

Health Consultation

**Budd Inlet
Thurston County
Olympia, Washington**

UPDATED

September 30, 2008

Prepared by:
Washington State Department of Health
under Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry



Foreword

The Washington State Department of Health (DOH) has prepared this health consultation in cooperation with the Agency for Toxic Substances and Disease Registry (ATSDR). ATSDR is part of the U.S. Department of Health and Human Services and is the principal federal public health agency responsible for health issues related to hazardous waste. This health consultation was prepared in accordance with methodologies and guidelines developed by ATSDR.

The purpose of this health consultation is to identify and prevent harmful human health effects resulting from exposure to hazardous substances in the environment. Health consultations focus on specific health issues so that DOH can respond to requests from concerned residents or agencies for health information on hazardous substances. DOH evaluates sampling data collected from a hazardous waste site, determines whether exposures have occurred or could occur, reports any potential harmful effects, and recommends actions to protect public health. The findings in this report are relevant to conditions at the site during the time of this health consultation, and should not necessarily be relied upon if site conditions or land use changes in the future.

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For more information about ATSDR, contact the ATSDR Information Center at 1-888-422-8737 or visit the agency's Web site: www.atsdr.cdc.gov/.

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Glossary

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| Agency for Toxic Substances and Disease Registry (ATSDR) | The principal federal public health agency involved with hazardous waste issues, responsible for preventing or reducing the harmful effects of exposure to hazardous substances on human health and quality of life. ATSDR is part of the U.S. Department of Health and Human Services. |
| Cancer Risk | A theoretical risk for developing cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower. |
| Cancer Risk Evaluation Guide (CREG) | The concentration of a chemical in air, soil or water that is expected to cause no more than one excess cancer in a million persons exposed over a lifetime. The CREG is a <i>comparison value</i> used to select contaminants of potential health concern and is based on the <i>cancer slope factor</i> (CSF). |
| Cancer Slope Factor | A number assigned to a cancer causing chemical that is used to estimate its ability to cause cancer in humans. |
| Carcinogen | Any substance that causes cancer. |
| Comparison value | Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process. |
| Contaminant | A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects. |
| Dermal Contact | Contact with (touching) the skin (see route of exposure). |
| Dose (for chemicals that are not radioactive) | The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An “exposure dose” is how much of a substance is encountered in the environment. An “absorbed dose” is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs. |
| Environmental Media Evaluation Guide (EMEG) | A concentration in air, soil, or water below which adverse non-cancer health effects are not expected to occur. The EMEG is a <i>comparison value</i> used to select contaminants of potential health concern and is based on ATSDR’s <i>minimal risk level</i> (MRL). |

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| Environmental Protection Agency (EPA) | United States Environmental Protection Agency. |
| Exposure | Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration, or long-term [chronic exposure]. |
| Groundwater | Water beneath the earth's surface in the spaces between soil particles and between rock surfaces [compare with surface water]. |
| Hazardous substance | Any material that poses a threat to public health and/or the environment. Typical hazardous substances are materials that are toxic, corrosive, ignitable, explosive, or chemically reactive. |
| Ingestion | The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see route of exposure]. |
| Ingestion rate | The amount of an environmental medium that could be ingested typically on a daily basis. Units for IR are usually liter/day for water, and mg/day for soil. |
| Inhalation | The act of breathing. A hazardous substance can enter the body this way [see route of exposure]. |
| Inorganic | Compounds composed of mineral materials, including elemental salts and metals such as iron, aluminum, mercury, and zinc. |
| Lowest Observed Adverse Effect Level (LOAEL) | The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals. |
| Maximum Contaminant Level (MCL) | A drinking water regulation established by the federal Safe Drinking Water Act. It is the maximum permissible concentration of a contaminant in water that is delivered to the free flowing outlet of the ultimate user of a public water system. MCLs are enforceable standards. |
| Media | Soil, water, air, plants, animals, or any other part of the environment that can contain contaminants. |

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| <p>Minimal Risk Level (MRL)</p> | <p>An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see reference dose].</p> |
| <p>Model Toxics Control Act (MTCA)</p> | <p>The hazardous waste cleanup law for Washington State.</p> |
| <p>No apparent public health hazard</p> | <p>A category used in ATSDR’s public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.</p> |
| <p>No Observed Adverse Effect Level (NOAEL)</p> | <p>The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.</p> |
| <p>Oral Reference Dose (RfD)</p> | <p>An amount of chemical ingested into the body (i.e., dose) below which health effects are not expected. RfDs are published by EPA.</p> |
| <p>Organic</p> | <p>Compounds composed of carbon, including materials such as solvents, oils, and pesticides that are not easily dissolved in water.</p> |
| <p>Parts per billion (ppb)/Parts per million (ppm)</p> | <p>Units commonly used to express low concentrations of contaminants. For example, 1 ounce of trichloroethylene (TCE) in 1 million ounces of water is 1 ppm. 1 ounce of TCE in 1 billion ounces of water is 1 ppb. If one drop of TCE is mixed in a competition size swimming pool, the water will contain about 1 ppb of TCE.</p> |
| <p>Plume</p> | <p>A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.</p> |
| <p>Reference Dose Media Evaluation Guide (RMEG)</p> | <p>A concentration in air, soil, or water below which adverse non-cancer health effects are not expected to occur. The RMEG is a <i>comparison value</i> used to select contaminants of potential health concern and is based on EPA’s oral reference dose (RfD).</p> |
| <p>Route of exposure</p> | <p>The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].</p> |

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| <p>Surface Water</p> | <p>Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs [compare with groundwater].</p> |
| <p>Time Weighted Approach (TWA)</p> | <p>The exposure concentration of a contaminant during a given period.</p> |
| <p>Volatile organic compound (VOC)</p> | <p>Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and methyl chloroform.</p> |

Summary and Statement of Issues

The Washington State Department of Health (DOH) has prepared this health consultation at the request of the Washington State Department of Ecology (Ecology) and the Thurston County Public Health and Social Services Department (TCHD). The purpose of this health consultation is to evaluate the potential human health hazard posed by contaminants in sediments, clams and bottom fish tissue, at Budd Inlet in Thurston County, Olympia, Washington. DOH prepares health consultations under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). This document was updated from the ATSDR document dated July 28, 2008. The Polycyclic aromatic hydrocarbons (PAHs) data in the previous document was based on total organic carbon and not on total sediment. An addendum in the form of a technical assistance was added to the July 28 ATSDR document.

Background

Budd Inlet is located in southern Puget Sound, near the city of Olympia, WA (Figure 1). Historically, the southern portion of Budd Inlet has supported wood product industries, recreational marinas, and boat industries. It is also the home of the Port of Olympia. Several sites are listed on Ecology's Confirmed and Suspected Contaminated Sites List as contributing contaminants to Budd Inlet (for example – dioxin from the former Cascade Pole). Budd Inlet is divided into the East and West Bays by a small peninsula, which extends from the southern point. The Olympia Harbor federal navigation channel and turning basin are maintained in inner West Bay. The northern portion or the North Inlet is lined with residential properties [1].

Past and current commercial and industrial activities identified in East Bay include cargo handling, boat manufacturing, marina operations and a wood-treating facility. Moxlie Creek flows through a culvert that discharges into the southern end of East Bay. The former Cascade Pole wood treating facility is a cleanup site located on the north end of the peninsula. The Swantown Marina and Boatworks is located on the eastern side of the peninsula.

Industries along the shoreline of West Bay include the Port of Olympia marine terminal, recreational marinas (Fiddlehead and West Bay marinas, Olympia Yacht Club), Hardel Mutual Plywood, and Reliable Steel (now Brown Minneapolis Tank-Northwest). Hardel is under an Ecology agreed order to conduct a site remedial investigation and feasibility study (RI/FS) to guide the selection of a cleanup remedy [2]. The Reliable Steel site was originally developed as a lumber mill and since 1941, the site has been used for boat building, welding, and steel fabrication. Reliable Steel site is under an Ecology proposed agreed order to conduct a site RI/FS to guide the selection of a cleanup remedy [3].

The North Inlet area extends north of the Olympia Harbor navigation channel entrance to the mouth of Budd Inlet. The east and west shorelines of the North Inlet consist primarily of residential properties.

Priest Point Park is a City of Olympia park with public beach access along East Bay (See figure 1). This beach is within the closure area for a sewage treatment plant outfall and is listed unsafe

for recreational shellfish harvesting. In the North Inlet area, Burfoot is a Thurston Count Park with public beach access. This beach has not been evaluated by the Department of Health for pollution problems and has an advisory due to unknown water quality.

Table 1. Maximum concentrations of contaminants detected in sediment within Budd Inlet in Olympia, Washington.

| Compounds | Maximum Concentration (ppm) | Comparison Value (ppm) | EPA Cancer Class | Comparison Value Reference | Contaminant of Concern (COC) |
|------------------------|-----------------------------|------------------------|------------------|----------------------------|------------------------------|
| Antimony | 0.97 J | 20 | D | RMEG | No |
| Arsenic | 10.5 | 20 | A | EMEG | No |
| Cadmium | 2.58 | 10 | B1 | EMEG | No |
| Chromium | 36.8 | 200 ^a | A | RMEG | No |
| Copper | 77.5 | 2,000 | D | IM EMEG | No |
| Lead | 70.3 | 250 | B2 | MTCA | No |
| Mercury | 0.91 | 1 | D | MTCA | No |
| Nickel | 28.2 J | 1,000 | | RMEG | No |
| Silver | 1.52 | 300 | D | RMEG | No |
| Zinc | 260 J | 20,000 | D | EMEG | No |
| 2-Methylnaphthalene | 0.18 | 200 | | RMEG | No |
| Acenaphthene | 2.0 | 3000 | | RMEG | No |
| Acenaphthylene | 0.12 | 2000* | D | | No |
| Anthracene | 3.0 | 20000 | D | RMEG | No |
| Benzo(a)anthracene | 0.49 | 0.62 | B2 | Region 9 | cPAH |
| Benzo(a)pyrene | 0.48 | 0.1 | B2 | CREG | cPAH |
| Benzo(b)fluoranthene | 0.59 | 0.62 | B2 | Region 9 | cPAH |
| Benzo(k)fluoranthene | 0.22 | 6.2 | B2 | Region 9 | cPAH |
| Benzo(ghi)perylene | 0.29 | 2000* | D | | No |
| Chrysene | 0.55 | 62 | B2 | Region 9 | cPAH |
| Dibenz(a,h)anthracene | 0.073 | 0.1** | | CREG | cPAH |
| Dibenzofuran | 1.95 U | 290 | D | Region 9 | No |
| Fluoranthene | 0.92 | 2000 | D | RMEG | No |
| Fluorene | 1.7 | 2000 | D | RMEG | No |
| Indeno(1,2,3-cd)pyrene | 0.31 | 0.62 | B2 | Region 9 | cPAH |
| Naphthalene | 0.71 | 30000 | C | IM EMEG | No |
| Phenanthrene | 6.2 | 2000* | D | | No |
| Pyrene | 0.83 | 2000 | D | RMEG | No |
| Hexachlorobenzene | 1.95 U | 0.4 | B2 | CREG | No |

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|----------------------------|----------------------|---------|----|----------|------------|
| Hexachlorobutadiene | 1.95 U | 9 | C | CREG | No |
| 1,2,4-Trichlorobenzene | 1.95 U | 500 | D | RMEG | No |
| 1,2-Dichlorobenzene | 1.95 U | 5000 | D | RMEG | No |
| 1,3-Dichlorobenzene | 1.95 U | 2000 | D | IM EMEG | No |
| 1,4-Dichlorobenzene | 1.95 U | 4000 | C | IM EMEG | No |
| 2,4-Dimethylphenol | 0.68 U | 1000 | | RMEG | No |
| 2-Methylphenol | 0.14 U | 3100 | | Region 9 | No |
| 4-Methylphenol | 0.34 | 310 | | Region 9 | No |
| Benzoic acid | 0.063U | 200000 | | RMEG | No |
| Benzyl alcohol | 1.7 U | 18000 | | Region 9 | No |
| Bis(2-ethylhexyl)phthalate | 181.82 | 3000 | B2 | EMEG | No |
| Butyl benzyl phthalate | 5.45 | 10000 | C | RMEG | No |
| Di-n-butyl phthalate | 3.89 U | 5000 | D | RMEG | No |
| Di-n-octylphthalate | 1.95 U | 20000 | | IM EMEG | No |
| Diethyl phthalate | 1.95 U | 300000 | D | IM EMEG | No |
| Dimethyl phthalate | 1.95 U | 100000 | D | Region 9 | No |
| Hexachloroethane | 1.95 U | 50 | C | CREG | No |
| N-Nitrosodiphenylamine | 1.95 U | 9.9 | B2 | Region 9 | No |
| Pentachlorophenol | 1.4 U | 6 | B2 | CREG | No |
| Phenol | 0.41 U | 20000 | D | RMEG | No |
| Total Aroclors | 2.88 [†] | 1*** | | EMEG | Yes |
| Total cPAH TEQ | 0.69 | 0.1 | B2 | CREG | Yes |
| Total Dioxin TEQ | 0.00006 [†] | 0.00005 | B2 | | Yes |

CREG - ATSDR's Cancer Risk Evaluation Guide (child)

RMEG - ATSDR's Reference Dose Media Evaluation Guide (child)

EMEG - ATSDR's Environmental Media Evaluation Guide (child)

IM EMEG - ATSDR's Intermediate Environmental Media Evaluation Guide (child)

J, E - data qualifier: The associated numerical result is an estimate

U- data qualifier: The analyte was not detected at this level

B2 - EPA: Probable human carcinogen (inadequate human, sufficient animal studies)

C - EPA: Possible human carcinogen (no human, limited animal studies)

D - EPA: Not classifiable as to health carcinogenicity

Region 9 - EPA: Preliminary Remediation Goals

* Fluoranthene RMEG value was used as a surrogate

** Benzo(a)pyrene CREG value was used as a surrogate

*** Arocolor 1254 EMEG value was used as a surrogate

[†] Maximum surface sediment

Total Dioxin TEQ - sum of dioxin/furans toxic equivalent (TEQ)

Total cPAH TEQ - sum of all carcinogenic polycyclic aromatic hydrocarbons (cPAH) toxic equivalent (TEQ), all cPAH in COC are added using the TEQ approach to obtain Total cPAH TEQ.

Table 2. Dioxin concentrations detected in fish and shellfish from Budd Inlet in Olympia, Washington.

| Species | Contaminant | Maximum Concentration (ppt) | Range of Concentration (ppt) | EPA Cancer Class |
|--------------------------|------------------|-----------------------------|------------------------------|------------------|
| Starry Flounder | Total Dioxin TEQ | 1.18 | 0.16 – 1.18 | B2 |
| English Sole | | 0.92 | 0.8 – 0.92 | |
| Manila / Littleneck clam | | 1.58 | 0.2 – 1.58 | |
| Bent Nose clam | | 2.55 | 0.74 – 2.55 | |
| Ghost Shrimp | | 5.64 | 2.59 – 5.64 | |

B2 - EPA: Probable human carcinogen (inadequate human, sufficient animal studies)
 Total Dioxin TEQ – sum of dioxin/furans toxic equivalent (TEQ)

Discussion

Contaminants of Concern

Contaminants of concern (COC) in sediment were determined by employing a screening process. Maximum sediment contaminant levels were screened against health-based soil comparison values. Several types of health-based comparison or screening values were used during this process [see the glossary for descriptions of “comparison value,” “cancer risk evaluation guide (CREG),” “environmental media evaluation guide (EMEG),” and “reference dose media evaluation guide (RMEG)”]. Comparison values such as the CREG and EMEG offer a high degree of protection and assurance that people are unlikely to be harmed by contaminants in the environment. For chemicals that cause cancer, the comparison values represent levels that are calculated to increase the risk of cancer by about one in a million. These types of comparison values often form the basis for cleanup. In general, if a contaminant’s maximum concentration is greater than its comparison value, then the contaminant is evaluated further.

Comparisons may also be made with legal standards such as the cleanup levels specified in the Washington State toxic waste cleanup law, the Model Toxics Control Act (MTCA). Legal standards may be strictly health-based or they may incorporate non-health considerations such as the cost or the practicality of attainment, or natural background levels.

Exposure Pathways

In order for any contaminant to be a health concern, the contaminant must be present at a high enough concentration to cause potential harm, and there must be a completed route of exposure to people.

Human use patterns and site-specific conditions were considered in the evaluation of exposure to lead and arsenic. Exposure to contaminants in sediment can occur through the following pathways and routes:

Ingestion exposure (swallowing)

Most people inadvertently swallow small amounts of sediments, soil and dust (and any contaminants they contain). Young children often put hands, toys, pacifiers, and other things in their mouths, and these may have dirt or dust on them that can be swallowed. Adults may ingest sediments, soil, and dust through activities such as gardening, mowing, construction work, dusting, and in this case, recreational activities.

Pica behavior is a persistent eating of non-food substances (such as dirt or paper). In a small percentage of children, pica behavior has been found to result in the ingestion of relatively large amounts of soil (one or more grams per day). Compared to typical children, those who swallow large amounts of contaminated soil may have added risks from short-term exposure. Some adults may also exhibit pica behavior.

Inhalation exposure (breathing)

Although people can inhale suspended sediment, soil or dust, airborne sediment usually consists of relatively large particles that are trapped in the nose, mouth, and throat and are then swallowed, rather than breathed into the lungs.

Skin exposure (dermal)

Dirt particles that can adhere to the skin may cause additional exposure to contaminants through dermal absorption. Although human skin is an effective barrier for many environmental contaminants, some chemicals can move easily through the skin.

The following discussion addresses human use patterns and site-specific conditions that are considered in the evaluation of exposure to dioxins and furans (dioxins), carcinogenic polycyclic aromatic hydrocarbons (cPAHs), and polychlorinated biphenyls (PCBs) as contaminants of concern in site sediments through the following pathways and routes:

- Inadvertent sediment ingestion, dust particles inhalation and dermal absorption of contaminants in sediment during beach play.

Beach Play Scenario

Although contact with sediments at the beaches may be an infrequent or seasonal exposure pathway, there is concern because areas along Budd Inlet have elevated levels of contaminants (see Table 1). Exposure to contaminants in sediment can occur by swallowing it (ingestion exposure), breathing it (inhalation exposure) or getting it on the skin (dermal exposure). During

recreational activities at the beaches, people are likely to be exposed to contaminants in sediments. In order for any contaminant to be a health concern, the contaminant must be present at a high enough concentration to cause potential harm, and there must be a completed route of exposure to people. Dioxins, PCBs and cPAHs are evaluated below since they exceed their health comparison values in sediments. See appendix A for evaluation of sediments.

Fish and Shellfish ingestion Scenario

Currently, most of the shell fishing areas in Budd Inlet is closed for harvesting of shellfish due to pollution or under a harvest advisory due to unknown water quality. Budd Inlet falls under the Puget Sound Fish Consumption Advisory Recreational Marine Area 13, with a fish advisory that states do not eat flatfish/bottom fish (English sole and Starry Flounder). For bottom fish and shellfish (Manila/Littleneck clams, Bent Nose clams and Ghost Shrimps) tissue, dioxin was the only contaminant analyzed for in these samples (see Table 2). Similarly, in order for any contaminant to be a health concern, the contaminant must be present at a high enough concentration to cause potential harm, and there must be a completed route of exposure to people. People may at times disregard these advisories and consume bottom fish and shellfish from Budd Inlet. In the event this scenario occurs, DOH evaluated bottom fish and Manila/Littleneck clams exposure to dioxin for the general population and the Squaxin Island Tribe (See appendix B). Manila/Littleneck clams are targeted shellfish species for human consumption. Bent Nose clams and Ghost Shrimps are not targeted shellfish species for human consumption.

Appendix B details the methodology and assumptions used by DOH to estimate dioxin exposure from eating fish and shellfish. For the general population adult, average ingestion rates of 17.5 g/day were used to calculate exposure doses for bottom fish caught and consumed in Budd Inlet for adults. This value represent the 90th percentile per capita ingestion rates for people of age 18 or older in the United States, including people that consumed fish and did not consume fish [4]. For the general population adult, average shellfish consumption rate of 1.7 g/day were used to calculate exposure doses. For the general population children, an average fish consumption rate of 6.5 g/day and average shellfish consumption rate of 0.57 g/day were used to calculate exposure doses.

For Squaxin Island Tribe adult, the 95th percentile per capita bottom fish intake rate of 24.1 g/day and shellfish intake rate of 59.4 g/day were used to calculate exposure doses [5]. For Squaxin Island Tribe children, the 90th percentile per capita total finfish intake rate of 12.4 g/day and shellfish intake rate of 8.6 g/day were used to calculate exposure doses [5].

Chemical Specific Toxicity

Below are general summaries of COC health effects. The public health implications of exposure to these COCs from sediments and tissues are discussed later.

Dioxins and Furans, and cPAHs TEQ concentrations

Although several dioxin and furan congeners were analyzed in tissue, only a single value, called a dioxin toxic equivalent (TEQ), is presented in this health consultation. Each dioxin/furan, or dioxin-like PCB congener, is multiplied by a Toxic Equivalency Factor (TEF) to produce the dioxin TEQ. The TEQs for each chemical are then summed to give the overall 2,3,7,8-tetrachlorodibenzo-p-dioxin TEQ. The TEQ approach is based on the premise that many dioxins/furans and dioxin-like PCB congeners are structurally and toxicologically similar to 2,3,7,8-tetrachlorodibenzo-p-dioxin. TEFs are used to account for the different potency of dioxins and furans relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin, and are available for ten chlorinated dibenzofurans and seven chlorinated dibenzodioxins using the World Health Organization (WHO) methodology [6]. A Similar TEQ approach is developed for each cPAH based on the relative potency to benzo(a)pyrene.

Dioxins and furans

Dioxins and furans (dioxins) consist of about 210 structural variations of dioxin congeners, which differ by the number and location of chlorine atoms on the chemical structure. The primary sources of dioxin releases to the environment are the combustion of fossil fuels and wood; the incineration of municipal, medical and hazardous waste; and certain pulp and paper processes. Dioxins also occur at very low levels from naturally occurring sources and can be found in food, water, air, and cigarette smoke.

The most toxic of the dioxin congeners, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) can cause chloracne (a condition of acne like lesions on the face and neck). Exposure to high levels of dioxins can cause liver damage, developmental effects and impaired immune function [7]. Long-term exposure to dioxins could increase the likelihood of developing cancer. Studies in rats and mice exposed to TCDD resulted in thyroid and liver cancer [8]. EPA considers TCDD to be a probable human carcinogen and developed a cancer slope factor of 1.5×10^5 mg/kg/day [9, 10].

Polycyclic Aromatic Hydrocarbons (PAHs)

Polycyclic aromatic hydrocarbons (PAHs) are generated by the incomplete combustion of organic matter, including oil, wood, and coal. They are found in materials such as creosote, coal, coal tar, and used motor oil. Base on structural similarities, metabolism, and toxicity, PAHs are often grouped together when one is evaluating their potential for adverse health effects. EPA has classified some PAHs – called cPAHs – as probable human carcinogens (B2) as a result of *sufficient* evidence of carcinogenicity in animals and *inadequate* evidence in humans [11].

Benzo(a)pyrene is the only cPAH for which EPA has derived a cancer slope factor. The benzo(a)pyrene cancer slope factor was used as a surrogate to estimate the total cancer risk of cPAHs in sediment. It should be noted, benzo(a)pyrene is considered the most carcinogenic of the cPAHs. The use of its cancer slope factor as a surrogate for total cPAH carcinogenicity may overestimate risk. To address this issue, DOH made an adjustment for each cPAH based on the relative potency to benzo(a)pyrene or TEQ [11].

Dietary sources make up a large percentage of PAH exposure in the U.S. population, and smoked or barbecued meats and fish contain relatively high levels of PAHs. The majority of dietary exposure to PAHs for the average person comes from ingestion of vegetables and grains (cereals) [12].

Polychlorinated biphenyls (PCBs)

PCBs are a mixture of man-made organic chemicals. There are no known natural sources of PCBs in the environment. The manufacture of PCBs stopped in the U.S. in 1977, because of evidence that PCBs could build up in the environment and cause toxic health effects. Although no longer manufactured, PCBs can still be found in certain products such as old fluorescent lighting fixtures, electrical devices or appliances containing PCB capacitors made before PCB use was stopped, old microscope oil, and old hydraulic oil. Prior to 1977, PCBs entered the environment (soil, sediment, water, air) during the manufacture and use of PCBs. Today, PCBs can still enter the environment from poorly maintained hazardous waste sites, illegal or improper dumping of PCB wastes such as old hydraulic oil, leaks from electrical transformers that contain PCB oils, and disposal of old consumer products that contain PCBs [13].

PCBs enter the environment as mixtures of individual components known as congeners. There are 209 variations of PCB congeners, which differ on the number and location of chlorine atoms on the chemical structure. Most PCBs commercially produced in the U.S. are composed of standard mixtures called Aroclors. The conditions for producing each Aroclor favor the synthesis of certain congeners, giving each Aroclor a unique pattern based on its congener composition. No Aroclor contains all 209 congeners. Once in the environment, PCBs do not easily breakdown and may stay in the soil for months or years. PCBs stick to soil and sediment and will not usually move deep into the soil with rainfall. Small amounts of PCBs can be found in almost all outdoor and indoor air, soil, sediments, surface water, and animals. As a result, PCBs are found worldwide. PCBs bioaccumulate in the food chain and are stored in the fat tissue. The major dietary source of PCBs is fish. PCBs are also found in meats and dairy products [13].

When direct exposure to contaminants occur, PCBs can get into people's bodies by ingestion, inhalation, and dermal (skin) contact. Some of the PCBs that enter the body are metabolized and excreted from the body within a few days; others stay in the body fat and liver for months and even years. PCBs collect in milk fat and can enter the bodies of infants through breast-feeding [13]. Skin irritation, vomiting, nausea, diarrhea, abdominal pain, eye irritation, and liver damage can occur in people exposed to PCBs [13].

Evaluating non-cancer hazards

Exposure assumptions for estimating contaminant doses from sediment and tissue exposures are found in Appendices A and B, Tables A1 and B1. In order to evaluate the potential for non-cancer adverse health effects that may result from exposure to contaminated media (i.e., air, water, soil, and sediment), a dose is estimated for each COC. These doses are calculated for situations (scenarios) in which a person might be exposed to the contaminated media. The estimated dose for each contaminant under each scenario is then compared to EPA's oral reference dose (RfD). RfDs are doses below which non-cancer adverse health effects are not expected to occur (so-called "safe" doses). They are derived from toxic effect levels obtained from human population and laboratory animal studies. These toxic effect levels can be either the lowest-observed adverse effect level (LOAEL) or a no-observed adverse effect level (NOAEL). In human or animal studies, the LOAEL is the lowest dose at which an adverse health effect is seen, while the NOAEL is the highest dose that does not result in any adverse health effects.

Because of data uncertainty, the toxic effect level is divided by "safety factors" to produce the lower and more protective RfD. If a dose exceeds the RfD, this indicates only the potential for adverse health effects. The magnitude of this potential can be inferred from the degree to which this value is exceeded. If the estimated exposure dose is only slightly above the RfD, then that dose will fall well below the observed toxic effect level. The higher the estimated dose is above the RfD, the closer it will be to the actual observed toxic effect level. This comparison is called a hazard quotient (HQ) and is given by the equation below:

$$HQ = \frac{\text{Estimated Dose (mg/kg-day)}}{\text{RfD (mg/kg-day)}}$$

Estimated exposure doses, exposure assumptions, and hazard quotients are presented in Appendix A for dioxins, cPAHs, and PCBs found in sediment. Based on exposure estimates quantified in Appendices A, the general population is not likely to experience adverse non-cancer health effects from exposure to chemical contaminants in Budd Inlet since, the exposure dose did not exceed the RfD.

Similarly, estimated exposure doses, exposure assumptions, and hazard quotients are presented in Appendix B for dioxins found in tissue. Although the Squaxin Island Tribe adult shellfish exposure scenario results in a dose that exceeds RfD, this exposure is likely to fall below the actual toxic effect levels due to the health protective nature of RfDs. Based on exposure estimates quantified in Appendices B, the general population and the Squaxin Island Tribe is not likely to experience adverse non-cancer health effects from exposure to chemical contaminants in Budd Inlet.

Evaluating Cancer Risk

Some chemicals have the ability to cause cancer. Theoretical cancer risk is estimated by calculating a dose similar to that described above and multiplying it by a cancer potency factor, also known as the cancer slope factor. Some cancer potency factors are derived from human

population data. Others are derived from laboratory animal studies involving doses much higher than are encountered in the environment. Use of animal data requires extrapolation of the cancer potency obtained from these high dose studies down to real-world exposures. This process involves much uncertainty.

Current regulatory practice assumes there is no “safe dose” of a carcinogen. Any dose of a carcinogen will result in some additional cancer risk. Theoretical cancer risk estimates are, therefore, not yes/no answers but measures of chance (probability). Such measures, however uncertain, are useful in determining the magnitude of a cancer threat because any level of a carcinogenic contaminant carries an associated risk. The validity of the “no safe dose” assumption for all cancer-causing chemicals is not clear. Some evidence suggests that certain chemicals considered to be carcinogenic must exceed a threshold of tolerance before initiating cancer. For such chemicals, risk estimates are not appropriate. Recent guidelines on cancer risk from EPA reflect the potential that thresholds for some carcinogenesis exist. However, EPA still assumes no threshold unless sufficient data indicate otherwise [14].

This document describes theoretical cancer risk that is attributable to site-related contaminants in qualitative terms like low, very low, slight and no significant increase in theoretical cancer risk. These terms can be better understood by considering the population size required for such an estimate to result in a single cancer case. For example, a low increase in cancer risk indicates an estimate in the range of one cancer case per ten thousand persons exposed over a lifetime. A very low estimate might result in one cancer case per several tens of thousands exposed over a lifetime and a slight estimate would require an exposed population of several hundreds of thousands to result in a single case. DOH considers theoretical cancer risk insignificant when the estimate results in less than one cancer per one million exposed over a lifetime. The reader should note that these estimates are for excess cancers that might result in addition to those normally expected in an unexposed population.

| <u>Theoretical Cancer Risk</u> | | |
|---|---------------------------|----------------------------|
| Theoretical Cancer risk estimates do not reach zero no matter how low the level of exposure to a carcinogen. Terms used to describe this risk are defined below as the number of excess cancers expected in a lifetime: | | |
| <u>Term</u> | | <u># of Excess Cancers</u> |
| moderate | is approximately equal to | 1 in 1,000 |
| low | is approximately equal to | 1 in 10,000 |
| very low | is approximately equal to | 1 in 100,000 |
| slight | is approximately equal to | 1 in 1,000,000 |
| insignificant | is less than | 1 in 1,000,000 |

Cancer is a common illness and its occurrence in a population increases with the age of the population. There are many different forms of cancer resulting from a variety of causes; not all are fatal. Approximately 1/4 to 1/3 of people living in the United States will develop cancer at some point in their lives [15].

Theoretical cancer risk estimates for exposure to bottom fish or shellfish by the general population and the Squaxin Island Tribe range from slight (2 cancers estimated per 1,000,000 exposed) to low (9 cancers estimated per 100,000 exposed). These estimates are within EPA’s acceptable risk for fish consumption. However, this is based on a limited number of shellfish samples. In addition, East Bay, West Bay and most of the North Inlet of Budd Inlet is closed for

shellfish harvesting, due to bacteriological conditions and or contaminants that may be present in concentrations that pose a risk for non-cancer related health effects for shellfish consumers.

Theoretical cancer risk estimates for exposure to cPAHs in sediments range from three cancers estimated per 10,000,000 exposed to nine cancers estimated per 10,000,000 exposed or insignificant. Cancer risk estimates for exposure to PCBs in sediments range from insignificant (5 cancers estimated per 10,000,000 exposed) to slight (1 cancer estimated per 1,000,000 exposed). Cancer risk estimates for exposure to dioxin in sediments range from insignificant (8 cancers estimated per 10,000,000 exposed) to slight (2 cancers estimated per 1,000,000 exposed).

Children's Health Concerns

The potential for exposure and subsequent adverse health effects often increases for younger children compared with older children or adults. ATSDR and DOH recognize that children are susceptible to developmental toxicity that can occur at levels much lower than those causing other types of toxicity. The following factors contribute to this vulnerability:

- Children are more likely to play outdoors in contaminated areas by disregarding signs and wandering onto restricted locations
- Children often bring food into contaminated areas, resulting in hand-to-mouth activities
- Children are smaller and receive higher doses of contaminant exposures per body weight
- Children are shorter than adults, therefore they have a higher possibility of breathing in dust and soil
- Fetal and child exposure to contaminants can cause permanent damage during critical growth stages

These unique vulnerabilities of infants and children demand special attention in communities that have contamination of their water, food, soil or air. Children's health was considered in the writing of this health consultation and the exposure scenarios treated children as the most sensitive population being exposed.

Conclusions

Based on the information provided, DOH concludes the following.

1. Dioxin and PCBs represents a "no apparent public health hazard" for children or adults exposed in a one-day-per-week or 52 days per year exposure scenario to contaminants present in sediments.

2. Dioxin represents a “no apparent public health hazard” for the general population and the Squaxin Island Tribe (children or adults) consuming bottom fish or shellfish in Budd Inlet.

Recommendations

1. DOH recommends Ecology perform additional beach sediment sampling at Priest Point Park, to obtain a statistically valid average for the evaluation of the data.
2. DOH recommends that TCHD signs at Priest Point Park beach remain until the beach sediment is fully characterized and evaluated.
3. DOH recommends Ecology perform additional biota sampling.

Public Health Action Plan

Action Planned

1. DOH will coordinate with TCHD and Ecology to provide an educational materials fact sheet.
2. DOH will provide copies of this health consultation to Ecology, TCHD, and concerned parties.

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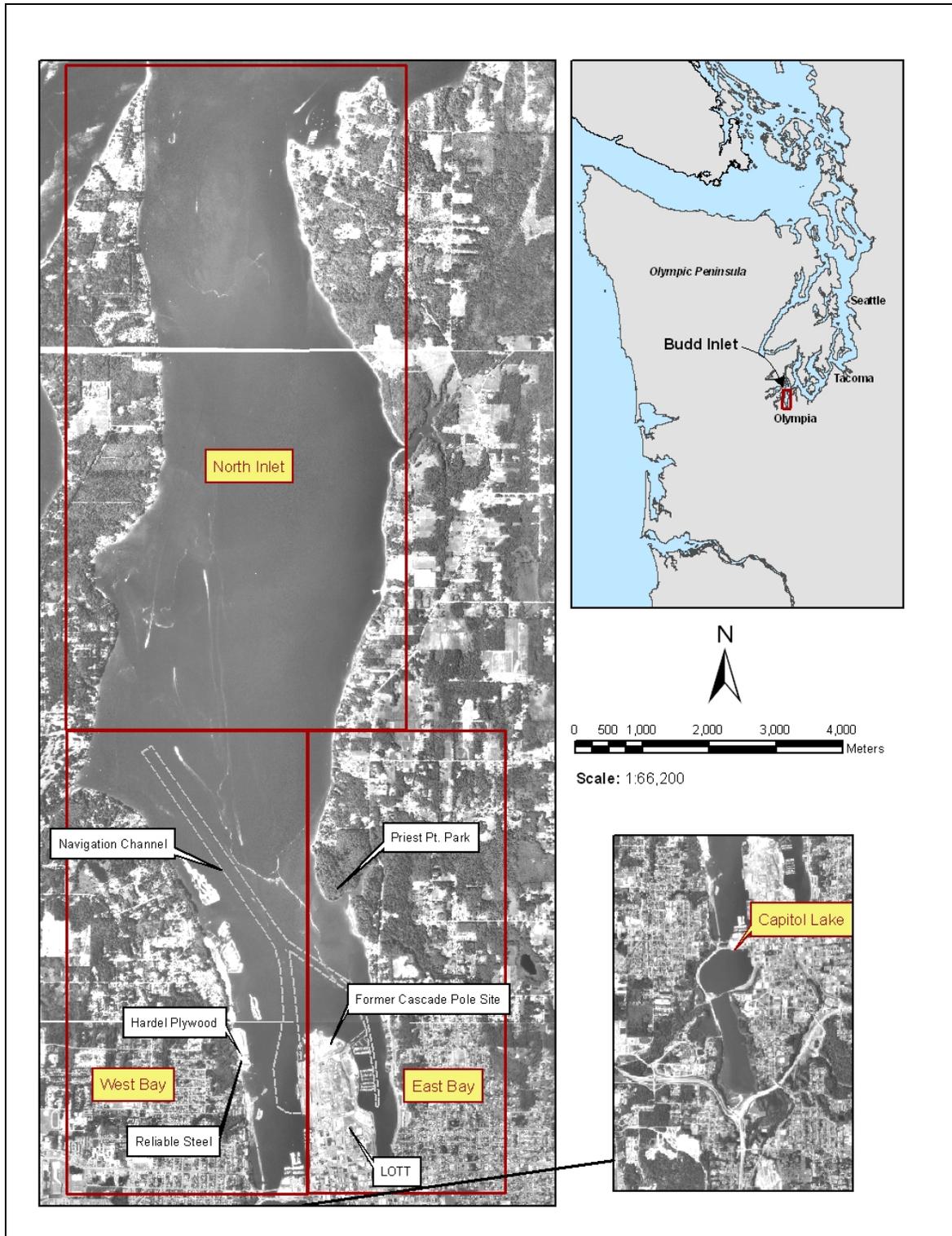
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Figure 1: Budd Inlet Site Overview



Appendix A

This section provides calculated exposure doses and assumptions used for exposure to chemicals in sediments at the Budd Inlet site. Three different exposure scenarios were developed to model exposures that might occur. These scenarios were devised to represent exposures to a child (0-5 yrs), an older child, and an adult. The following exposure parameters and dose equations were used to estimate exposure doses from direct contact with chemicals in soil.

Exposure to chemicals in soil via ingestion, inhalation, and dermal absorption.

Total dose (non-cancer) = Ingested dose + inhaled dose + dermally absorbed dose

Ingestion Route

$$\text{Dose}_{\text{(non-cancer (mg/kg-day))}} = \frac{C \times CF \times IR \times EF \times ED}{BW \times AT_{\text{non-cancer}}}$$

$$\text{Cancer Risk} = \frac{C \times CF \times IR \times EF \times CPF \times ED}{BW \times AT_{\text{cancer}}}$$

Dermal Route

$$\text{Dermal Transfer (DT)} = \frac{C \times AF \times ABS \times AD \times CF}{ORAF}$$

$$\text{Dose}_{\text{(non-cancer (mg/kg-day))}} = \frac{DT \times SA \times EF \times ED}{BW \times AT_{\text{non-cancer}}}$$

$$\text{Cancer Risk} = \frac{DT \times SA \times EF \times CPF \times ED}{BW \times AT_{\text{cancer}}}$$

Inhalation of Particulate from Sediment Route

$$\text{Dose}_{\text{non-cancer (mg/kg-day)}} = \frac{C \times SMF \times IHR \times EF \times ED \times 1/PEF}{BW \times AT_{\text{non-cancer}}}$$

$$\text{Cancer Risk} = \frac{C \times SMF \times IHR \times EF \times ED \times CPF \times 1/PEF}{BW \times AT_{\text{cancer}}}$$

Table A1. Exposure assumptions used for exposure to cPAHs, PCBs, and dioxin in sediments from Budd Inlet, Olympia, WA.

| Parameter | Value | Unit | Comments |
|---|---------------|-------------------------|---|
| Concentration (C) | Variable | mg/kg | Maximum detected value |
| Conversion Factor (CF) | 0.000001 | kg/mg | Converts contaminant concentration from milligrams (mg) to kilograms (kg) |
| Ingestion Rate (IR) – adult | 100 | mg/day | Exposure Factors Handbook [16] |
| Ingestion Rate (IR) – older child | 100 | | |
| Ingestion Rate (IR) - child | 200 | | |
| Exposure Frequency (EF) | 52 | Days/year | One days a week |
| Exposure Duration (Ed) | 30 (5, 10,15) | years | Number of years at one residence (child, older child, adult yrs). |
| Body Weight (BW) - adult | 72 | kg | Adult mean body weight |
| Body Weight (BW) – older child | 41 | | Older child mean body weight |
| Body Weight (BW) - child | 15 | | 0-5 year-old child average body weight |
| Surface area (SA) - adult | 5700 | cm ² | Exposure Factors Handbook |
| Surface area (SA) – older child | 2900 | | |
| Surface area (SA) - child | 2900 | | |
| Averaging Time _{non-cancer} (AT) | 1825 | days | 5 years |
| Averaging Time _{cancer} (AT) | 27375 | days | 75 years |
| Cancer Potency Factor (CPF) | 7.3 | mg/kg-day ⁻¹ | Source: EPA |
| 24 hr. absorption factor (ABS) | 0.13 0.14 | unitless | Source: EPA (Chemical Specific) PAH PCBs |
| Oral route adjustment factor (ORAF) | 1 | unitless | Non-cancer (nc) / cancer (c) - default |
| Adherence duration (AD) | 1 | days | Source: EPA |
| Adherence factor (AF) | 0.2 | mg/cm ² | Child, older child |
| | 0.07 | | Adult |
| Inhalation rate (IHR) - adult | 15.2 | m ³ /day | Exposure Factors Handbook [16] |
| Inhalation rate (IHR) – older child | 14 | | |
| Inhalation rate (IHR) - child | 8.3 | | |
| Soil matrix factor (SMF) | 1 | unitless | Non-cancer (nc) / cancer (c) - default |
| Particulate emission factor (PEF) | 1.45E+7 | m ³ /kg | Model Parameters |

Budd Inlet Sediment Exposure Route –Non-cancer

Table A2. Non-cancer hazard calculations resulting from exposure to cPAHs, PCBs, and dioxin in sediments from Budd Inlet, Olympia, WA.

| Contaminant | TEQ Concentration (ppm) (mg/kg) | Scenarios | Estimated Dose (mg/kg/day) | | | Total Dose | RfD/ MRL/ LOAEL (mg/kg/day) | Total Dose/ (RfD/ MRL/ LOAEL) |
|-----------------------|---------------------------------|-------------|------------------------------|--------------------------|----------------------------|------------|-----------------------------|-------------------------------|
| | | | Incidental Ingestion of Soil | Dermal Contact with Soil | Inhalation of Particulates | | | |
| cPAH | 0.69 | Child | 1.31E-6 | 4.94E-7 | 9.07E-11 | 1.80E-6 | 1.0E+1 | 0.0000002 |
| | | Older Child | 2.40E-7 | 1.81E-7 | 5.60E-11 | 4.12E-7 | | 0.00000004 |
| | | Adult | 1.37E-7 | 7.08E-8 | 3.46E-11 | 2.07E-7 | | 0.00000002 |
| Total PCBs (Aroclors) | 2.88 | Child | 5.47E-6 | 2.22E-6 | 3.79E-10 | 7.69E-6 | 2.0E-5 | 0.4 |
| | | Older Child | 1.00E-6 | 8.13E-7 | 2.34E-10 | 1.81E-6 | | 0.09 |
| | | Adult | 5.70E-7 | 3.18E-7 | 1.45E-10 | 8.88E-7 | | 0.04 |
| Total Dioxin TEQ | 0.00006 | Child | 1.14E-10 | 4.63E-11 | 7.89E-15 | 1.60E-10 | 1.0E-9 | 0.16 |
| | | Older Child | 2.08E-11 | 1.69E-11 | 4.87E-15 | 3.78E-11 | | 0.04 |
| | | Adult | 1.19E-11 | 6.63E-12 | 3.01E-15 | 1.85E-11 | | 0.02 |

Budd Inlet Sediment Exposure Route – Cancer

Table A3. Cancer hazard calculations resulting from exposure to cPAHs, PCBs, and dioxin in sediments from Budd Inlet, Olympia, WA.

| Contaminant | Concentration (ppm) | EPA cancer Group | Cancer Potency Factor (mg/kg-day ⁻¹) | Scenarios | Increased Cancer Risk | | | Total Cancer Risk |
|-----------------------|---------------------|------------------|--|-------------|------------------------------|--------------------------|----------------------------|-------------------|
| | | | | | Incidental Ingestion of Soil | Dermal Contact with Soil | Inhalation of Particulates | |
| Total cPAH TEQ | 0.69 | B2 | 7.3 | Child | 6.38E-7 | 2.40E-7 | 4.42E-11 | 8.78E-7 |
| | | | | Older Child | 2.33E-7 | 1.76E-7 | 5.45E-11 | 4.09E-7 |
| | | | | Adult | 1.99E-7 | 1.03E-7 | 5.05E-11 | 3.03E-7 |
| Total PCBs (Aroclors) | 2.88 | B2 | 2 | Child | 7.29E-7 | 2.96E-7 | 5.05E-11 | 1.03E-6 |
| | | | | Older Child | 2.67E-7 | 2.17E-7 | 6.23E-11 | 4.84E-7 |
| | | | | Adult | 2.28E-7 | 1.27E-7 | 5.78E-11 | 3.55E-7 |
| Total Dioxin TEQ | 0.00006 | B2 | 1.5E+5 | Child | 1.14E-6 | 4.63E-7 | 7.89E-11 | 1.60E-6 |
| | | | | Older Child | 4.17E-7 | 3.39E-7 | 9.74E-11 | 7.56E-7 |
| | | | | Adult | 3.56E-7 | 1.99E-7 | 9.03E-11 | 5.55E-7 |

Appendix B

This section provides calculated exposure doses and exposure assumptions used for chemicals in bottom fish and shellfish samples taken from Budd Inlet. These exposure scenarios were developed to model exposures that might occur and were devised to represent exposures to the general population and Squaxin Island Tribe. The following exposure parameters and dose equations were used to estimate exposure doses from ingestion of contaminants in bottom fish and shellfish.

Ingestion Route

$$\text{Dose}_{\text{(non-cancer (mg/kg-day))}} = \frac{C \times CF_1 \times IR \times CF_2 \times EF \times ED}{BW \times AT_{\text{non-cancer}}}$$

$$\text{Cancer Risk} = \frac{C \times CF_1 \times IR \times CF_2 \times EF \times CPF \times ED}{BW \times AT_{\text{cancer}}}$$

Table B1. Exposure assumptions used in exposure evaluation of contaminants in bottom fish and shellfish samples taken from Budd Inlet, in Olympia, Washington.

| Parameter | Value | Unit | Comments | |
|---|----------|--|---|--|
| Concentration (C) | Variable | ng/kg | Average detected value | |
| Conversion Factor (CF ₁) | 0.000001 | mg/ng | Converts contaminant concentration from nanograms (ng) to milligrams (mg) | |
| Conversion Factor (CF ₂) | 0.001 | kg/g | Converts mass of bottom fish and shellfish from grams (g) to kilograms (kg) | |
| Ingestion Rate (IR) | 0.57 | g/day | Body weight-adjusted consumption rates to account for children eating nearly 1.6 times as much fish per body weight as do adults (see table B2) - Shellfish | |
| | 6.5 | | Average general population child - fish | |
| Ingestion Rate (IR) | 8.61 | | 90 th percentile Squaxin Island Tribe child (all shellfish) [5] | |
| | 12.39 | | 90 th percentile Squaxin Island Tribe child (total finfish) | |
| Ingestion Rate (IR) | 1.7 | | Average general population adult - Shellfish | |
| | 17.5 | | Average general population adult - Fish | |
| Ingestion Rate (IR) | 59.43 | | 95 th percentile Squaxin Island Tribe adult (all shellfish) [5] | |
| | 24.15 | | 95 th percentile Squaxin Island Tribe adult (bottom fish) [5] | |
| Exposure Frequency (EF) | 365 | | Days/year | Assumes daily exposure |
| Exposure Duration (ED) | 6 | | years | Number of years at one residence (child) |
| Exposure Duration (ED) | 30 | Number of years at one residence (adult) | | |
| Body weight (BW) | 15 | kg | Mean body weight child | |
| Body weight (BW) | 70 | | Mean body weight adult | |
| Averaging Time _{non-cancer} (AT) | Variable | days | Equal to Exposure Duration | |
| Averaging Time _{cancer} (AT) | 25550 | days | 70 years | |
| Cancer Potency Factor (CPF) | Variable | mg/kg-day ⁻¹ | Source: EPA – Chemical specific | |

Table B2. Derivation of a child’s shellfish consumption rate for the general U.S. population.

| Row | Parameter | Adult | Child (0-6 yrs) |
|-----|---|----------------|-----------------|
| 1 | Reported All Fish Consumption Rate-gram fish per kg bodyweight per day (g/kg/day) | 0.277 | 0.433 |
| 2 | Ratio to Adult All Fish Consumption Rate | 1 | 1.6 |
| 3 | Reported Shellfish Consumption (g/day) | 1.70 (average) | Not Reported |
| 4 | Average Body Weight (kg) | 70 | 15 |
| 5 | Ratio to Adult BW | 1 | 0.21 |
| 6 | Adjusted Shellfish Consumption Rates (g/day) = Row 2 x Row 3 x Row 5 | 1.70 (average) | 0.57 (average) |

Table B3. Exposure dose and non-cancer risk from ingesting bottom fish and shellfish at the maximum concentrations of dioxin from Budd Inlet in Olympia, Washington.

| Species | Contaminant | Maximum Concentration (ppt) | | Estimated Dose (mg/kg/day) | | RfD (mg/kg/day) | Hazard quotient Average population | Hazard quotient 90-95 th percentile Squaxin Island Tribe |
|-------------|-------------|-----------------------------|-------|----------------------------|--|-----------------|------------------------------------|---|
| | | | | Average population | 95 th percentile Squaxin Island Tribe | | | |
| Shellfish | Dioxin | 1.58 | Child | 6.00E-11 | 9.07E-10 | 1.0E-9 | 0.06 | 0.91 |
| | | | Adult | 3.84E-11 | 1.34E-9 | | 0.04 | 1.34 |
| Bottom fish | | 1.18 | Child | 5.11E-10 | 9.75E-10 | | 0.51 | 0.97 |
| | | | Adult | 2.95E-10 | 4.07E-10 | | 0.30 | 0.41 |

Table B4. Cancer risk from ingesting bottom fish at the maximum concentrations of dioxin from Budd Inlet in Olympia, Washington.

| Species | Contaminant | Maximum Concentration (ppt) | Cancer Potency Factor (mg/kg-day ⁻¹) | | Increased Cancer Risk | |
|-------------|-------------|-----------------------------|--|-------|-----------------------|--|
| | | | | | Average population | 95 th percentile Squaxin Island Tribe |
| Shellfish | Dioxin | 1.58 | 1.5E+5 | Adult | 2.47E-6 | 8.62E-5 |
| Bottom fish | | 1.18 | | Adult | 1.95E-5 | 2.62E-5 |

Certification

The Washington State Department of Health prepared this Health Consultation under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It was completed in accordance with approved methodology and procedures existing at the time the health consultation was initiated. Editorial review was completed by the Cooperative Agreement partner.

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The Division of Health Assessment and Consultation, ATSDR, has reviewed this public health consultation and concurs with the findings.

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