	**31.1**	**0.031**	**2.86**	$\mathbf{2.9*10^{-3}}$	**3.73**	$\mathbf{3.7*10^{-3}}$
Pyrene	0.001	10,000	**7.0**	$\mathbf{7.0*10^{-3}}$	**30**	**0.030**	**27.9**	**0.028**	**3.96**	$\mathbf{4.0*10^{-3}}$	**4.50**	$\mathbf{4.5*10^{-3}}$
Total BaP_{eq}				5.65		31.8		20.4		4.43		4.04

Concentrations in bold were detected in the environmental samples. Non-bolded concentrations were not detected, but levels were substituted with one-half the limit of detection for this calculation.

Table 4. Exposure scenarios and inputs.

Exposure Parameter	Occupational Worker Scenario (Indoor)	Nearby Adult Resident Scenario	Nearby Child Resident Scenario (Birth to <1y)
Hours per day	8.0	24.0	24.0
Days per week	5.0	7.0	7.0
Weeks per year	50	52.14	52.14
Years	6.6	12.0	1.0
Exposure Factor (unitless)			
Acute (0-14 d)	1	1	1
Intermediate (15-364 d)	0.71	1	1
Chronic (>365 d)	0.68	1	1
Cancer (>365 d)	0.061	0.154	0.013
Body Weight, kg (lbs)	80	80	7.8
Soil/Sediment Ingestion Rate, mg/d			
Central Tendency Exposure (CTE)	30	10	25
Reasonable Maximum Exposure (RME)	80	50	70
Skin Surface Area, cm^2	3470	6030	1772
Soil-Skin Adherence Factor, mg/cm^2	0.07	0.07	0.2
Dermal Absorption Factor (unitless)			
Arsenic	0.03	0.03	0.03
Lead	NA	NA	NA
Total PAHs	0.13	0.13	0.13

Table 5. Contaminant health guidance values and cancer slope factors.

Contaminant	Health Guidance Value (mg/kg/d)			Cancer Slope Factor (mg/kg/d)
	Acute	Intermediate	Chronic	
Arsenic	$5.0*10^{-3}$		$3.0*10^{-4}$	1.5
Lead^				$8.5*10^{-3}$ [+]
Chromium		$5.0*10^{-3}$	$9.0*10^{-4}$	0.5
Total PAHs			$3.0*10^{-4}$	1.0
Dibromoethane			$9.0*10^{-3}$	2.0

^ATSDR PHAST does not currently calculate lead exposure doses. The lead exposure doses are estimated by the authors.

[+]The value was provided by the California Office of Environmental Health Hazard Assessment for oral slope factor (Chou and Harper 2007).

Table 6. Occupational worker scenario, exposure dose calculations.

Contaminant	Estimated Dose (mg/kg/d)*						Hazard Quotient						Cancer Risk#	
	CTE			RME			CTE			RME			CTE	RME
	Acute	Intermediate	Chronic	Acute	Intermediate	Chronic	Acute	Intermediate	Chronic	Acute	Intermediate	Chronic		
Lot 155														
Lead^	$4.6*10^{-4}$	$3.3*10^{-4}$	$3.1*10^{-4}$	$1.2*10^{-3}$	$8.7*10^{-4}$	$8.3*10^{-4}$							$2.4*10^{-7}$	$6.3*10^{-7}$
Total PAHs	$4.3*10^{-6}$	$3.1*10^{-6}$	$3.0*10^{-6}$	$7.9*10^{-6}$	$5.6*10^{-6}$	$5.4*10^{-6}$			0.010			0.018	$2.7*10^{-7}$	$4.8*10^{-7}$
Lot 268														
Lead^	$7.5*10^{-4}$	$5.3*10^{-4}$	$5.1*10^{-3}$	$2.0*10^{-3}$	$1.4*10^{-3}$	$1.4*10^{-3}$							$3.9*10^{-7}$	$1.0*10^{-6}$
Total PAHs	$2.4*10^{-5}$	$1.7*10^{-5}$	$1.7*10^{-5}$	$4.4*10^{-5}$	$3.2*10^{-5}$	$3.0*10^{-5}$			0.056			0.100	$1.5*10^{-6}$	$2.7*10^{-6}$
Lot 267														
Arsenic	$7.3*10^{-6}$	$5.2*10^{-6}$	$5.0*10^{-6}$	$1.6*10^{-5}$	$1.1*10^{-5}$	$1.1*10^{-5}$	$1.5*10^{-3}$		0.017	$3.2*10^{-3}$		0.036	$6.7*10^{-7}$	$1.5*10^{-6}$
Lead^	$2.7*10^{-4}$	$2.0*10^{-4}$	$2.0*10^{-4}$	$7.6*10^{-4}$	$5.4*10^{-4}$	$5.2*10^{-4}$							$1.5*10^{-7}$	$4.0*10^{-7}$
Chromium&	$2.9*10^{-5}$	$2.0*10^{-5}$	$2.0*10^{-5}$	$4.0*10^{-5}$	$2.8*10^{-5}$	$2.7*10^{-5}$		$4.1*10^{-3}$	0.022		$5.7*10^{-3}$	0.030	$8.8*10^{-7}$	$1.2*10^{-6}$
Total PAHs	$1.6*10^{-5}$	$1.1*10^{-5}$	$1.1*10^{-5}$	$2.8*10^{-5}$	$2.0*10^{-5}$	$1.9*10^{-5}$			0.036			0.065	$9.7*10^{-7}$	$1.7*10^{-6}$
Dibromoethane	$8.9*10^{-8}$	$6.4*10^{-8}$	$6.1*10^{-8}$	$2.1*10^{-7}$	$1.5*10^{-7}$	$1.4*10^{-7}$			$6.8*10^{-6}$			$1.6*10^{-5}$	$1.1*10^{-8}$	$2.6*10^{-8}$
Lot 272														
Arsenic	$4.0*10^{-6}$	$2.8*10^{-6}$	$2.7*10^{-6}$	$8.6*10^{-6}$	$6.2*10^{-6}$	$5.9*10^{-6}$	$7.9*10^{-4}$		$9.0*10^{-3}$	$1.7*10^{-3}$		0.020	$3.6*10^{-7}$	$8.0*10^{-7}$
Lead^	$1.1*10^{-4}$	$7.6*10^{-5}$	$7.2*10^{-4}$	$2.8*10^{-4}$	$2.0*10^{-4}$	$1.9*10^{-4}$							$5.5*10^{-8}$	$1.5*10^{-7}$
Chromium&	$1.8*10^{-5}$	$1.3*10^{-5}$	$1.2*10^{-5}$	$2.5*10^{-5}$	$1.8*10^{-5}$	$1.7*10^{-5}$		$2.6*10^{-3}$	0.014		$3.6*10^{-3}$	0.019	$5.6*10^{-7}$	$7.8*10^{-7}$
Total PAHs	$3.4*10^{-6}$	$2.4*10^{-6}$	$2.3*10^{-6}$	$6.2*10^{-6}$	$4.4*10^{-6}$	$4.2*10^{-6}$			$7.8*10^{-3}$			0.014	$2.1*10^{-7}$	$3.8*10^{-7}$
Lot 248														
Arsenic	$2.7*10^{-6}$	$1.9*10^{-6}$	$1.9*10^{-6}$	$6.0*10^{-6}$	$4.3*10^{-6}$	$4.1*10^{-6}$	$5.4*10^{-4}$		$6.2*10^{-3}$	$1.2*10^{-4}$		0.014	$2.5*10^{-7}$	$5.5*10^{-7}$
Lead^	$9.2*10^{-5}$	$6.6*10^{-5}$	$6.3*10^{-4}$	$2.5*10^{-4}$	$1.8*10^{-4}$	$1.7*10^{-4}$							$4.8*10^{-8}$	$1.3*10^{-7}$
Chromium&	$1.8*10^{-5}$	$1.3*10^{-5}$	$1.2*10^{-5}$	$2.6*10^{-5}$	$1.8*10^{-5}$	$1.8*10^{-5}$		$2.6*10^{-3}$	0.014		$3.7*10^{-3}$	0.020	$5.7*10^{-7}$	$7.9*10^{-7}$
Total PAHs	$3.1*10^{-6}$	$2.2*10^{-6}$	$2.1*10^{-6}$	$5.6*10^{-6}$	$4.0*10^{-6}$	$3.9*10^{-6}$			$7.1*10^{-3}$			0.013	$1.9*10^{-7}$	$3.5*10^{-7}$

Highlighted cells indicated an elevated risk for non-cancer or cancer health effects.

*Estimated doses were combined for soil ingestion and dermal contact.

#ATSDR considers increased health risks to be Hazard Quotient>1.0 or Cancer Risk>$1.0*10^{-6}$.

^ATSDR PHAST does not currently calculate lead exposure doses. The lead exposure doses are estimated by the authors.

&Assumed to be hexavalent chromium.

Table 7. Nearby adult resident scenario, exposure dose calculations.

Contaminant	Estimated Dose (mg/kg/d)*						Hazard Quotient						Cancer Risk#	
	CTE			RME			CTE			RME			CTE	RME
	Acute	Intermediate	Chronic	Acute	Intermediate	Chronic	Acute	Intermediate	Chronic	Acute	Intermediate	Chronic		
Lot 155														
Lead^	$1.5*10^{-4}$	$1.5*10^{-4}$	$1.5*10^{-4}$	$7.6*10^{-4}$	$7.6*10^{-4}$	$7.6*10^{-4}$							$2.0*10^{-7}$	$1.0*10^{-6}$
Total PAHs	$4.6*10^{-6}$	$4.6*10^{-6}$	$4.6*10^{-6}$	$7.6*10^{-6}$	$7.6*10^{-6}$	$7.6*10^{-6}$			0.015			0.025	$7.0*10^{-7}$	$1.1*10^{-6}$
Lot 268														
Lead^	$2.5*10^{-4}$	$2.5*10^{-4}$	$2.5*10^{-4}$	$1.3*10^{-3}$	$1.3*10^{-3}$	$1.3*10^{-3}$							$3.3*10^{-7}$	$1.6*10^{-6}$
Total PAHs	$2.6*10^{-5}$	$2.6*10^{-5}$	$2.6*10^{-5}$	$4.2*10^{-5}$	$4.2*10^{-5}$	$4.2*10^{-5}$			0.086			0.140	$4.0*10^{-6}$	$6.4*10^{-6}$
Lot 267														
Arsenic	$5.4*10^{-6}$	$5.4*10^{-6}$	$5.4*10^{-6}$	$1.2*10^{-5}$	$1.2*10^{-5}$	$1.2*10^{-5}$	$1.1*10^{-3}$		0.018	$2.5*10^{-3}$		0.041	$1.2*10^{-6}$	$2.8*10^{-6}$
Lead^	$9.5*10^{-5}$	$9.5*10^{-5}$	$9.5*10^{-5}$	$4.8*10^{-4}$	$4.8*10^{-4}$	$4.8*10^{-4}$							$1.3*10^{-7}$	$6.2*10^{-7}$
Chromium&	$4.0*10^{-5}$	$4.0*10^{-5}$	$4.0*10^{-5}$	$4.9*10^{-5}$	$4.9*10^{-5}$	$4.9*10^{-5}$		$8.0*10^{-3}$	0.045		$9.8*10^{-3}$	0.055	$3.1*10^{-6}$	$3.8*10^{-6}$
Total PAHs	$1.7*10^{-5}$	$1.7*10^{-5}$	$1.7*10^{-5}$	$2.7*10^{-5}$	$2.7*10^{-5}$	$2.7*10^{-5}$			0.055			0.089	$2.5*10^{-6}$	$4.1*10^{-6}$
Dibromoethane	$5.4*10^{-8}$	$5.4*10^{-8}$	$5.4*10^{-8}$	$1.5*10^{-7}$	$1.5*10^{-7}$	$1.5*10^{-7}$			$6.0*10^{-6}$			$1.7*10^{-5}$	$1.8*10^{-8}$	$4.6*10^{-8}$
Lot 272														
Arsenic	$2.9*10^{-6}$	$2.9*10^{-6}$	$2.9*10^{-6}$	$6.7*10^{-6}$	$6.7*10^{-6}$	$6.7*10^{-6}$	$5.8*10^{-4}$		$9.7*10^{-3}$	$1.3*10^{-3}$		0.022	$6.7*10^{-7}$	$1.5*10^{-6}$
Lead^	$3.6*10^{-5}$	$3.6*10^{-5}$	$3.6*10^{-5}$	$1.8*10^{-4}$	$1.8*10^{-4}$	$1.8*10^{-4}$							$4.6*10^{-8}$	$2.3*10^{-7}$
Chromium&	$2.5*10^{-5}$	$2.5*10^{-5}$	$2.5*10^{-5}$	$3.1*10^{-5}$	$3.1*10^{-5}$	$3.1*10^{-5}$		$5.1*10^{-3}$	0.028		$6.2*10^{-3}$	0.035	$2.0*10^{-6}$	$2.4*10^{-6}$
Total PAHs	$3.6*10^{-6}$	$3.6*10^{-6}$	$3.6*10^{-6}$	$5.8*10^{-6}$	$5.8*10^{-6}$	$5.8*10^{-6}$			0.012			0.019	$5.5*10^{-7}$	$8.9*10^{-7}$
Lot 248														
Arsenic	$2.0*10^{-6}$	$2.0*10^{-6}$	$2.0*10^{-6}$	$4.6*10^{-6}$	$4.6*10^{-6}$	$4.6*10^{-6}$	$4.0*10^{-4}$		$6.7*10^{-3}$	$9.2*10^{-4}$		0.015	$4.6*10^{-7}$	$1.1*10^{-6}$
Lead^	$3.1*10^{-5}$	$3.1*10^{-5}$	$3.1*10^{-5}$	$1.5*10^{-4}$	$1.5*10^{-4}$	$1.5*10^{-4}$							$4.0*10^{-8}$	$2.0*10^{-7}$
Chromium&	$2.6*10^{-5}$	$2.6*10^{-5}$	$2.6*10^{-5}$	$3.2*10^{-5}$	$3.2*10^{-5}$	$3.2*10^{-5}$		$5.2*10^{-3}$	0.029		$6.3*10^{-3}$	0.035	$2.0*10^{-6}$	$2.4*10^{-6}$
Total PAHs	$3.3*10^{-6}$	$3.3*10^{-6}$	$3.3*10^{-6}$	$5.3*10^{-6}$	$5.3*10^{-6}$	$5.3*10^{-6}$			0.011			0.018	$5.0*10^{-7}$	$8.1*10^{-7}$

Highlighted cells indicated an elevated risk for non-cancer or cancer health effects.
*Estimated doses were combined for soil ingestion and dermal contact.
#ATSDR considers increased health risks to be Hazard Quotient>1.0 or Cancer Risk>$1.0*10^{-6}$.
^ATSDR PHAST does not currently calculate lead exposure doses. The lead exposure doses are estimated by the authors.
&Assumed to be hexavalent chromium.

Table 8. Nearby child resident scenario (birth to <1y), exposure dose calculations.

Contaminant	Estimated Dose (mg/kg/d)*						Hazard Quotient						Cancer Risk[#,+]	
	CTE			RME			CTE			RME			CTE	RME
	Acute	Intermediate	Chronic	Acute	Intermediate	Chronic	Acute	Intermediate	Chronic	Acute	Intermediate	Chronic		
Lot 155														
Lead^	$3.9*10^{-3}$	$3.9*10^{-3}$	$3.9*10^{-3}$	0.011	0.011	0.011							$2.5*10^{-6}$	$7.0*10^{-6}$
Total PAHs	$5.1*10^{-5}$	$5.1*10^{-5}$	$5.1*10^{-5}$	$8.4*10^{-5}$	$8.4*10^{-5}$	$8.4*10^{-5}$			0.170			0.280	$2.3*10^{-5}$	$3.5*10^{-5}$
Lot 268														
Lead^	$6.4*10^{-3}$	$6.4*10^{-3}$	$6.4*10^{-3}$	0.018	0.018	0.018							$4.0*10^{-6}$	$1.1*10^{-5}$
Total PAHs	$2.9*10^{-4}$	$2.9*10^{-4}$	$2.9*10^{-4}$	$4.7*10^{-4}$	$4.7*10^{-4}$	$4.7*10^{-4}$			0.970			1.60	$1.3*10^{-4}$	$2.0*10^{-4}$
Lot 267														
Arsenic	$7.6*10^{-5}$	$7.6*10^{-5}$	$7.6*10^{-5}$	$1.6*10^{-4}$	$1.6*10^{-4}$	$1.6*10^{-4}$	0.015		0.250	0.031		0.520	$9.5*10^{-6}$	$1.9*10^{-5}$
Lead^	$2.5*10^{-3}$	$2.5*10^{-3}$	$2.5*10^{-3}$	$6.9*10^{-3}$	$6.9*10^{-3}$	$6.9*10^{-3}$							$1.5*10^{-6}$	$4.4*10^{-6}$
Chromium[&]	$3.8*10^{-4}$	$3.8*10^{-4}$	$3.8*10^{-4}$	$4.9*10^{-4}$	$4.9*10^{-4}$	$4.9*10^{-4}$		0.077	$3.8*10^{-4}$		0.098	0.540	$8.7*10^{-5}$	$1.1*10^{-4}$
Total PAHs	$1.9*10^{-4}$	$1.9*10^{-4}$	$1.9*10^{-4}$	$3.0*10^{-4}$	$3.0*10^{-4}$	$3.0*10^{-4}$			0.620			1.00	$8.2*10^{-5}$	$1.3*10^{-4}$
Dibromoethane	$8.7*10^{-7}$	$8.7*10^{-7}$	$8.7*10^{-7}$	$2.0*10^{-6}$	$2.0*10^{-6}$	$2.0*10^{-6}$			$9.7*10^{-5}$			$2.2*10^{-4}$	$1.4*10^{-7}$	$3.1*10^{-7}$
Lot 272														
Arsenic	$4.1*10^{-5}$	$4.1*10^{-5}$	$4.1*10^{-5}$	$8.4*10^{-5}$	$8.4*10^{-5}$	$8.4*10^{-5}$	$8.2*10^{-3}$		0.140	0.017		0.280	$5.2*10^{-6}$	$1.0*10^{-5}$
Lead^	$9.1*10^{-4}$	$9.1*10^{-4}$	$9.1*10^{-4}$	$2.6*10^{-3}$	$2.6*10^{-3}$	$2.6*10^{-3}$							$5.7*10^{-7}$	$1.6*10^{-6}$
Chromium[&]	$2.4*10^{-4}$	$2.4*10^{-4}$	$2.4*10^{-4}$	$3.1*10^{-4}$	$3.1*10^{-4}$	$3.1*10^{-4}$		0.049	0.270		0.062	0.340	$5.5*10^{-5}$	$6.8*10^{-5}$
Total PAHs	$4.0*10^{-5}$	$4.0*10^{-5}$	$4.0*10^{-5}$	$6.6*10^{-5}$	$6.6*10^{-5}$	$6.6*10^{-5}$			0.130			0.220	$1.8*10^{-5}$	$2.8*10^{-5}$
Lot 248														
Arsenic	$2.8*10^{-5}$	$2.8*10^{-5}$	$2.8*10^{-5}$	$5.8*10^{-5}$	$5.8*10^{-5}$	$5.8*10^{-5}$	$5.7*10^{-3}$		0.094	0.012		0.190	$3.6*10^{-6}$	$6.9*10^{-6}$
Lead^	$7.9*10^{-4}$	$7.9*10^{-4}$	$7.9*10^{-4}$	$2.2*10^{-3}$	$2.2*10^{-3}$	$2.2*10^{-3}$							$5.0*10^{-7}$	$1.4*10^{-6}$
Chromium[&]	$2.5*10^{-4}$	$2.5*10^{-4}$	$2.5*10^{-4}$	$3.1*10^{-4}$	$3.1*10^{-4}$	$3.1*10^{-4}$		0.050	0.280		0.063	0.350	$5.6*10^{-5}$	$6.9*10^{-5}$
Total PAHs	$3.7*10^{-5}$	$3.7*10^{-5}$	$3.7*10^{-5}$	$6.0*10^{-5}$	$6.0*10^{-5}$	$6.0*10^{-5}$			0.120			0.200	$1.6*10^{-5}$	$2.5*10^{-5}$

Highlighted cells indicated an elevated risk for non-cancer or cancer health effects.
*Estimated doses were combined for soil ingestion and dermal contact.
[#]ATSDR considers increased health risks to be Hazard Quotient>1.0 or Cancer Risk>$1.0*10^{-6}$.
[+]Cancer risk calculations assumed a child resident lived nearby for 12 years (EPA 2017) from birth.
^ATSDR PHAST does not currently calculate lead exposure doses. The lead exposure doses are estimated by the authors.
[&]Assumed to be hexavalent chromium.

APPENDICES

Appendix A. Full contaminant analyte list for Waites Wharf soil samples.

Chemical Class	Contaminant	Surface Soil	Subsurface Soil	Groundwater	Soil-Gas
Metals	Arsenic	X	X		
	Barium	X	X		
	Cadmium	X			
	Chromium	X	X		
	Lead	X	X		
	Mercury	X	X		
	Selenium				
	Silver				
Volatile Organic Compounds (VOCs)	1-Chlorohexane				
	1,1-Dichloroethane				
	1,1-Dichloroethene				
	1,1-Dichloropropene				
	1,1,1-Trichloroethane				
	1,1,1,2-Tetrachloroethane				
	1,1,2-Trichloroethane				
	1,1,2,2-Tetrachloroethane				
	1,2-Dibromo-3-Chloropropane	X			
	1,2-Dibromoethane	X			
	1,2-Dichlorobenzene				
	1,2-Dichloroethane				
	1,2-Dichloropropane				
	1,2,3-Trichlorobenzene				
	1,2,3-Trichloropropane				
	1,2,4-Trichlorobenzene				
	1,2,4-Trimethylbenzene				
	1,3-Dichlorobenzene				
	1,3-Dichloropropane				
	1,3,5-Trimethylbenzene				
	1,4-Dichlorobenzene				
	1,4-Dioxane				
	2-Butanone				
	2-Chlorotoluene				
	2-Hexanone				
	2,2-Dichloropropane				
	4-Chlorotoluene				
	4-Isopropyltoluene				
	4-Methal-2-Pentanone				
	Acetone	X			
	Benzene	X	X	X	
	Bromobenzene				
	Bromochloromethane				
	Bromodichloromethane				

Appendix A Continued.

Chemical Class	Contaminant	Surface Soil	Subsurface Soil	Groundwater	Soil-Gas
Volatile Organic Compounds (VOCs)	Bromoform				
	Bromomethane				
	Carbon Disulfide				
	Carbon Tetrachloride				
	Chlorobenzene				
	Chloroethane				
	Chloroform				
	Chloromethane				
	cis-1,2-Dichloroethene				
	cis-1,3-Dichloropropene				
	Dibromochloromethane				
	Dibromomethane				
	Dichlorodifluoromethane				
	Diethyl Ether				
	Di-isopropyl Ether				
	Ethyl Tertiary-Butyl Ether				
	Ethylbenzene	X	X	X	
	Hexachlorobutadiene				
	Isopropylbenzene				
	Methyl tert-Butyl Ether				
	Methylene Chloride	X		X	
	Naphthalene	X			
	n-Butylbenzene				
	n-Propylbenzene				
	sec-Butylbenzene				
	Styrene				
	tert-Butylbenzene				
	Tertiary-Amyl Methyl Ether				
	Tetrachloroethene	X			
	Tetrahydrofuran				
	Toluene	X	X	X	
	trans-1,2-Dichloroethene				
	trans-1,3-Dichloropropene				
	Trichloroethene	X			
	Trichlorofluoromethane				
	Vinyl Acetate				
	Vinyl Chloride	X			
	Xylene O				
	Xylene P,M				
	Xylenes (Total)	X	X	X	

Appendix A Continued.

Chemical Class	Contaminant	Surface Soil	Subsurface Soil	Groundwater	Soil-Gas
Polycyclic Aromatic Hydrocarbons (PAHs)	2-Methylnaphthalene	X			
	Acenaphthene	X	X		
	Acenaphthylene	X			
	Anthracene	X	X		
	Benzo(a)anthracene	X	X		
	Benzo(a)pyrene	X	X		
	Benzo(b)fluoranthene	X	X		
	Benzo(ghi)perylene	X	X		
	Benzo(k)fluoranthene	X	X		
	Chrysene	X	X		
	Dibenzo(ah)anthracene	X	X		
	Fluoranthene	X	X		
	Fluorene	X			
	Indeno(123-cd)pyrene	X	X		
	Naphthalene	X	X		
	Phenanthrene	X	X		
	Pyrene	X	X		
	Total Petroleum Hydrocarbons	X	X	X	
	Cyanide				

Appendix B. Background soil PAH levels in New England.

PAH	Units	New England Background Level (upper 95% interval)	Waites Wharf Soil Samples, Western Lots (maximum level detected)	Waites Wharf Soil Samples, Eastern Lots (maximum level detected)
Acenaphthylene	mg/kg	0.208	ND (<4.6)	0.367
Anthracene	mg/kg	0.535	ND (<4.6)	6.49
Benzo(a)anthracene	mg/kg	1.86	18	16.4
Benzo(a)pyrene	mg/kg	1.82	22	12.4
Benzo(b)fluoranthene	mg/kg	1.97	22	13.3
Benzo(ghi)perylene	mg/kg	1.20	18	5.87
Benzo(k)fluoranthene	mg/kg	2.52	21	9.75
Chrysene	mg/kg	2.69	21	15.4
Dibenzo(ah)anthracene	mg/kg	0.521	ND (<4.6)	3.07
Fluoranthene	mg/kg	4.44	36	35.1
Indeno(1,2,3-cd)pyrene	mg/kg	1.29	11	5.68
Naphthalene	mg/kg	0.149	6.4	1.45
Phenanthrene	mg/kg	2.98	10	31.1
Pyrene	mg/kg	2.95	30	27.9

ND: Not detected
(Bradley, Magee, and Allen 1994)

Appendix C. Michigan Particulate Soil Inhalation Criteria.

Contaminant	Units	Michigan PSIC (Industrial/ Commercial)	Michigan PSIC (Residential)	Waites Wharf Soil Samples, Western Lots (maximum level detected)	Waites Wharf Soil Samples, Eastern Lots (maximum level detected)
Benzo(a)pyrene	mg/kg	1900	1500	31.8 (total PAHs)	20.4 (total PAHs)
Arsenic	mg/kg	910	720	-	23.1
Chromium	mg/kg	260	240	-	18.0
Lead	mg/kg	100,000	44,000	1994	763

(Michigan Department of Community Health 2005)