Health Consultation

BURNETT CREEK FISH TISSUE
BRUNSWICK WOOD PRESERVING
BRUNSWICK, GLYNN COUNTY, GEORGIA

CERCLIS NO: GAD981024466

Prepared by
Georgia Department of Public Health

AUGUST 15, 2014

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

Health Consultation: A Note of Explanation

A health consultation is a verbal or written response from ATSDR or ATSDR's Cooperative Agreement Partners to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR or ATSDR's Cooperative Agreement Partner which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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Summary

The Glynn Environmental Coalition (GEC) requested a public health investigation of fish and seafood caught in Burnett Creek near the Brunswick Wood Preserving federal Superfund site. In response, the Georgia Department of Public Health (DPH) and GEC partnered to collect fish samples, so that the samples could be analyzed for contaminants that included dioxins, furans, metals, PAHs, and PCBs. DPH evaluated the sample results to determine if people exposed to these contaminants via fish consumption might be harmed. The conclusions presented below were based on a review and evaluation of the samples submitted for analysis. It is important to note that DPH's conclusions were based on a one-time sampling event. More sampling results from the same species (in addition to more species) captured for this health consultation over a longer period of time would more accurately describe any temporal and life-stage fluctuations in the contaminant levels found in fish inhabiting Burnett Creek. Site-specific fish consumption rates were determined by the GEC based on familiarity with the people who catch and eat fish and seafood from Burnett Creek.

Conclusion 1

Eating one to two meals per week of fish harvested from Burnett Creek is not likely to harm people from the very low levels of dioxins and furans found in the species analyzed.

Basis for Conclusion

Total dioxin/furan levels were below the U.S. Environmental Protection Agency's (EPA) screening levels for fish, as well as more conservative screening levels developed by DPH (using EPA's oral reference dose). DPH cannot calculate the cancer risks from exposure to the levels of dioxin and furans found in fish because EPA does not currently have a cancer potency factor from which to calculate a cancer risk. However, whole-fish samples have the maximum levels of dioxins and furans (as opposed to gutted and cleaned or filleted fish), and the estimated maximum possible dioxin/furan concentrations in fish ranged from 3 to 12 times below the most conservative screening values for non-cancer health effects. In addition, the most toxic form of dioxin (2,3,7,8-TCDD) was not detected in any sample.

Conclusion 2

Eating one to two meals per week of fish harvested from Burnett Creek is not likely to harm people from the very low levels of polycyclic aromatic hydrocarbons (PAHs) found in the species analyzed.

Basis for Conclusion

Total PAH levels were below EPA's screening levels for fish.

Conclusion 3

Eating two meals per week of seatrout, whiting, and black drum harvested from Burnett Creek may cause adverse non-cancer health effects from the levels of polychlorinated biphenyls (PCBs) found in these species.

Basis for Conclusion

The lack of human studies and unknown variability in toxicity between humans and laboratory animals (used to gather PCB toxicity data) leaves uncertainties regarding conclusions that can be drawn on the potential for adverse health effects from consuming fish harvested in Burnett Creek. Estimated PCB exposure doses from the consumption of seatrout and whiting are above the Agency for Toxic Substances and Disease Registry (ATSDR) minimal risk levels (MRLs) for both children and adults; while the estimated PCB exposure dose for children consuming black drum is also higher than the MRL. Furthermore, assuming that children and adults may be eating 2 meals of seatrout per week, they are subsequently exposed to PCB levels that are only 17 to 25 times lower the lowest observed adverse effects levels (LOAELs) found in the scientific literature, respectively. Moreover, children and adults that may be eating 2 meals of whiting per week are subsequently exposed to PCB levels that are only 25 to 62 times lower the LOAELs, respectively.

Conclusion 4

Eating one to two meals per week of fish harvested from Burnett Creek is not likely to harm people from the levels of inorganic arsenic found in the species analyzed.

Basis for Conclusion

Children and adults consuming two meals per week of the species analyzed would have an estimated exposure dose less than or equal to minimum risk level (MRL): an estimate of daily human exposure to a hazardous substance that is likely to be without an appreciable risk of adverse non-cancer health effects over a specified route and duration of exposure.

Conclusion 5

Based on the likelihood of joint, dual interaction of methylmercury and PCBs, people eating seatrout, whiting, and spot harvested in Burnett Creek once or twice a week, might be harmed by this consumption. People following the DNR fish consumption guidance by eating seatrout, whiting, and spot harvested in Burnett Creek only once a month would not likely be harmed.

Basis for Conclusion

There is *in vitro* evidence from one study that PCBs and methylmercury may synergistically decrease dopamine levels in rat brain cells presumably via disruption of calcium homeostatic mechanisms in neural cells leading to changes in neurotransmitter release (e.g., dopamine) or cell damage. But obvious synergism or additive joint action in affecting neurobehavioral endpoints was not demonstrated in a mouse *in vivo* study. If seatrout, whiting, and spot are consumed at the rate given in the scenarios described in this health consultation (one or two fish meals per week), the potential for impaired neurological function exists for adults and children eating these fish harvested from Burnett Creek. As long as community members fishing on Burnett Creek adhere to the fish consumption guidelines published by DNR: eat no more than one meal per month of seatrout, whiting, and spot, and no more than one meal per week of black drum, red drum and sheepshead, DPH concludes that community members will not likely be harmed by fish harvested in Burnett Creek.

Conclusion 6

Eating one to two meals per week of fish harvested from Burnett Creek has a low lifetime cancer risk associated with the levels inorganic arsenic, PCBs, and dioxin or dioxin-like compounds found in the species analyzed.

Basis for Conclusion

The estimated lifetime cancer risk for adults exposed to arsenic and PCBs in fish harvested and consumed from Burnett Creek over a 40 year period is low. For arsenic, approximately seven excess cancers can be expected from this exposure in 100,000 people eating two meals per week. For PCBs, approximately eight excess cancers can be expected from this exposure in 100,000 people eating two meals per week. And for dioxin and dioxin-like compounds, 5 excess cancers can be expected in 1,000,000 people eating two meals per week.

Conclusion 7

Following the DNR's *2013 Guidelines for Eating Fish from Georgia Waters*, and specifically adhering to the "Upper Turtle and Buffalo Rivers, Upriver of State Highway 303" recommendations, will be protective of human health for people catching and eating fish caught from Burnett Creek.

Basis for Conclusion

The recommendations published in the Guidelines are based on health-based risk calculations for someone eating fish over a period of 30 years or more. These Guidelines are not intended to discourage people from eating fish, but should be used as a guide for choosing which type (species) and size of fish to eat from Georgia waters. These Guidelines were designed to protect both children and adults from cancer and non-cancer health effects of these chemicals.

Next Steps

DPH will distribute this health consultation and a fact sheet summarizing our findings to the public, and work with the Glynn Environmental Coalition to ensure that health education reaches those residents fishing in Burnett Creek. DPH will provide the Coastal Health District and Glynn Environmental Coalition with multiple copies of the *2013 Guidance for Eating Fish from Georgia Waters* or distribution to area residents and visitors. As additional data become available, DPH will review the information and take appropriate actions. DPH will continue to respond to all requests for information and health concerns regarding the safety of consuming fish from Burnett Creek.

Statement of Issues

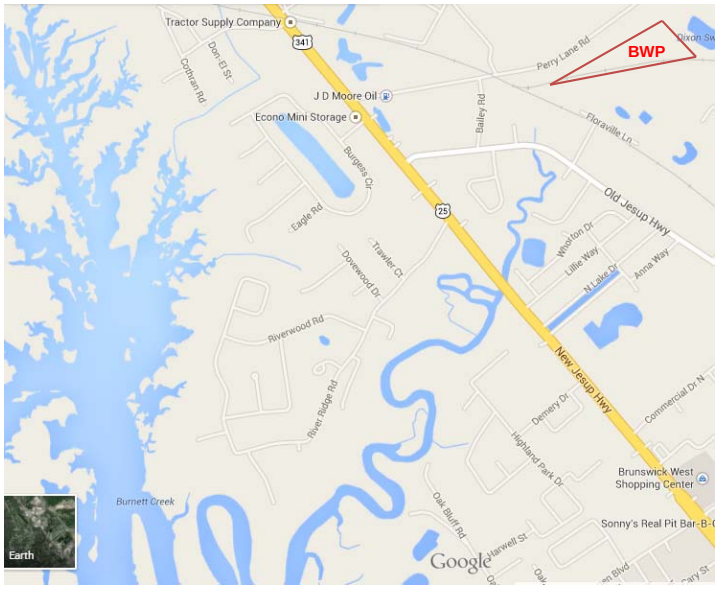
On June 26, 2012, the Glynn Environmental Coalition (GEC) petitioned the Agency for Toxic Substances and Disease Registry (ATSDR) to conduct a public health assessment to quantify human health risk from consuming fish from Burnett Creek, located adjacent to the Brunswick Wood Preserving Superfund site in Brunswick, Glynn County, Georgia. Specifically, GEC asked ATSDR to quantify human health risks from fish sampling data collected in 2000 and 2011. Under an existing Cooperative Agreement, ATSDR referred GEC's request to the Georgia Department of Public Health (DPH) for consideration [1]. DPH determined that in order to evaluate whether people eating fish from Burnett Creek were exposed to harmful levels of contaminants, fish would need to be harvested from Burnett Creek and analyzed for contaminant levels.

The purpose of this health consultation is to determine whether the community may have been harmed by exposure to site-related contaminants from eating fish caught in Burnett Creek, and what public health actions need to be taken to reduce harmful exposures.

Site Description and History

Brunswick Wood Preserving (BWP) operated as a wood treatment facility from 1958 to 1991.The site is located on Perry Lane Road in northeast Brunswick, Glynn County, Georgia. The site occupies 84 acres with a residential area to the south and Burnett Creek on the site's western boundary. Railroads and woodlands are located to the north and east of the site [2]. Drainage from the site flows into Burnett Creek, a tidally influenced estuarine stream used for fishing by local anglers and visitors. Figure 1 in the appendix shows an aerial view of Burnett Creek looking south of New Jesup Highway.

BWP used creosote, pentachlorophenol (PCP), and chromate copper arsenate as wood preservatives. Unlined surface impoundments, located on the east and west portions of the site, received wastewater from on-site processes. The site ceased operation in 1991, after the owners filed bankruptcy [2]. EPA conducted an emergency removal at the site, and subsequent federal and state regulatory agency investigations resulted in this site being listed as a federal Superfund[1]



[1] The Comprehensive Environmental Response, Compensation, and Liability Act of 1980, also known as Superfund, is the federal law that concerns the removal or cleanup of hazardous substances in the environment and at hazardous waste sites.

site in 1997 for releases of various regulated chemicals to soil and groundwater.

In response to the presence of contamination resulting from nearby industry unrelated to BWP (mercury and PCBs), the Georgia Department of Natural Resources (DNR) issued a fish consumption advisory for the "Upper Turtle and Buffalo Rivers, Upriver of State Highway 303" which includes Burnett Creek[2] in the late 1990s. This advisory recommends that no more than one meal of blue crab, sheepshead, red drum, black drum, and striped mullet caught upriver of Highway 303 be consumed per week. DNR also recommends that no more than one meal of croaker, spot, spotted seatrout, and whiting be consumed per month [3]. Because of the density of development in the vicinity of the upper Turtle and Buffalo Rivers upriver of Highway 303, the National Shellfish Sanitation Program has issued a shellfish ban, which recommends no consumption of shellfish (clams, mussels, and oysters) collected from those areas. No restriction has been recommended for the consumption of shrimp and flounder. DPH recommends that the local community follow these fish advisories and restrictions.

Area Demographics

Using 2010 U.S. Census data, ATSDR calculated population information for individuals living within a 1-mile radius beyond the property boundary of BWP. The population within one mile of the perimeter is approximately 3,073 people in 1,240 households. In this population are 603 women of child-bearing age and 282 children below age six. Figure 2 shows detailed demographic information as well as a scaled site location in relation to Burnett Creek.

Previous Public Health Involvement

Public Health involvement at BWP started in 1992 with the completion of a health consultation (HC) by ATSDR in response to a petition request to evaluate potential exposure to soil contaminants. The conclusions of this initial evaluation indicated that the contaminants, including PCP, dioxins, and polycyclic aromatic hydrocarbons (PAHs), were present in on-site soil at levels exceeding established health screening levels. ATSDR recommended that access to the site be restricted until soil cleanup was completed [4, 5].

BWP was added for the EPA's National Priorities List on April 1, 1997. In December 1997, a second petitioned HC was completed by ATSDR to evaluate available groundwater data and the potential for contaminated groundwater to migrate from the site. ATSDR concluded that public drinking water supply wells posed no apparent public health hazards as a result of site-related contamination based on 1995 and 1996 sampling of municipal wells. However, the potential public health implications from exposure to contaminants in private drinking water wells could not be evaluated. The conclusions of the December 1997 HC indicated that data to adequately characterize the groundwater conditions were not available. Private wells in the vicinity of BWP had not been regularly sampled for site-related contaminants since 1991. Because edible fish tissue data was not available for Burnett Creek at the time, ATSDR also concluded that consumption of fish from the creek by the local community could not be evaluated [6].

[2] The *2013 Guidelines for Eating Fish from Georgia Waters* can be found at www.gaepd.org/Files_PDF/gaenviron/GADNR_FishConsumptionGuidelines_Y2013.pdf.

In response to a community concerns, a HC was completed by ATSDR in December 1998 to further evaluate the potential for exposure to contaminants present in on-site soils [7]. Although elevated concentrations of contaminants, including PCP, PAHs, dioxins and furans, arsenic, and chromium were detected in on-site soil and sediment, ATSDR concluded that the potentially exposed individuals (such as workers and trespassers) were not likely to have frequent, long-term exposure to contaminants. Therefore, exposure to these individuals was not expected to results in adverse health effects. ATSDR concluded that soil samples collected along the perimeter of the BWP property contained low-level contamination and indicated that off-site soil was not likely to pose a public health hazard. While Burnett Creek sediment contained slightly elevated concentrations of dioxins, PAHs, and arsenic, ATSDR also concluded that exposure to sediments in Burnett Creek was infrequent and not likely to occur on a routine long-term basis. Therefore, exposure to sediment from Burnett Creek was not expected to pose a public health hazard. The December 1998 HC recommended that fish samples be collected from Burnett Creek to evaluate the possible health impacts from human consumption [7].

The results of both the December 1997 and December 1998 HCs were combined to create a public health assessment dated February 9, 1999 [8]. ATSDR received an additional request to evaluate off-site soil and sediment data obtained by EPA [3]. ATSDR evaluated this data and documented it in a HC released October 30, 2000 [9]. The HC concluded that none of the soil samples collected from five (off-site) residential areas exceeded the established health criteria. Therefore, adverse health effects from exposure to off-site soil were not likely to result. Off-site sediment samples from Burnett Creek revealed the presence of five PAHs, arsenic, and chromium at concentrations exceeding established health guidelines. However, further evaluation of the likely exposure, which included frequency and duration of typical exposures, and a review of available scientific literature indicated that adverse non-cancerous and cancerous health effects were not likely to occur.

Since the October 2000 HC was completed, additional data was collected at BWP by EPA. These data included more sediment samples collected from Burnett Creek, groundwater samples collected from nearby private drinking water wells, and fish and shellfish samples. Consequently, ATSDR released another HC evaluating these data on May 31, 2002 [4]. Sediment samples collected in November 2000 were taken downstream from sediment samples evaluated in the October 2000 HC. These samples were analyzed for semi-volatile organic compounds (SVOCs), including PAHs and PCP. Samples were also analyzed for dioxins and furans. Total dioxin TEQs[3] (toxic equivalencies) were the only contaminants detected in sediment above their established comparison value (0.0007 milligrams per kilogram [mg/kg] of sediment). However, based on further evaluation, ATSDR concluded that cancer and non-cancer health effects were not expected to result among individuals exposed through ingestion and dermal contact to total dioxin compounds in Burnett Creek sediment. Fish and shellfish sampling did not include larger edible fish samples because they were not available for capture during the field investigation and only 10 small mullet were caught at the time of the sampling effort. Mummichogs were also collected from Burnett Creek; however, they are not consumed by humans, but rather used as bait. The primary contaminants of concern were total dioxin TEQs. ATSDR concluded that although bioaccumulation in Burnett Creek is not expected and fish

[3] TEQ is defined as the sum of the products of the concentration of each dioxin and furan compound multiplied by its Toxic Equivalent Factor (TEF) value.

advisories recommended for Burnett Creek to reduce consumption of fish (due to other non-site-related contaminants, such as mercury and PCBs), the potential for exposure to dioxin compounds in larger fish species could not be completely evaluated in the absence of fish data for these larger species. Groundwater sample results from private wells at various residences and businesses were also evaluated in this HC. ATSDR concluded that adverse health effects from ingestion and dermal contact with groundwater from these private wells were not expected. However, ATSDR recommended that additional fish sampling be conducted, in an effort to collect large fish species that are most likely to be consumed by the community. These fish species include seatrout, red fish, flounder, and black drum.

Recent Public Health Involvement

During Fall 2012, DPH and GEC worked together to organize and host a fishing tournament with members of the GEC and other community participants. The purpose of this tournament was to collect larger, legal size, edible species of fish from Burnett Creek for whole fish laboratory analyses. A total of 33 legal-length fish were caught of various species including Atlantic croaker (*Micropogonias undulatus*), black drum (*Pogonias cromis)*, red drum (*Sciaenops ocellatus*), spot croaker (*Leiostomus xanthurus*), southern kingfish-whiting (*Menticirrhus americanus*) and spotted seatrout (*Cynoscion nebulosus*).

Local resident of Burnett Creek area during the 2012 fishing tournament sponsored by DPH/GEC

Prior to the fishing tournament, a significant rainfall event occurred on/or around October 8, 2012 when approximately six inches of rain fell in the Burnett Creek watershed. This amount of freshwater significantly reduced the normal salinity levels of Burnett Creek. Because the salinity levels in an estuarine river affect the feeding migration pattern of various species of saltwater fish, a return to normalcy was important to ensure that edible species of interest for sampling would populate Burnett Creek on the day of the fishing tournament. On various days from October 9, 2012 until the day of the fishing tournament on October 20, 2012, the GEC Project Manager measured the salinity at four different locations on Burnett Creek and at various depths to assess the salinity levels as they approached normalcy. In addition to salinity, the dissolved oxygen concentration and temperature were also measured (see Appendix A for all water quality measurements). High tide conditions existed during the fishing tournament; considered desirable for most fishing in the area. For the tournament, three sampling zones were established from which fish could be harvested. Sampling zones and the species harvested from these zones were:

- Zone 1: The mouth of Burnett Creek at the Cowpen Creek junction to US 341 (immediately downstream of US 341 Bridge – black drum, seatrout, whiting.
- Zone 2: US 341 to Old Jesup Road (immediately upstream of US 341 and Downstream of Old Jesup Road) – red drum.

- Zone 3: Old Jesup Road to Perry Lane Road (along a 100-foot reach between Perry Lane and Old Jesup Road) – no fish were caught.

Fish Sample Collection

Fish Sampling Areas

Fish were mainly captured in Zone 1 and no fish were captured in Zone 3. Because this sampling event was primarily a screening event, obtaining samples from Zone 1 was the priority area according to the GEC. The goal was to capture three fish per species of legal length; where the smallest fish in a composite sample was at least 75% of the size of the largest fish [10]. Three composite samples meeting the 75% rule[4] were captured from Zone 1 and a single fish sample was captured from Zone 2, which were submitted for laboratory analysis (Table 1).

Table 1: Composite Fish Samples Submitted for Laboratory Analysis

Sample Submitted	Species	Zone Capture	Weight (g)	Length (mm)	Legal Length (mm)
Composite 1	Black Drum	1	440 438 390	296 289 289	254
Composite 2	Spotted Seatrout	1	410 400 495	337 348 384	330.2
Composite 3	Southern Kingfish-Whiting	1	365 300 280	318 295 288	254
Single Fish	Red Drum	2	770	411	355.6

mm: millimeters
g: grams

Fish Sample Preparation and Handling

Captured fish were placed on ice and held in coolers until they were measured in millimeters and weighed in grams at the end of the tournament. Subsequently, the species that met the 75% rule were selected for composite samples 1 through 3 (as well as the single fish sample submitted) and measured again. This information was recorded and labels were made for each of the samples to be submitted. The fish were individually wrapped in aluminum foil and each sample was placed in plastic freezer storage bags and into a cooler filled with ice. Samples were transported to the University of Georgia (UGA) Marine Extension Service in Brunswick, placed in a laboratory freezer and held at -20° Celsius overnight. The following day, frozen samples were placed on ice and transported by vehicle to Atlanta. Frozen fish samples were kept on ice

[4]Adopted from the Georgia Department of Natural Resources guidelines from 1992: *Recommendations for a Fish Tissue Monitoring Strategy for Freshwater Lakes, Rivers and Streams.*

overnight and transported by vehicle to the UGA Agriculture and Environmental Services laboratory in Athens, Georgia the following morning.

Laboratory Information

GEC members prepare fish caught in Burnett Creek for storage, transport and laboratory analyses.

The contaminants chosen for whole-fish analysis were dioxins and furans, metals, pesticides, PCBs, and PAHs. For all analyses, whole-fish homogenates were used in order to evaluate potential maximum exposure to the contaminants. The UGA Agriculture and Environmental Services laboratory contracted the analysis of dioxins/furans and PAHs to SGS Analytical Perspectives, LLC in Wilmington, North Carolina, which analyzed whole-fish homogenates following the laboratory's quality assurance requirements. Data validation for the results was considered acceptable for use as qualified by DPH. All samples were analyzed for polychlorinated dibenzodioxins (dioxins) and polychlorinated dibenzofurans (furans) by high resolution gas chromatography/high resolution mass spectrometry using EPA Method 1613B. Twenty-five grams per sample were extracted for analysis. The laboratory met detection limits as follows: 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) for all samples at less than 0.08 parts per trillion (ppt) and 1,2,3,7,8-pentachlorodibenzo-p-dioxin (PeCDD) at 0.1 ppt or lower for all samples. The detection limit for all other dioxin and furan congeners[5] in the samples analyzed was 0.5 ppt or lower. PAHs were analyzed by gas chromatography/mass spectrometry using EPA Method 8270D-SIM[6]. Approximately 40 grams of each sample homogenate was used to prepare 0.5 milliliter extracts for analysis. The detection limits for all samples analyzed was approximately 0.06 parts per billion (ppb) for most PAHs except fluoranthene (approximately 0.1 ppb) and pyrene (less than 0.1 ppb).

The UGA Agriculture and Environmental Services laboratory conducted the metals, pesticides, and PCB analyses. For metals analysis, an acid digestion (EPA Method 3050B) was conducted followed by Inductively Coupled Plasma-Atomic Emission Spectrometry using EPA Method 6010B. The detection limit for all samples analyzed was 0.03 milligrams per kilogram (mg/kg--or parts per million). Mercury was analyzed using a manual cold vapor technique using EPA Method 7471B. The detection limit for mercury was 0.01 mg/kg. Pesticides were analyzed by first preparing the samples using a blender method for non-fatty foods using a method described

[5] A dioxin and/or furan congener is any single, well-defined chemical compound in the dioxin or furan family. There are 75 polychlorinated dibenzo-p-dioxin congeners, and 7 of them are specifically toxic. There are 135 polychlorinated dibenzofurans congeners, and 10 of them have dioxin-like properties.

[6]Selective Ion Monitoring (SIM): many remediation projects have clean-up objectives with the lowest reporting limit possible and the legal defensibility of mass spectrophotometer detection. This combination can be difficult because complex matrices can damage the mass spectrophotometer. Since GC/MS analysis is significantly more costly, the industry has developed Selective Ion Monitoring (SIM). The SIM technique allows environmental laboratories to analyze highly complex samples with minimal dilutions which results in minimal damage to the mass spectrophotometer. That allows low reporting limits and the enhanced defensibility of mass spectrophotometer analysis.

in FDA PAM Section 303[7]. The extract was cleaned by gel-permeation chromatography (GPC[8]) gel permeation cleanup using EPA Method 3640A. Organochlorine pesticides were then analyzed by gas chromatography using EPA Method 8081. The detection limit for pesticides ranged between 0.01 to 0.1 mg/kg. PCB's were analyzed by gas chromatography using EPA Method 8082. The detection limit for PCBs was 0.03 mg/kg.

Fish Tissue Analyses

Exposure Pathway

When a hazardous substance is released to the environment, people are not always exposed to it. Exposure happens when people breathe, eat, drink, or make skin contact with a contaminant. Several factors determine the type and severity of health effects associated with exposure to contaminants. Such factors include exposure concentration, frequency and duration of exposure, route of exposure, and cumulative exposures (i.e., the combination of contaminants and routes). Once exposure takes place, individual characteristics–such as age, sex, nutritional status, genetics, lifestyle, and health status–influence how that person absorbs, distributes, metabolizes, and excretes the contaminant. These characteristics, together with the exposure factors discussed above and the toxicological effects of the substance, determine health effects that may result.

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. A pathways analysis considers five principle elements: a source of contamination, transport through an environmental medium, a point of exposure, a route of human exposure, and a receptor population. Completed exposure pathways are those in which all five elements are present, and indicate that exposure to a contaminant has occurred in the past, is presently occurring, or will occur in the future. DPH regards people who come into contact with contamination as exposed. It should be noted that the identification of an exposure pathway does not imply that health effects will occur. Exposures may, or may not be substantive. Thus, even if exposure has occurred, human health effects may not necessarily result [11].

In general, people can be exposed to contaminants through ingesting soil and food, drinking water, inhaling vapors and dust, and by skin contact. Site-specific conditions and fish consumption patterns were considered in evaluating exposure to total TEQ dioxins/furans, PAHs, PCBs, pesticides, and metals from eating fish captured in Burnett Creek. Exposure to these contaminants in fish captured from Burnett Creek can occur through ingestion.

For chemicals like dioxins and PCBs that are persistent in the environment and build up in the food chain over time, contaminants in food are the primary source of exposure. Meat, dairy

[7]U.S. Department of Health and Human Services. Pesticide Analytical Manual, Volume 1, Section 303. Food and Drug Administration. 3rd Edition, October 1994.

[8] GPC is recommended for the elimination from the sample of lipids, polymers, copolymers, proteins, natural resins, cellular components, viruses, steroids, and dispersed high-molecular weight compounds. GPC is appropriate for both polar and non-polar analytes; therefore, it can be effectively used to cleanup extracts containing a broad range of analytes.

products, and fish contribute more than 90% of the dioxin intake for the public [12, 13]. Therefore, everyone has some dioxin in their body. Yet, for most, it is not life threatening; the health threat depends on the amount of and length of time a person is exposed to a contaminant.

Evaluation Process

A two-stage evaluation process was used in the assessment of whole-fish data. The first step involves a review of available sampling data and the selection of contaminants that warrant further evaluation, based on the potential for exposure to these contaminants to result in adverse health effects. DPH examines the types and concentrations of contaminants of concern, which are then screened with comparison values generally established by ATSDR and EPA. Comparison Values (CVs) are concentrations of a contaminant that can reasonably (and conservatively) be regarded as harmless to human health, assuming default conditions of exposure. CVs include ample uncertainty factors to ensure protection of sensitive populations. Because CVs do not represent thresholds of toxicity, exposure to contaminant concentrations above CVs will not necessarily lead to adverse health effects [11]. DPH then considers how people may come into contact with the contaminants. Because the level of exposure depends on the route, frequency, and duration of exposure and the concentration of the contaminants, this exposure information is essential to determine if a public health hazard exists.

The next step in the evaluation process involves an in-depth health-effects evaluation of the contaminants detected in the site media (in this case, fish) above their respective CVs. The primary focus of this effort is to evaluate the potential for the contaminant(s) to produce cancer and non-cancer health effects as a result of human exposure. This involves the consideration of site-specific factors, such as the exposure route (for example: ingestion, inhalation, of direct contact), the concentration of a contaminant in a particular media, and the frequency and duration of exposure. A more detailed description of both steps of the evaluation process is presented in Appendix B.

DPH used a conservative approach to evaluate whether contaminants in fish from Burnett Creek pose a possible health concern. Contaminants of concern (from the contaminants detected in the fish samples) were determined by employing a screening process. In general, health-based CVs or screening values used include EPA Regional Screening Levels (RSLs) for fish ingestion and ATSDR cancer risk evaluation guides (CREGs). CVs such as the RSL and CREG 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 CVs represent levels that are calculated to increase the estimated risk of cancer by about one additional cancer in a million people exposed.

DPH uses ATSDR CVs whenever available to make health-based decisions. In the absence of ATSDR CVs, DPH may also use EPA's health guideline values or other available values. In this health evaluation, total dioxin/furan levels were screened against EPA's fish CVs for non-cancer health effects, as well as more conservative, site-specific screening levels developed by DPH using EPA's oral reference dose (RfD) and site-specific fish consumption rates determined by the GEC based on community member interviews and familiarity with the fish and seafood consumption behaviors of local anglers and visitors to the Burnett Creek area (see Appendix B).

Consumption Scenarios

DPH established two consumption scenarios for fish captured in Burnett Creek[9]. From personal communication with the GEC, community members who capture fish in Burnett Creek usually eat one meal per week, and a maximum of two meals per week from the Burnett Creek harvest. Portion sizes are approximately 8 ounces per meal (oz./meal) (approximately 227 g/meal). Children's portion sizes are approximately 4 oz./meal (approximately 113 g/meal). The consumption rate for adults eating one meal per week (meal/week) is approximately 32.4 grams of fish per day (g/day), while the consumption rate for children eating one meal/week is approximately 16.2 g/day. The consumption rate for adults eating two meals/week is approximately 64.8 g/day, while the consumption rate for children eating one meal/week is approximately 32.4 g/day. According to the GEC, subsistence fishing is not known to occur in Burnett Creek alone. However, some subsistence fishing does occur in the community in the nearby Turtle River and Atlantic Ocean. For the purpose of estimating oral exposure doses from eating fish caught in Burnett Creek, DPH used the U.S. mean adult weight[10] (80 kg), and the U.S. mean child weight (18.6 kg) for children ages 3 to less than 6 years old.

Results

Chemicals of Concern

Dioxins and Furans

Polychlorinated dibenzo-*p*-dioxins and dibenzofurans (dioxins and furans) are two similar classes of chlorinated aromatic hydrocarbons that are produced as contaminants or byproducts. They have no known commercial or natural use. Dioxins are primarily produced during the incineration or burning of waste, the bleaching processes used in pulp and paper mills, and the synthesis of trichlorophenoxyacetic acid, trichlorophenol, and pentachlorophenol (PCP). Also, synthesis and heat-related degradation of PCBs will produce furan byproducts. Releases from industrial sources have decreased approximately 80% since the 1980s. Today, the largest release of these chemicals occurs as a result of open burning of household and municipal trash, landfill fires, and agricultural and forest fires [12].

As a result of the processes described above, most soil and water samples reveal trace amounts of dioxins and furans when advanced analytical techniques are applied. People in the general population are exposed primarily through ingestion of foods that are contaminated with dioxins and furans as a result of the accumulation of these substances in the food chain including high-fat foods, such as dairy products, eggs, and animal fats, and some fish and wildlife. Breast feeding is a substantial source for infants [12].

The most noted health effect in people exposed to large amounts dioxin is chloracne, a severe skin disease with acne-like lesions that occur mainly on the face and upper body. Changes in blood and urine that may indicate liver damage, alterations in glucose metabolism, and subtle

[9]Personal communication with Daniel Parshley, Project Manager, Glynn Environmental Coalition. June 2013.
[10]U.S. EPA analysis of NHANES 1999-2006 data.

changes in hormonal levels also are seen in people. Several studies suggest that exposure to dioxin increases the risk of several types of cancer in people. Very little is known about the health effects in people or animals from breathing or touching furans, and they have not been classified as to whether they can cause cancer.

Lipid-adjusted serum measurements and whole weight-based measurements of dioxins, furans, and coplanar PCBs (dioxin-like PCBs) were measured in a subsample of NHANES[11] 2003-2004 participants aged 20 years or older. Participants were selected within a specified age range to be a representative sample of the U.S. population. Of the dioxins and furans, octachlorodibenzo-*p*-dioxin (OCDD) typically is present at the highest concentration in the U.S. population but contributes little to the TEQ, with the other commonly detected dioxin and furan congeners being more than eight-fold lower in concentration [13]. The geometric mean serum concentration of OCDD is 220 picograms per gram of lipid (or parts per trillion) in the U.S. population ages 20 years and older from the 2003-2004 sample results [13].

Table 2 shows total dioxins/furans found in the four species of fish sampled. Sample results were expressed in picograms per gram of fish tissue (parts per trillion), which were converted to milligrams per kilogram of fish tissue for comparative purposes. DPH calculated the TEQs according to the 2005 World Health Organization re-evaluation of Human Toxic Equivalency Factors (TEF)[12] for dioxins and dioxin-like compounds [14]. Undetected congeners were assigned a concentration equal to half (1/2) the detection limit. Dioxin and dioxin-like laboratory sample results are presented in Appendix C Table 1. Not all congeners analyzed for were detected. The most toxic dioxin, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) was not detected in any sample [15]. The congeners found were:

- 1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin (1,2,3,6,7,8-HxCDD) detected in all four samples;
- 1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin (1,2,3,4,6,7,8-HpCDD) and octachlorodibenzo-*p*-dioxin (OCDD) detected in three samples;
- 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF) and 1,2,3,7,8-pentachlorodibenzofuran (1,2,7,8-PeCDF) detected in all four samples;
- 2,3,4,6,7,8-hexachlorodibenzofuran (2,3,4,6,7,8-HxCDF) and 1,2,3,4,6,7,8-heptachlorodibenzofuran (1,2,3,4,6,7,8-HpCDF) detected in one sample.

In general, organic compounds such as dioxins accumulate in lipid-rich tissues. The higher the lipid content, the higher the dioxin concentration. The UGA Agriculture and Environmental Services laboratory reported the black drum sample had a total lipid content of 1.24%, the seatrout sample had a total lipid content of 1.0%, the whiting sample had a total lipid content of 2.03%, and the red drum sample had a total lipid content of 0.64% [16].

[11]NHANES: National Health and Nutrition Examination Survey

[12] TEFs are used to weight the measured levels of the congeners present in a sample in relation to the most toxic dioxin congener, TCDD, which is defined as having a TEF of 1. The measured concentration of each congener is multiplied by the TEF weighting factor. The total dioxin-like toxic equivalency, or TEQ, is the sum of these products.

Table 2: Dioxin TEQ Concentrations Detected in Fish from Burnett Creek. Based on Consumption Scenarios Established for this Community

Species*	Total Dioxin TEQ[a] Concentration mg/kg	Estimated Maximum Possible TEQ Concentration[b] mg/kg	EPA Comparison Value[c] mg/kg	DPH Screening Value 1 meal/week mg/kg	DPH Screening Value 2 meals/week mg/kg
Black Drum	2.0×10^{-7}	4.6×10^{-7}	1.1×10^{-6}	Adult: 1.7×10^{-6} Child: 8.0×10^{-7}	Adult: 8.6×10^{-7} Child: 4.0×10^{-7}
Seatrout	5.0×10^{-8}	1.5×10^{-7}			
Whiting	3.0×10^{-7}	3.9×10^{-7}			
Red Drum	7.0×10^{-8}	1.85×10^{-7}			

*Black drum, seatrout, and whiting sample results are based on composite samples containing three fish per sample that were caught in Zone 1. The red drum sample result is based on a single fish caught in Zone 2.
[a] Toxic equivalent (TEQ): TEQs were calculated using mammalian dioxin and furan Toxic Equivalence Factors (TEFs) from Van den Berg et.al. (2006) and one-half the detection limit for undetected congeners [15]. The TEF for 1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin is 0.1, the TEF for 1,2,3,4,6,7,8-heptachlorodibenzi-*p*-dioxin is 0.01, the TEF for octachlorodibenzo-*p*-dioxin is 0.0008, the TEF for 2,3,7,8-tetrachlorodibenzofuran is 0.1, the TEF for 1,2,3,7,8-pentachlorodibenzofuran is 0.03, the TEF for 2,3,4,6,7,8-hexachlorodibenzofuran is 0.1, and the TEF for 1,2,3,4,6,7,8-heptachlorodibenzofuran is 0.01.
[b] EMPC: Represents an Estimated Maximum Possible Concentration. EMPC's arise in cases where the signal/noise ratio is not sufficient for peak identification (the determined ion-abundance ratio is outside the allowed theoretical range), or where there is co-eluting interference. The EMPC also represent all other PeCDD, HxCDD, HpCDD, PeCDF, HxCDF, and HpCDF congeners that may have been in the samples aside from the specific analytes screened for.
[c] Regional Screening Level (May 2014)
mg/kg: milligrams per kilogram

None of the fish sampled exceeded EPA's CV for TCDD or the DPH screening values. DPH will not further evaluate the potential for adverse non-cancer health effects from eating fish caught from Burnett Creek that are contaminated with dioxins and dioxin-like compounds, because:

- Whole-fish homogenates were sampled to show the maximum levels of dioxins and furans in all the fish sampled. Since the entire fish is generally not eaten (e.g. organs, brains, eyes, bones, cartilage, scales and fins), fish prepared for consumption will contain lower levels of contaminants; especially contaminants with an affinity to lipid-rich tissues.
- Maximum possible TEQ concentrations range from 3 to 12 times below conservative DPH screening values for non-cancer health effects.
- The most toxic dioxin, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) was not detected in any sample.

Dioxin and dioxin-like compounds were evaluated further for potential cancer risk and no increased risk for cancer was found—see Cancer Risk section below.

DPH concludes that based on the October 2012 sampling event, people (children and adults) eating one to two meals per week of fish harvested from Burnett Creek are not likely to be harmed by the levels of dioxins and furans found in some fish.

Polycyclic Aromatic Hydrocarbons

PAHs are a class of chemicals that result from the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances, such as tobacco and charbroiled meat. Exposure to these chemicals usually occurs as an exposure to mixtures of PAHs and not as individual chemicals. Exposure can occur through air, water, soil, or food. PAHs enter the air from motor vehicle exhaust, residential and industrial furnaces, tobacco smoke, volcanoes, agricultural burning, residential wood burning, and wildfires. Seasonal variations in exposure to PAHs are known to occur. The soil and water near industrialized areas can contain elevated concentrations of PAHs. Foods that contain PAHs include smoked, charcoal-broiled, roasted foods, and plant foods that become contaminated by atmospheric deposition. Cereal products (e.g., wheat, corn, oats, and barley) may contain PAHs because of methods used to dry them.

None of the fish samples exceeded EPA's CVs for the PAHs[13] (Appendix C, Table 2). Because CVs offer a high degree of protection and assurance that people are unlikely to be harmed by contaminants in the environment, DPH will not further evaluate the potential for adverse non-cancer health effects from eating fish contaminated with PAHs that were caught in Burnett Creek. Moreover, whole-fish (generally not consumed by humans) homogenates were sampled to show the maximum levels of PAHs in all the fish sampled. Benzo(b)fluoranthene, benzo(e)pyrene, and 1-methylnaphthalene were evaluated further for potential cancer risk and no increased risk for cancer was found—see Cancer Risk section below.)

Therefore, DPH concludes that based on the October 2012 sampling event, people (children and adults) eating one to two meals per week of fish harvested from Burnett Creek are not likely to be harmed by the levels of PAHs found in some fish.

Pesticides

Pesticides were not detected in any of the fish samples (Appendix C, Table 3).

Polychlorinated Biphenyls

PCBs are man-made organic chemicals that were commercially produced in the United States and other countries. PCBs inability to burn easily and resistance to degradation resulted in their wide use as coolants, lubricants, and insulation materials for transformers, capacitors, and other electrical equipment. Manufacturing of PCBs ceased in the U.S. in 1977 after evidence showed that PCBs persist in the environment, biomagnify in the food chain, and may potentially cause harmful health effects to people who are exposed [17]. In general, organic compounds such as PCBs accumulate in lipid-rich tissues. The higher the lipid content, the higher the PCB concentration. The most commonly observed health effects in people exposed to large amounts

[13]Since fish CVs are not available from ATSDR, the EPA RSLs for fish (November 2012) were used in the screening process.

of PCBs are skin conditions such as acne and rashes. Studies in exposed workers have shown changes in blood and urine that may indicate liver damage [17].

PCBs can move from water and sediments into fish through the ingestion of these media. Fish can accumulate much higher concentrations of PCBs than are observed in the water or sediment to which they are exposed, and then people can be exposed to PCBs by eating the contaminated fish. PCBs are known to accumulate in lipid-rich tissues. The higher the lipid content, the higher the PCB concentration. According to the EPA PCB Fish Advisory Fact Sheet, chemicals such as PCBs accumulate mainly in fatty tissues (i.e. belly flap, lateral line, subcutaneous and dorsal fat, dark muscle, gills, eye, brain, and internal organs). Removal of internal organs and skin and trimming the fat before cooking will decrease PCB exposure [18].

An Aroclor is a PCB mixture produced from approximately 1930 to 1979. It is one of the most commonly known trade names for PCB mixtures. Sample results showed that of all the PCBs analyzed for, only Aroclor-1268 was detected (Appendix C, Table 3). Although a CV specific to Aroclor-1268 does not exist, DPH used the EPA CV for Aroclor-1254 (a more commonly studied Aroclor for comparative and conservative purposes. Because the concentration of Aroclor-1268 exceeded the EPA CV for Anoclor-1254 (0.027 mg/kg) in all four fish samples, DPH evaluated the potential for adverse non-cancer and cancer health effects from eating fish caught from Burnett Creek that are contaminated with PCBs.

Metals

Samples were analyzed for antimony, total arsenic, beryllium, cadmium, chromium, copper, lead, mercury, nickel, selenium, silver, thallium, and zinc. Arsenic was the only metal that exceeded the EPA CV of 0.41 mg/kg for inorganic arsenic in all four fish samples (Appendix C, Table 4).

Arsenic is a naturally occurring element that is widely distributed in the Earth's crust. Elemental arsenic (sometimes referred to as metallic arsenic) is a steel grey solid material. In the environment, arsenic is combined with oxygen, chlorine, and sulfur to form inorganic arsenic compounds. Arsenic in animals and plants combines with carbon and hydrogen to form organic arsenic compounds that are much less harmful than inorganic arsenic.

Arsenic cannot be destroyed in the environment. It can only change its form, or become attached to or separated from particles. It may change its form by reacting with oxygen or other molecules present in air, water, or soil, or by the action of bacteria that live in soil or sediment.
Many common arsenic compounds can dissolve in water. Thus, arsenic can get into lakes, rivers, or groundwater by dissolving in rain or snow or through the discharge of industrial wastes. Some of the arsenic will stick to particles in the water or sediment on the bottom of lakes or rivers, and some will be carried along by the water. Ultimately, most arsenic ends up in the soil or sediment. Although some fish and shellfish take in arsenic, which may build up in tissues, most of this arsenic is converted by biotransformation to an organic form called arsenobetaine (commonly called "fish arsenic") [19].

In the past, BWP used chromate-copper arsenate as a wood preservative. Past disposal practices may have contributed to arsenic contamination in Burnett Creek. Because arsenic levels

exceeded the CV in all four fish samples (ranging from 0.68 – 1.65 mg/kg), DPH evaluated the potential for adverse non-cancer and cancer health effects from eating fish contaminated with arsenic.

Non-cancer Health Effects

Polychlorinated Biphenyls

Although only one sample from each of four different species was submitted for analysis, DPH chose to evaluate oral exposure dose estimations from consumption of each species. Each species may have different consumption patterns in Burnett Creek as well as in the other interconnected estuarine streams in the area where they feed [3]. Table 3 shows the estimated PCB oral exposure doses that people may be receiving by eating seatrout and whiting caught in Burnett Creek based on the consumption scenario established for this health consultation. The exposure dose estimations assume a 50% reduction in the level of PCBs from cleaning, skinning and cooking the fish[14]. DNR recommends no more than 1 meal/month of these two species captured in Burnett Creek be consumed.

Table 3: Estimated PCB Exposure Doses from Eating Seatrout and Whiting Captured in Burnett Creek Based on Consumption Scenarios Established for this Community

Species	Estimated Exposure Dose from Consuming 1 Meal/Week mg/kg/day	Estimated Exposure Dose from Consuming 2 Meals/Week mg/kg/day	ATSDR[a] MRL mg/kg/day
Seatrout	Adult: 0.00008 Child: 0.0002	Adult: 0.0002 Child: 0.0003	0.00002
Whiting	Adult: 0.00004 Child: 0.00009	Adult: 0.00008 Child: 0.0002	

mg/kg/day: milligrams of PCBs per kilogram of body weight per day
[a]ATSDR Health Guidelines (March 2013) based on Aroclor 1254

Table 4 shows the estimated PCB oral exposure doses that people may be receiving by eating black drum and red drum caught in Burnett Creek based on the consumption scenario established for this health consultation. DNR recommends that no more than 1 meal/week of these species captured in Burnett Creek be consumed.

[14] Based on a recommendation in the following reference: Great Lakes Fish Advisory Task Force Protocol Drafting Committee. *Protocol for a Uniform Great Lakes Sport Fish Consumption Advisory.* September 1993.

Table 4: Estimated PCB Exposure Doses from Eating Black Drum and Red Drum Captured in Burnett Creek Based on Consumption Scenarios Established for this Community

Species	Estimated Exposure Dose from Consuming 1 Meal/Week mg/kg/day	Estimated Exposure Dose from Consuming 2 Meals/Week mg/kg/day	ATSDR[a] MRL mg/kg/day
Black Drum	Adult: 0.00002 Child: 0.00005	Adult: 0.00005 Child: 0.0001	0.00002
Red Drum	Adult: 0.000007 Child: 0.0000015	Adult: 0.00001 Child: 0.00003	

mg/kg/day: milligrams of PCBs per kilogram of body weight per day
[a]ATSDR Health Guidelines (March 2013) based on Aroclor 1254

When deriving an MRL, ATSDR staff scientists review the toxicological literature to identify the lowest doses (in either animals or humans) that cause a harmful effect. These doses are referred to as the lowest observed adverse health effects level (LOAEL). When appropriate, ATSDR scientists select one of these LOAELs to derive the MRL. For some chemicals, the MRL is derived from a dose that does not cause harmful effects. This dose is referred to as the no observed adverse effects level (NOAEL).

For PCBs, ATSDR has developed a chronic oral MRL of 0.00002 milligrams per kilogram of body weight per day (mg/kg/day). The lowest dose identified (LOAEL) to cause harmful effects and the endpoint used for the ATSDR MRL derivation was 0.005 mg/kg/day based on a study of Rhesus monkeys self-ingesting capsules containing Aroclor 1254 in a glycerol/corn oil mixture (1:1). Monkeys who were exposed daily to this PCB dose for 23 months showed reduced antibody response when their immune system was challenged by sheep red blood cells [17]. To derive the chronic oral MRL, ATSDR divided the LOAEL of 0.005 mg/kg/day by an uncertainty factor of 300. The uncertainty factor used in the MRL determination included 10x for the use of the LOAEL, 3x for extrapolation from animals to humans, and 10x for human variability, resulting in an MRL of 0.000016 mg/kg/day. This dose was rounded to 0.00002 mg/kg/day [17].

It is important to know that the estimated PCB exposure doses in people who eat fish caught in Burnett Creek will be compared to ATSDR's chronic oral MRL of 0.00002 mg/kg/day. An easy way to determine if the estimated dose is less than or greater than the MRL is to determine the PCB hazard quotient (HQ). The HQ is derived by dividing the estimated PCB dose by the MRL of 0.00002 mg/kg/day. Whenever the HQ is below 1, then the estimated dose is below the MRL and non-cancerous harmful effects are not expected. When the HQ exceeds 1, then the estimated dose exceeds the MRL.

The HQ exceeds 1 for children eating 2 meals/week for all four species analyzed. The HQ ranges from 1.5 for a child eating 2 meals/week of red drum to 15 for a child eating 2 meals/week of seatrout. For adults eating 2 meals/week, the HQ exceeds one for all species analyzed except red drum. The HQ ranges from 0.5 for an adult eating 2 meals/week of red drum to 10 for an adult eating 2 meals/week of seatrout.

The HQ exceeds 1 for children eating 1 meal/week for all species analyzed except red drum. The HQ ranges from 0.75 for a child eating 1 meal/week of red drum to 10 for a child eating 1

meal/week of seatrout. For adults eating 1 meal/week, the HQ also exceeds one for all species sampled except red drum. The HQ ranges from 0.35 for an adult eating 1 meal/week of red drum to 4 for an adult eating 1 meal/week of seatrout.

It is important to note that both seatrout and whiting (where the DNR recommendation is no more than 1 meal/month of these two species) contained the highest PCB concentrations of the four species analyzed. Interestingly, whiting contained the highest total lipid content (2.03%) measured by UGA in the samples analyzed, while seatrout contained the third highest total lipid content (1%). Red drum contained the lowest total lipid content (0.64%) as well as the lowest PCB concentration. Except for red drum, the HQ's are greater than 1 for both adults and children eating 1 to 2 meals of the three remaining species of fish harvested from Burnett Creek per week. Therefore, we have to more thoroughly evaluate whether harmful health effects are expected from this consumption.

The most sensitive endpoints identified in animal studies showed immunological, dermal, developmental effects in monkeys given daily PCB doses of 0.005 to 0.0075 mg/kg/day. The exposure duration for most of these monkeys was 23 to 72 months [17]. At slightly higher daily doses (0.02 to 0.04 mg/kg/day), PCBs caused fetal and post-partum deaths in pregnant monkeys along with significantly reduced conception rates and decreased serum cholesterol levels [17].

Immunological Effects in Animals

Low-level PCB exposure in monkeys showed reduced IgG and IgM antibodies and a temporary reduction in B lymphocytes from exposure to sheep red blood cells (SRBC). While this effect was observed at a daily dose of 0.005 mg/kg/day Aroclor 1254 in monkeys, this and other immunological effects were observed at higher doses. For example, at a daily dose of 0.2 mg/kg/day Aroclor 1248 for 11 months, monkeys showed decreased anti-SRBC hemolysin titers. Guinea pigs exposed to a daily dose of 0.8 mg/kg/day for 8 weeks showed decreased gamma globulin-containing cells in their lymph nodes. At higher daily doses (0.5 to 1.3 mg/kg/day) ranging from 1 to 6 months, mice showed increased susceptibility to leukemia virus and increased sensitivity to bacterial endotoxin [17].

Skin Effects in Animals

Low-level PCB exposure of 0.005 mg/kg/day in monkeys exposed for 72 months has shown damage to fingernails and toenails. At slightly higher doses (0.1 mg/kg/day for 2 months), harmful effects in monkeys included facial edema, acne, inflammation of hair follicles, and hair loss. Longer exposure to 0.1 mg/kg/day in monkeys also caused fingernail loss and cellular changes in gums [17].

Developmental Effects During and After Pregnancy in Animals

Developmental effects refer to effects that occur during pregnancy and following birth as the infant grows. In animals, lower birth rate and hyperpigmentation of the skin was reported in the offspring of monkeys treated before mating and during gestation with 0.03 mg/kg/day Aroclor 1016. Monkeys exposed during pregnancy to 0.005 mg/kg/day Aroclor 1254 and via breast milk

for 22 weeks after birth resulted in offspring with inflamed and enlarged tarsal glands[15], as well as nail and gum lesions [17].

For chronic exposure studies greater than 1 year, the lowest known level to cause harmful health effects in monkeys (0.005 mg/kg/day) used Aroclor 1254; therefore, some uncertainty exists when using this value to assess the effects of other Aroclor mixtures. However, because Aroclor 1268 is more chlorinated than Aroclor 1254, it is likely to be more toxic.

In this evaluation, the estimated exposure dose for children eating 2 meals/week of seatrout is approximately 17 times lower than the LOAEL (observed in monkeys) and ranges up to approximately 167 times lower than the LOAEL for children eating 2 meals/week of red drum. The estimated exposure dose for children eating 1 meal/week of seatrout is approximately 25 times lower than the LOAEL and ranges up to approximately 3,333 times lower than the LOAEL for children eating 1 meal/week of red drum. For adults, the estimated exposure dose for eating 2 meals/week of seatrout is approximately 25 times lower than the LOAEL and ranges up to approximately 500 times lower than the LOAEL for adults eating 2 meals/week of red drum. The estimated exposure dose for adults eating 1 meal/week of seatrout is approximately 63 times lower than the LOAEL and ranges up to approximately 714 times lower than the LOAEL for eating 1 meal/week of red drum.

The lack of human studies and unknown variability in toxicity between humans and laboratory animals (used to gather PCB toxicity data) leaves uncertainties regarding conclusions that can be drawn on the potential for adverse health effects from consuming fish harvested in Burnett Creek. However, assuming that children and adults may be eating 2 meals of seatrout per week, they are subsequently exposed to PCB levels that are only 17 to 25 times lower the LOAELs, respectively. Moreover, children and adults that may be eating 2 meals of whiting per week are subsequently exposed to PCB levels that are only 25 to 62 times lower the LOAELs, respectively. Therefore, DPH expects that both children and adults may be at increased risk for immune deficiencies (even if they are asymptomatic) and dermal effects (damage to fingernails and toenails) from exposure to PCBs in fish harvested from Burnett Creek.

DPH concludes that based on the October 2012 sampling event, people (children and adults) eating one to two meals of seatrout and whiting per week of fish harvested from Burnett Creek are likely to experience adverse non-cancer health effects from the levels of PCBs found in these fish. This conclusion is especially targeted towards children consuming 2 meals/week of not only seatrout and whiting, but also black drum.

Arsenic

As previously mentioned, past disposal practices from the wood treating operation at BWK were a likely source of arsenic contamination in Burnett Creek. In general, inorganic arsenic, the more toxic form of arsenic, accounts for 1.5% of the total arsenic in fish and 20% of arsenic in shellfish, but this percentage varies widely [20]. The general consensus in the literature is that

[15] The tarsal glands are sebaceous glands on the rim of the eyelids. They supply sebum, an oily substance that stops evaporation of the eye's tear film, prevents tear spillage onto the cheek, and makes the closed lids airtight. Glands are located on the upper and lower eyelids.

about 85-90% of the arsenic in the edible parts of marine fish and shellfish is organic arsenic (e.g., primarily arsenobetaine, but also arsenochloline, dimethylarsinic acid) and that approximately 10% is inorganic arsenic [21]. Let us assume conservatively that the amount of inorganic arsenic generally present in fish is 10% is used this estimate an exposure dose to inorganic arsenic from fish consumed in Burnett Creek. Therefore, if the total arsenic concentration found in seatrout is 0.68 mg/kg, 10% of that arsenic concentration (0.068 mg/kg) would be derived from inorganic arsenic. Similarly, the inorganic arsenic concentrations of the remaining species analyzed are expected to be: whiting (0.077 mg/kg), black drum (0.165 mg/kg), and red drum (0.095 mg/kg).

Although only one sample from each of four different species was submitted for analysis, DPH chose to evaluate oral exposure dose estimations for each species. Each species may have different consumption patterns in Burnett Creek as well as in the other interconnected estuarine streams in the area where they feed [3]. Table 5 shows the estimated inorganic arsenic oral exposure doses that people may be receiving by eating seatrout and whiting caught in Burnett Creek based on the consumption scenario established for this health consultation. These exposure dose estimations are conservatively based on a 10% inorganic arsenic concentration. DNR recommends no more than 1 meal/month of these two species captured in Burnett Creek be consumed.

Table 5: Estimated Inorganic Arsenic Exposure Doses from Eating Seatrout and Whiting Captured in Burnett Creek Based on Consumption Scenarios Established for this Community

Species	Estimated Exposure Dose from Consuming 1 Meal/Week mg/kg/day	Estimated Exposure Dose from Consuming 2 Meals/Week mg/kg/day	ATSDR[a] MRL mg/kg/day
Seatrout	Adult: 0.00003 Child: 0.00006	Adult: 0.00006 Child: 0.0001	0.0003
Whiting	Adult: 0.00003 Child: 0.00007	Adult: 0.00006 Child: 0.0001	

mg/kg/day: milligrams of PCBs per kilogram of body weight per day
[a]ATSDR Health Guidelines (March 2013) for inorganic arsenic

Table 6 shows the estimated inorganic arsenic oral exposure doses that people may be receiving by eating black drum and red drum caught in Burnett Creek based on the consumption scenario established for this health consultation. DNR recommends that no more than 1 meal/week of these species captured in Burnett Creek be consumed.

Table 6: Estimated Inorganic Arsenic Exposure Doses from Eating Black Drum and Red Drum Captured in Burnett Creek Based on Consumption Scenarios Established for this Community

Species	Estimated Exposure Dose from Consuming 1 Meal/Week mg/kg/day	Estimated Exposure Dose from Consuming 2 Meals/Week mg/kg/day	ATSDR[a] MRL mg/kg/day
Black Drum	Adult: 0.00007 Child: 0.0001	Adult: 0.0001 Child: 0.0003	0.0003
Red Drum	Adult: 0.00004 Child: 0.00008	Adult: 0.00008 Child: 0.0002	

mg/kg/day: milligrams of PCBs per kilogram of body weight per day
[a]ATSDR Health Guidelines (March 2013) for inorganic arsenic

For arsenic, ATSDR has a chronic oral MRL of 0.0003 mg/kg/day. It is important to note that this MRL is based on inorganic arsenic consumed by drinking well water. Arsenic found in fish is most likely organic arsenic in the form of arsenobetaine. However, almost no information is available on the effects of organic arsenic on humans. ATSDR's chronic oral MRL for arsenic is based on a study conducted in Taiwan where a large number of farmers were exposed to high levels of naturally occurring arsenic in well water. In this study, the incidence of blackfoot disease and dermal lesions (hyperkeratosis and hyperpigmentation) was investigated. In cases of low-level chronic arsenic exposure (usually from water), these skin lesions appear to be the most sensitive indication of an adverse health effect. A control group used in the study showed NOAEL effects at 0.0008 milligrams per kilogram per day (mg/kg/day). In this study, the LOAEL was determined to be 0.014 mg/kg/day, where hyperpigmentation and keratosis of the skin were observed. To derive the chronic oral MRL, ATSDR divided the NOAEL of 0.0008 mg/kg/day by an uncertainty factor of 3 for human variation, resulting in an MRL of 0.00026 mg/kg/day. This dose was rounded to 0.0003 mg/kg/day [19].

In this evaluation, estimated inorganic arsenic exposure doses are below the MRL for both children and adults eating 1 meal/week of any of the four species reviewed in this health consultation. Except for children eating 2 meals/week of black drum, where the estimated inorganic arsenic exposure dose equaled the MRL; the estimated inorganic arsenic exposure doses were below the MRL for both children and adults eating 2 meals/week any of the remaining three species reviewed for this health consultation. An MRL is an estimate of daily human exposure to a hazardous substance at or below which that substance in unlikely to pose a measurable risk of harmful, noncancerous health effects. Moreover, a more accurate assumption of the concentration of inorganic arsenic likely to be found in the fish sampled (1.5 to 2%) would show estimated exposure doses for children and adults consuming fish caught in Burnett Creek to be approximately 5 times lower than the exposure doses estimated by DPH

Therefore, DPH concludes that based on the October 2012 sampling event, people (children and adults) eating one to two meals per week of fish harvested from Burnett Creek are not likely to experience non-cancer health effects from the levels of arsenic found in some fish.

Interactions of Chemicals Found in Fish

Chlorinated dibenzo-*p*-dioxins (CDDs), methylmercury, and PCBs can occur with high frequency in water, sediment and fish in certain water bodies in the United States, and occur, to varying degrees in human milk, dairy products, and meat. The major limitation of this health consultation is that each chemical found in fish harvested from Burnett Creek is treated individually and conclusions are derived from individual components. It is not entirely accurate to base fish advisories on the most toxic/highest concentration scenarios, without considering joint toxicity of chemicals that have similar toxicity endpoints.

To carry out exposure-based assessments of possible health hazards associated with oral exposure scenarios involving frequent dietary exposure to mixtures of CDDs, methylmercury, and PCBs, component-based approaches that assume additive joint action of the components are

recommended for exposure based screening assessments [22]. The additivity assumption appears to be in the interest of public health since the components have several shared toxicity targets. This approach is recommended because of the lack of studies that examine relevant endpoints and describe dose-response relationships for oral exposures that contain the three components of concern. For noncancer endpoints, a target-organ toxicity dose modification of the hazard index approach is recommended by ATSDR given that a wide range of overlapping toxicity targets can be affected by the components [22].

Because CDDs, methylmercury, and PCBs share target organs of toxicity; namely neurological development, ATSDR recommends the use of Hazard Index (HI) to evaluate the whole mixture. For example,

$$HI_{DEV} = \frac{E_{TCDD}}{MRL_{TCDD\ DEV}} + \frac{E_{MeHg}}{MRL_{MeHg\ DEV}} + \frac{E_{PCB}}{MRL_{PCB\ DEV}}$$

where HI_{DEV} is the hazard index for developmental toxicity (the most sensitive biological endpoint), E_{TCDD} is the exposure dose to 2,3,7,8-TCDD (expressed in the same units as the corresponding MRL), $MRL_{TCDD\ DEV}$ is the MRL for 2,3,7,8-TCDD which is based on developmental toxicity ($1x10^{-9}$ mg/kg/day), and so forth. *MeHg* stands for methylmercury.

Preliminary evidence that an exposure to the mixture may constitute a hazard is provided when the HI for a particular exposure scenario and health endpoint exceeds 1. In practice, concern for the possibility of a health hazard increases with increasing value of the HI above 1.

DPH used this approach to determine if the component-based approach HI, and therefore potential for developmental toxicity, is above 1 when looking at the combined HI's for TCDD, methyl mercury, and PCB's. The individual HI's and combined HI for adults and children eating one or two meals a week are shown in Table 7.

Table 7: Hazard indexes for developmental toxicity components (TEQ, methyl mercury, and PCBs) individually and combined.

Exposure Scenario	Exposure $Dose_{TEQ}/MRL_{TCDD}$	Exposure $Dose_{MeHg}/MRL_{MeHg}$	Exposure $Dose_{PCB}/MRL_{PCB}$	Hazard Index
Adult (1 meal/week)	0.1	0.33	10	10.34
Adult (2 meals/week)	0.2	1.0	15	16.2
Child (1 meal/week)	0.3	1.0	15	16.3
Child (2 meals /week)	0.5	2.0	35	37.5

$Dose_{TEQ}/MRL_{TCDD}$: for example, 1.2×10^{-10} (mg/kg/day)/1.0×10^{-9} (mg/kg/day) for an adult eating one meal per week.
$Dose_{MeHg}/MRL_{MeHg}$: for example, 1.0×10^{-4} (mg/kg/day)/3.0×10^{-4} (mg/kg/day) for an adult eating one meal per week.
$Dose_{PCB}/MRL_{PCB}$: for example, 3.0×10^{-4} (mg/kg/day)/2.0×10^{-5} (mg/kg/day) for an adult eating one meal per week.

Note: The highest dioxin total TEQ concentration found in whiting, highest concentration of methylmercury found seatrout and whiting, and the highest concentration of PCBs found seatrout were used to estimate exposure doses for the consumption scenarios described. All significant digits for estimated exposure doses were rounded to the nearest whole number.

Table 7 shows that in all cases, using the highest concentrations found in the fish samples, the combined HI is greater than one, with PCBs being the strongest component contributing to the HI. Thus, the chemical mixture warrants a further health hazard assessment. With component-based approaches to assessing health hazards from mixtures, it is important to assess the joint additive action assumption, and consider the possibility that less-than-additive or greater-than additive joint actions may occur among the components. To do this, ATSDR developed a binary weight-of-evidence (BINWOE) system for the assessment of chemical interactions based on the quality of available research data. The numerical scale of this system range from -1 for high confidence that a less-than-additive joint action will occur, through 0 for evidence that additive joint action will occur or for indeterministic evidence for the mode of joint action, up to +1 for high confidence that a greater-than-additive interaction would occur. Appendix D details the BINWOE classification system that uses factors to describe the direction of chemical interaction as well as an approach to weigh the quality of the data for these components. The results of this component-based interactive approach is detailed in Appendix D.

From looking at the potential joint interactive effects that dioxins, methylmercury, and PCBs might have on each other's toxicity by looking at dual combinations of these chemicals, it appears that the combined effects may exacerbate the potential for adverse health effects on people consuming fish harvested from Burnett Creek. However, when looking at dioxins, we have to consider that all the binary studies reviewed are based on the toxicity of TCDD; not the TEFs of the dioxins found in Burnett Creek fish. The TEFs of dioxins and furans found in Burnett Creek fish range from 0.1 to 0.0003, which specifies that these components are between 10 to 30,000 times less toxic than TCDD. In addition, no dioxin-like PCBs were detected in fish samples. Some of the studies reviewed for dual, joint action were conducted with dioxin-like PCBs. However, most of the evidence for joint, dual toxicity comes from the action of methylmercury on PCBs and vice versa. Chemicals such as PCBs accumulate mainly in fatty tissues (i.e. belly flap, lateral line, subcutaneous and dorsal fat, dark muscle, gills, eye, brain, and internal organs) and removal of internal organs and skin and trimming the fat before cooking will decrease PCB exposure.

Therefore, as long as community members fishing on Burnett Creek adhere to the fish consumption guidelines published by DNR; that is eat no more than one meal per month of seatrout, whiting, and spot, and no more than one meal per week of black drum, red drum and sheepshead, DPH concludes that community members will not likely be harmed by fish harvested in Burnett Creek.

However, if seatrout, whiting, and spot are consumed at the rate of the scenarios described in this health consultation, the potential for subtle neurological impairment exists for adults and children eating these fish harvested from Burnett Creek.

Cancer Risks

2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) is considered by the International Agency for Research on Cancer (IARC) to be carcinogenic to humans (sufficient human evidence) [23], and to be a known human carcinogen by the National Toxicology Program (NTP). A cancer re-assessment is currently underway at EPA.

Benzo(b)fluoranthene is considered by EPA to be a probable human carcinogen and by the IARC as possibly carcinogenic to humans (inadequate human evidence; sufficient evidence in animal studies). Benzo(e)pyrene is considered by EPA to be a probable human carcinogen (based on benzo(a)pyrene), by the IARC as carcinogenic to humans, and by the NTP as reasonably anticipated to be a carcinogen. 1-methylnaphthalene does not have a cancer class; however, EPA has derived a cancer potency factor for 1-methylnaphthalene based on carcinogenic evidence[16].

PCBs are considered by EPA to be a probable human carcinogen and by the IARC as carcinogenic to humans (sufficient evidence in humans). The NTP reasonably anticipates PCBs to be carcinogenic.

Inorganic arsenic is considered by EPA, the IARC, and the NTP as a known carcinogen with sufficient human evidence.

The estimated risk for cancer from exposure to contaminants is usually calculated by multiplying the exposure dose by a cancer potency factor; usually EPA's corresponding cancer slope factor in $(mg/kg/day)^{-1}$ for a carcinogen. This cancer slope factor (CSF) is equivalent to the 95% upper-bound lifetime cancer risk to an individual, rather than the average risk, suggesting that cancer risk is actually lower, perhaps by several orders of magnitude. EPA and the broader scientific community consider a cancer risk range of between one in a million to one in ten thousand (10^{-6} to 10^{-4}) as an acceptable range. That means that it is used by EPA for evaluation of human food-chain exposures because it provides assurance that risk is not underestimated. An increased cancer risk of one in a million or less is generally considered an insignificant increase in cancer risk. Because a cancer re-assessment for dioxin is currently underway at EPA, DPH used the California Environmental Protection Agency (CalEPA) oral slope factor for TCDD to estimate cancer risk from exposure to total dioxin TEQ.

Exposure to a cancer-causing chemical, even at low concentrations, is assumed to be associated with some increased risk of cancer for evaluation purposes. To estimate lifetime cancer risk from consuming fish caught in Burnett Creek, DPH used an exposure period of 40 years. Table 8 shows the estimated cancer risk for adults who grew up fishing in Burnett Creek most of their life.

[16]Provisional Peer Reviewed Toxicity Values (PPRTVs) derived by EPA's Superfund Health Risk Technical Support Center (STSC) for the EPA Superfund Program.

Table 8: Estimated Cancer Risks from Eating Fish Caught in Burnett Creek over a Lifetime of Exposure

Chemical Contaminant	Estimated Cancer Risk 1 Meal/Week	Estimated Cancer Risk 2 Meals/Week
Arsenic[a]	3.5×10^{-5}	7.1×10^{-5}
Benzo(b)fluoranthene[b]	7.0×10^{-8}	1.4×10^{-7}
Benzo(e)pyrene[b]	1.3×10^{-7}	2.5×10^{-7}
1-Methylnaphthalene[b]	4.3×10^{-9}	8.6×10^{-9}
PCBs[c]	4.3×10^{-5}	8.5×10^{-5}
Total Dioxin TEQ[d]	2.3×10^{-6}	4.7×10^{-6}

[a]Arsenic cancer risk based on the mean concentration of arsenic found in the four species sampled and assuming that 10% of the total arsenic found was inorganic arsenic. Cancer risk is based on 40 years or exposure.
[b]The mean PAH concentrations found in the four species sampled samples were used to estimate exposure doses and cancer risk based on 40 years of exposure. Benzo(b)fluoranthene and benzo(e)pyrene were not detected in one or more species. Non detect concentrations used in calculating the mean were zero.
[c] The mean PCB concentrations found in the four species sampled were used to estimate exposure doses and cancer risk based on 40 years of exposure. A cleaning reduction of 50% was used in the PCB exposure dose estimations.
[d]The mean Total Dioxin TEQ concentration found in the fish sampled was used to estimate exposure doses and cancer risks based on 40 years of exposure. A cleaning reduction of 50% was used in Total Dioxin TEQ exposure dose estimations.
Note: EPA cancer slope factors used in the cancer risk estimation were as follows: 1.5 $(mg/kg/day)^{-1}$ for arsenic, 1.2 $(mg/kg/day)^{-1}$ for benzo(b)fluoranthene, 7.3 $(mg/kg/day)^{-1}$ for benzo(a)pyrene, 0.029 $(mg/kg/day)^{-1}$ for 1-methylnaphthalene, and 2.0 $(mg/kg/day)^{-1}$ for PCBs. Individual PAH cancer risks were summed to give a total cancer risk from PAH exposure stated below. The California EPA (CalEPA) oral cancer slope factor 1.3×10^{5} $(mg/kg/day)^{-1}$ was used to estimate cancer risk for Total Dioxin TEQ.

For perspective, the lifetime risk in the U.S. that an individual will develop cancer from all causes is slightly less than 1 in 2 for men (50,000/100,000) and a little more than 1 in 3 for women (33,000/100,000) [24].

- The estimated lifetime cancer risk for adults exposed to arsenic and PCBs in fish harvested and consumed from Burnett Creek over a 40 year period is approximately 7 to 8 excess cancer cases that can be expected from this exposure in 100,000 people eating 2 meals/week and approximately 3 to 4 excess cancer cases that can be expected from this exposure in 100,000 people eating 1 meal/week.

- By summing all the individual PAH estimated cancer risks, the estimated lifetime cancer risk for PAHs found in fish captured from Burnett Creek ranges from approximately 4 excess cancer cases that can be expected from this exposure in 10,000,000 people eating 2 meals/week and approximately 2 excess cancer cases that can be expected from this exposure in 10,000,000 people eating 1 meal/week.

Therefore, DPH concludes that the estimated lifetime cancer risk for adults exposed to arsenic and PCBs in some fish harvested and consumed from Burnett Creek over a 40 year period is low.

Child Health Considerations

In communities faced with contamination of the water, soil, air, or food, ATSDR and DPH recognize that the unique vulnerabilities of infants and children demand special emphasis. Due to their immature and developing organs, infants and children are usually more susceptible to toxic substances than are adults. Children are more likely to be exposed because they play outdoors and they often bring food into contaminated areas. They are also more likely to encounter dust, soil, and contaminated vapors close to the ground. Children are generally smaller than adults, which results in higher doses of chemical exposure because of their lower body weights relative to adults. In addition, the developing body systems of children can sustain permanent damage if toxic exposures occur during critical growth stages.

From consuming fish captured in Burnett Creek, young children may have been exposed to all the contaminants found in the fish sampled. In addition, since dioxin, furans, and PCBs can cross the placenta and enter fetal tissues and can also concentrate in breast milk, infants and toddlers could be exposed to these chemicals *in utero* and during breast feeding. Very few studies have looked at how dioxins can affect children's health. Chloracne has been observed in children exposed to much higher than current background levels of 2,3,7,8-TCDD. The children appeared to be more sensitive (effects occurred at a lower body burden) than adults. With PCBs, a few studies have reported a correlation between umbilical cord blood levels and reduced birth rate and size, and behavioral effects in infants. Because the brain, nervous system, thyroid, and reproductive organs are still developing in the fetus and neonate, the effect of PCBs on these target systems may be more profound after exposure during the prenatal and neonatal periods [18]. There is some evidence that inhaled or ingested inorganic arsenic can injure pregnant women and/or their unborn babies, although the studies are not definitive. Studies in animals show that large doses of inorganic arsenic that cause illness in pregnant females can also cause low birth weight, fetal malformations, and even fetal death. Arsenic can cross the placenta and has been found in fetal tissues. Arsenic is also found at low levels in breast milk [20]. Therefore, pregnant women, women who may become pregnant, and young children should follow the DNR *2013 Guidelines for Eating Fish from Georgia Waters* and limit their consumption of fish caught in Burnett Creek.

Conclusions

DPH evaluated past, current, and future exposure to dioxins and furans, pesticides, metals, PCBs, and PAHs in fish samples from Burnett Creek. This evaluation included an estimation of exposure doses from oral ingestion of contaminants present in the fish sampled in October 2012. The conclusions presented below were based on a review and evaluation of the samples submitted for analysis. It is important to note that DPH's conclusions were based on a one-time sampling event. More sampling results from the same species (in addition to more species) captured for this health consultation over a longer period of time would more accurately describe

any temporal and life-stage fluctuations in the contaminant levels found in fish inhabiting Burnett Creek. Another important caveat is that the fish analyzed were whole-fish samples that would contain the maximum amount of contaminants that could be analyzed. Cleaned and filleted fish are likely to contain lower levels of the contaminants found.

1. DPH concludes that people (children and adults) eating one to two meals per week of fish harvested from Burnett Creek are not likely to be harmed by the very low levels of dioxins and furans found in the fish species analyzed.

2. DPH concludes that people (children and adults) eating one to two meals per week of fish harvested from Burnett Creek is not likely to be harmed by the very low levels of PAHs found in the fish species analyzed.

3. DPH concludes that people (children and adults) eating one to two meals per week of seatrout and whiting harvested from Burnett Creek are likely to be harmed from the levels of PCBs found in these fish. Children consuming 2 meals/week of black drum are also likely to be harmed from the levels of PCB exposure.

4. DPH concludes that people (children and adults) eating one to two meals per week of fish harvested from Burnett Creek are not likely to experience non-cancer health effects from the very low levels of arsenic found in the fish species analyzed.

5. DPH concludes that based on the likelihood of joint, dual interaction of methylmercury on PCBs and vice versa, people eating seatrout, whiting and spot harvested in Burnett Creek based on the fish consumption scenarios used in this health consultation, might be harmed by this consumption. People following the DNR fish consumption guidance by eating seatrout, whiting, and spot harvested in Burnett Creek only once a month would not likely be harmed.

6. DPH concludes that the estimated lifetime cancer risk for adults exposed to arsenic and PCBs in fish harvested and consumed from Burnett Creek over a 40 year period is low. For arsenic, approximately 7 excess cancers can be expected from this exposure in 100,000 people eating 2 meals per week. For PCBs, approximately 8 excess cancers can be expected from this exposure in 100,000 people eating 2 meals per week. And for dioxin and dioxin-like compounds, 5 excess cancers can be expected in 1,000,000 people eating two meals per week.

7. DPH concludes that the *2013 Guidelines for Eating Fish from Georgia Waters* and specifically adhering to the "Upper Turtle and Buffalo Rivers, Upriver of State Highway 303" recommendations will be protective of human health for people eating fish from Burnett Creek.

Recommendations

DPH recommends that the public follow the *2013 Guidelines for Eating Fish from Georgia Waters* published by the Georgia Department of Natural Resources (DNR); specifically, recommendations for the "Upper Turtle and Buffalo Rivers, Upriver of State Highway 303", which includes Burnett Creek. Recommendation are that no more than one meal of blue crab, sheepshead, red drum, black drum and striped mullet caught upriver of Highway 303 be consumed per week. DNR also recommends that no more than one meal of croaker, spot, spotted seatrout, and whiting be consumed per month. The National Shellfish Sanitation Program has issued a shellfish ban, which recommends no consumption of shellfish (clams, mussels, and oysters) collected from those areas. The Guidelines can be found at: www.gaepd.org/Files_PDF/gaenviron/GADNR_FishConsumptionGuidelines_Y2013.pdf.

The consumption recommendations published in the Guidelines are based on health-risk calculations for someone eating fish with known contamination over a period of 30 years or more. These Guidelines are not intended to discourage people from eating fish, but should be used as a guide for choosing which type (species) and size of fish to eat from Georgia waters. Additionally, these guidelines were designed to protect both children and adults from cancer and other potential toxic effects of these chemicals.

In addition, DPH recommends that EPA, EPD, and the GEC work collaboratively to post signage at strategic locations near Burnett Creek that reminds potential anglers not to consume more than one meal per month of croaker, spot, spotted seatrout, and whiting harvested from Burnett Creek.

Public Health Action Plan

1. DPH will distribute this health consultation and a fact sheet summarizing our findings to the public and work with the Glynn Environmental Coalition to ensure that health education reaches those residents fishing in Burnett Creek.

2. DPH will provide the Coastal Health District and Glynn Environmental Coalition with multiple copies of the *2013 Guidance for Eating Fish from Georgia Waters* for distribution to area residents and visitors.

3. As additional data become available, DPH will review the information and take appropriate actions.

4. DPH will continue to respond to all requests for information and health concerns regarding the safety of consuming fish from Burnett Creek.

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Report Preparation

This Health Consultation for fish captured from Burnett Creek near the Brunswick Wood Preserving NPL Site was prepared by the Georgia Department of Public Health under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with the approved agency methods, policies, procedures existing at the date of publication. ATSDR has reviewed this document and concurs with its findings based on the information presented. ATSDR's approval of this document has been captured in an electronic database, and the approving agency reviewers are listed below.

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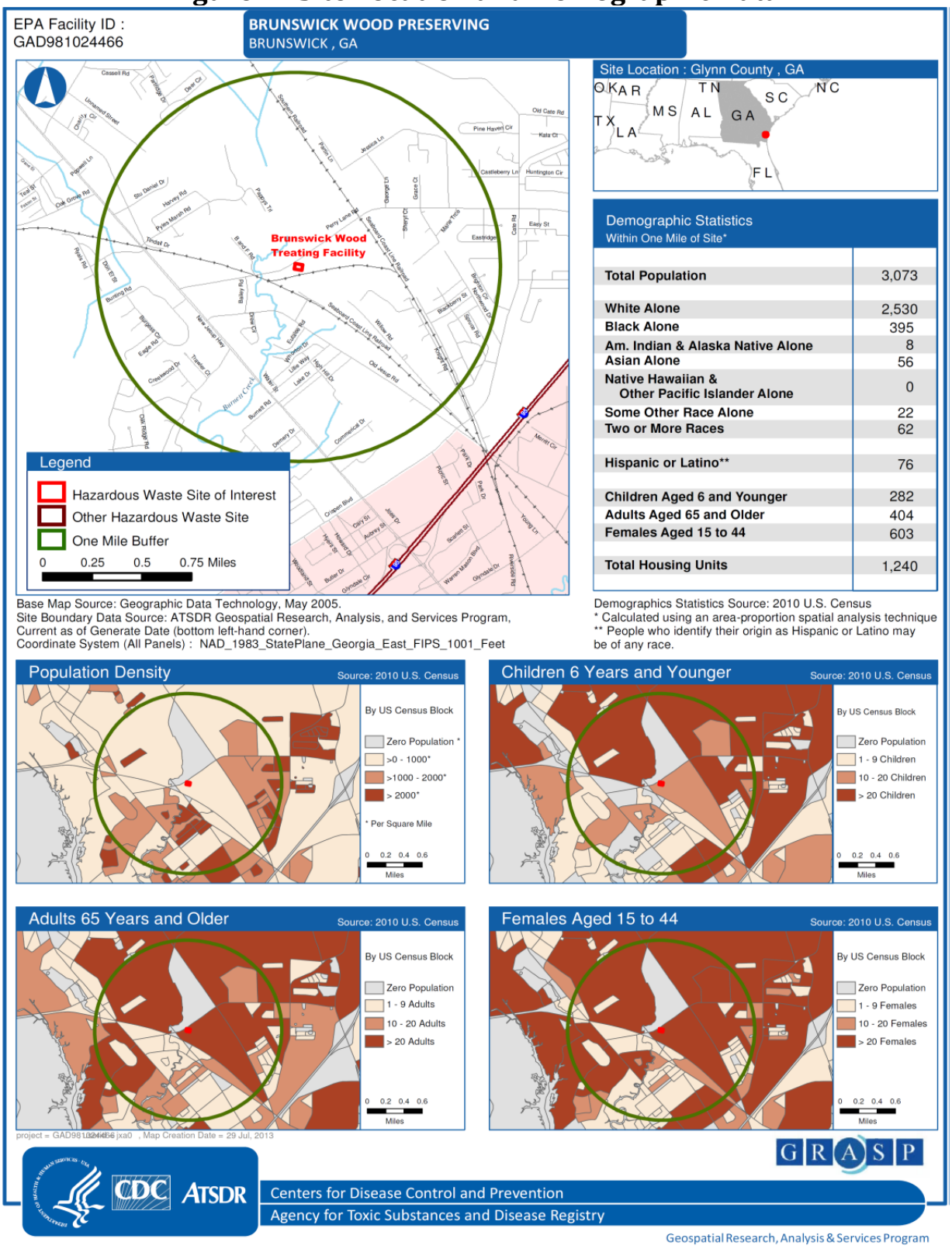
FIGURES

Figure 1: Aerial View of Burnett Creek (looking south of New Jesup Highway)

Figure 2: Site Location and Demographic Data



APPENDICES

Appendix A: Burnett Creek Water Quality Parameters

Burnett Creek Water Sampling Data October-November 2012

A fishing tournament to collect seafood samples from Burnett Creek was held on October20, 2012. A rainfall event that occurred the week of October 7, 2013 resulted in up to 6 inches of rain in a single event into the Burnett Creek watershed. Since the rainfall event would affect ecological conditions in Burnett Creek, water monitoring was conducted to document the salinity, dissolved oxygen levels, and temperature in the three statistical zones being used for seafood collection before, during, and after the sampling event.

Statistical Zones

Zone One – Cowpen Creek to the Rt. 341 Bridge
Zone Two – Rt. 341 Bridge to the Old Jesup Road Bridge
Zone Three – Old Jesup Road Bridge to the Perry Land Road Bridge

Locations

The water parameters sampling locations were based upon the statistical zones established for the seafood collection event.

Location	Latitude	Longitude
Perry Lane Road Bridge	31.21.381N	081.32.107W
Old Jesup Road Bridge	31.14.346N	081.32.036W
Rt. 341 (Redding Dock)	31.13.981N	081.32.154W
Blythe Island Boat Ramp (background)	31.11.418N	081.32.746W

Water Sampling Data (water data from the date of the seafood sampling is in **bold**)

Location	Date/Time	Depth Ft.	Dissolved Oxygen mg/L	Salinity (Parts per thousand)	Temperature °C	Comments
Perry Lane	10-9-12 15:57	2	4.70	0.1	20.4	High ebb Brown water
Old Jesup	10-9-12 16:06	2	4.97	0.1	20.3	High ebb Very brown water
Rt. 341	10-9-12 16:17	2	3.90	10.7	23.6	High ebb, very brown, scum on top of water
		1		3.6		
		3		12.6		
		5		15.4		
		9		15.8		
Perry Lane	10-12-12 09:55	2	5.00	0.1	19.1	Strong ebb, very brown water
Old Jesup	10-12-12 10:05	2	5.11	0.2	19.1	Strong ebb, very brown water
Rt. 341	10-12-12 10:18	2	3.64	7.3	21.5	Strong ebb, very brown water
		8		7.5		
Perry Lane	10-14-12 09:59	2	3.25	8.5	23.1	High first of ebb

Old Jesup	10-14-12 10:06	2	3.05	15.9	23.4	High first of ebb
Rt. 341	10-14-12 10:13	2	3.00	19.5	23.7	High first of ebb
	10:14	9	2.95	19.8	23.8	High first of ebb
Perry Lane	10-16-12 10:08	2	3.70	11.0	22.1	High flood tide
Old Jesup	10-16-13 10:14	2	3.41	17.2	22.9	High flood tide
Rt. 341	10-16-12 10:28	1	3.40	20.4	23.3	High Flood tide
		9	3.41	20.5	23.6	
Perry Lane	10-18-12 12:13	2	3.88	13.7	21.9	High tide flooding, brown water
Old Jesup	10-18-12 12:20	2	3.61	18.7	22.7	High tide flooding, brown water with scum
		4		19.0		
Rt. 341	10-18-12 12:35	2	3.65	21.3	23.2	High tide flooding, brown water, no scum
		9		21.3	23.2	
Rt. 341	**10-20-12 11:11**	**2**	**3.50**	**18.6**	**22.3**	**½ Flood tide**
		5	**3.78**	**18.8**	**22.3**	**½ Flood tide**
Perry Lane	**10-20-12 13:49**	**2**	**4.00**	**12.1**	**21.6**	**High tide slack**
Old Jesup	**10-20-12 13:56**	**2**	**4.05**	**18.6**	**22.9**	**High tide near slack**
Rt. 341	**10-20-12 14:27**	**2**	**4.19**	**21.7**	**24.00**	**High tide near slack**
		9		**21.8**	**23.8**	
Perry Lane	10-26-12 09:33	2	2.98	17.0	23.1	High tide, just starting ebb
Old Jesup	10-26-12 09:40	2	3.07	20.8	23.1	High tide, ebb, brown water
Rt. 341	10-26-12 09:50	2	3.62	22.6	23.1	High tide, ebb
Blythe Isl. (background)	11-8-12 14:56	2	6.80	27.7	18.0	High tide slack
Perry Lane	11-8-12 15:14	2	4.47	18.6	17.0	High tide, end of flood
Old Jesup	11-8-12 15:21	2	5.51	22.9	17.3	High tide, end of flood
Rt. 341	11-8-12 15:31	2	6.16	25.1	17.3	High tide, slack

Appendix B: Explanation of Evaluation Process

Step 1--The Screening Process

In order to evaluate the available data, DPH used comparison values (CVs) to determine which chemicals to examine more closely. CVs are contaminant concentrations found in a specific environmental media (air, soil, water, sediment, and food) and are used to select contaminants for further evaluation. CVs incorporate assumptions of daily exposure to the chemical and a standard amount of environmental media that someone may inhale or ingest each day. CVs are generated to be conservative and non-site specific. The CV is used as a screening level during the public health assessment (PHA) or health consultation process. CVs are not intended to be environmental clean-up levels or to indicate that health effects occur at concentrations that exceed these values.

CVs can be based on either carcinogenic (cancer-causing) or non-carcinogenic effects. Cancer-based CVs are calculated from the U.S. Environmental Protection Agency's (EPA) oral cancer slope factors for ingestion exposure, or inhalation risk units for inhalation exposure. Non-cancer CVs are calculated from ATSDR's minimal risk levels, EPA's reference doses, or EPA's reference concentrations for ingestion and inhalation exposure. When a cancer and non-cancer CV exist for the same chemical, the lower of these values is used as a conservative measure.

DPH also developed a more conservative dioxin screening value based on EPA's chronic oral reference dose (RfD) for 2,3,7,8-tetrachlorodibenzo –p-dioxin (the most toxic congener) and site-specific ingestion values based on community knowledge. The dioxin screening values were calculated as follows:

Adult Screening Value

$$SV_F = \frac{RfD \times BW}{IR}$$

where;

SV_F = dioxin contaminant screening value (mg/kg)
RfD = EPA chronic oral reference dose for 2,3,7,8-tetrachlorodibenzo-p-dioxin (7×10^{-10} mg/kg/day)
BW = adult body weight (80kg)
IR = ingestion rate (0.0324 kg/day for eating one meal per week, and 0.0648 kg/day for eating two meals per week). One meal is 226 grams (8 oz) divided by 7 days per week.

For example, the following screening value is based on an adult eating one meal per week of fish caught from Burnett Creek:

$$SV_F = \frac{7 \times 10^{-10} \text{mg/kg/day} \times 80 \text{ kg}}{0.0324 \text{ kg/day}}$$

$= 1.73 \times 10^{-6}$ mg/kg (or parts per million dioxin contamination in fish)

The following screening value is based on an adult eating two meals per week of fish caught from Burnett Creek:

$$SV_F = \frac{7 \times 10^{-10} \text{ mg/kg/day} \times 80 \text{ kg}}{0.0648 \text{ kg/day}}$$

$= 8.64 \times 10^{-7}$ mg/kg (or parts per million dioxin contamination in fish)

Child Screening Value

$$SV_F = \frac{RfD \times BW}{IR}$$

where;

SV_F = dioxin contaminant screening value (mg/kg)
RfD = EPA chronic oral reference dose for 2,3,7,8-tetrachlorodibenzo-p-dioxin (7×10^{-10} mg/kg/day)
BW = child body weight (18.6 kg)
IR = ingestion rate (0.0162 kg/day for eating one meal per week, and 0.0324 kg/day for eating two meals per week). One meal is 113 grams (4 oz) divided by 7 days per week.

For example, the following screening value is based on a child eating one meal per week of fish caught from Burnett Creek:

$$SV_F = \frac{7 \times 10^{-10} \text{ mg/kg/day} \times 18.6 \text{ kg}}{0.0162 \text{ kg/day}}$$

$= 8.0 \times 10^{-7}$ mg/kg (or parts per million dioxin contamination in fish)

The following screening value is based on a child eating two meals per week of fish caught from Burnett Creek:

$$SV_F = \frac{7 \times 10^{-10} \text{ mg/kg/day} \times 18.6 \text{ kg}}{0.0324 \text{ kg/day}}$$

$= 4.0 \times 10^{-7}$ mg/kg (or parts per million dioxin contamination in fish)

Step 2--Evaluation of Public Health Implications

The next step in the evaluation process is to take those contaminants that are above their respective CVs and further identify which chemicals and exposure situations are likely to be a health hazard. Separate child and adult exposure doses (or the amount of a contaminant that gets into a person's body) are calculated for site-specific scenarios, using assumptions regarding an individual's likelihood of exposure to contaminants in Burnett Creek fish. A brief explanation of the calculation of estimated exposure doses used in this health consultation is presented below.

Consumption of contaminants present in fish caught in Burnett Creek. Exposure doses for the consumption of contaminants present in fish were calculated using the measured concentration of PCBs and arsenic in milligrams per kilogram (mg/kg) of fish tissue. The following equation is used to estimate the exposure doses resulting from ingestion of contaminated fish:

$$ED_F = \frac{C \times IR \times EF \times CR \times CF}{BW}$$

where;

ED_F = exposure dose from eating fish (mg/kg/day)
C = contaminant concentration mean (mg/kg)
IR = ingestion rate of contaminated fish (based on 8 ounces per week (32,400 mg/day) or 16 ounces per week (64,800 mg/day) for an adult and 4 ounces per week (16,200 mg/day) or 8 ounces per week (32,400 mg/day) for a child).
EF = exposure factor (based on frequency of exposure, exposure duration, and time of exposure). The exposure factor used for the purpose of this analysis was one. This is the most conservative exposure factor assuming exposure is occurring 24 hours per day, 7 days per week.

CR = cleaning reduction of 50% (0.50) used for PCB exposure doses. This factor was not used to calculate exposure doses from arsenic.
CF = conversion factor (10^{-6} kg/mg)
BW = body weight (based on average body weight for an adult (80 kg); and the average body weight of a child age 3< 6 years old (18.6 kg)

For example, the following is an estimated exposure dose for an adult eating one meal per week of seatrout with a PCB concentration of 0.39 mg/kg:

$$ED_F = \frac{0.39 \text{ mg/kg} \times 32{,}400 \text{ mg/day} \times 1 \times 0{,}50 \times 10^{-6} \text{ kg/mg}}{80 \text{ kg}}$$

$$= 7.9 \times 10^{-5} \text{ mg/kg/day (or 0.00008 mg/kg/day) PCBs}$$

Of the total arsenic concentration, the amount of inorganic arsenic in fish is approximately 1.5 % and the amount of arsenic in shellfish is approximately 20%. As a conservative measure, DPH assumed that the inorganic arsenic concentration in fish would be approximately 10%, which over estimates the actual inorganic arsenic concentrations that are likely to be found in the fish sampled from Burnett Creek. In this example, the following is an estimated exposure dose for an adult eating one meal per week of black drum with an arsenic concentration of 1.65 mg/kg:

10% of 1.65 mg/kg = 0.165 mg/kg inorganic arsenic; therefore,

$$ED_F = \frac{0.165 \text{ mg/kg} \times 32{,}400 \text{ mg/day} \times 1 \times 10^{-6} \text{ kg/mg}}{80 \text{ kg}}$$

$$= 6.7 \times 10^{-5} \text{ mg/kg/day (or 0.0001 mg/kg/day) arsenic}$$

Non-cancer Health Risks

The doses calculated for exposure to individual chemicals are then compared to an established health guideline, such as an ATSDR minimal risk level (MRL[17]) or an EPA reference dose, in order to assess whether adverse health impacts from exposure are expected. Health guidelines are chemical-specific values that are based on available scientific literature and are considered protective of human health. Non-carcinogenic effects, unlike carcinogenic effects, are believed to have a threshold, that is, a dose below which adverse health effects will not occur. As a result, the current practice to derive health guidelines is to identify, usually from animal toxicology experiments, a no observed adverse effect level (NOAEL).This is the experimental exposure level in animals (and sometimes humans) at which no adverse toxic effect is observed. The values are summarized in ATSDR's *Toxicological Profiles (www.atsdr.cdc.gov/toxpro2.html).*The NOAEL is modified with an uncertainty (or safety) factor. The magnitude of the uncertainty factor considers various factors such as sensitive subpopulations (e.g., children, pregnant women, and the elderly), extrapolation from animals to humans, and the completeness of the available data. Thus, exposure doses at or below the established health guideline are not expected to cause adverse health effects because these guidelines are lower (and more human health protective) than doses that do not cause adverse health effects in laboratory animal studies.

For non-cancer health effects, MRLs were used in this PHA. A direct comparison of site-specific exposures and doses to study-derived exposures and doses found to cause adverse health effects is the basis for deciding whether health effects are likely to occur. If the estimated exposure dose to an individual is less than the MRL, the exposure is unlikely to result in non-cancer health effects. If the calculated exposure dose is greater than the MRL, the exposure dose is compared to known toxicological values for the particular chemical and is discussed in more detail in the text of the PHA.

[17] **Minimal Risk Levels (MRLs)** are developed by ATSDR for contaminants commonly found at hazardous waste sites. The MRL is developed for ingestion and inhalation exposure, and for lengths of exposures: acute (less than 14 days); intermediate (between 15-364 days), and chronic (365 days or greater). ATSDR has not developed MRLs for dermal exposure (absorption through skin).

It is important to consider that the methodology used to develop health guidelines does not provide any information on the presence, absence, or level of cancer risk. Therefore, a separate cancer risk evaluation is necessary for potentially cancer-causing contaminants detected at this site.

Cancer Risks

Exposure to a cancer-causing chemical, even at low concentrations, is assumed to be associated with some increased risk for evaluation purposes. The estimated risk for developing cancer from exposure to contaminants associated with the site was calculated by multiplying the site-specific doses by EPA's chemical-specific cancer slope factors (CSFs) available at *www.epa.gov/iris*. This calculation estimates an excess cancer risk expressed as a proportion of the population that may be affected by a carcinogen during a lifetime of exposure. For example, an estimated risk of 1×10^{-6} predicts the probability of one additional cancer over background in a population of 1 million. An increased lifetime cancer risk is not a specified estimate of expected cancers. Rather, it is an estimate of the increase in the probability that a person may develop cancer sometime in his or her lifetime following exposure to a particular contaminant under specific exposure scenarios. For children, the estimated excess cancer risk is not calculated for a lifetime of exposure, but from a fraction of lifetime; based on known or suspected length of exposure, or years of childhood.

Example Cancer Risk Calculation

Mean PCB Exposure Dose (0.000037 mg/kg/day) from eating one meal per week x CSF x years of exposure/70 years

Therefore,

Adult Cancer Risk = 0.000037 mg/kg/day x 2.0 $(mg/kg/day)^{-1}$ for PCBs x 40/70
= 4.3×10^{-5}

Appendix C: Burnett Creek Whole Fish Sampling Results

Table 1: Analytical Results for Dioxins and Furans Found in the Fish Sampled

Analyte	Black Drum Composite		Seatrout Composite		Whiting Composite		Red Drum Composite	
	Conc. (pg/g)	EMPC (pg/g)	Conc. (pg/g)	EMPC (pg/g)	Conc. (pg/g)	EMPC (pg/g)	Conc. (pg/g)	EMPC (pg/g)
Dioxins								
2378-TCDD	ND		ND		ND		ND	
12378-PeCDD	EMPC	0.0987 J	ND		ND		ND	
123478-HxCDD	ND		ND		ND		ND	
123678-HxCDD	0.5 J		0.112		0.623		EMPC	0.284
123789-HxCDD	ND		ND		ND		ND	
1234678-HpCDD	EMPC	0.194 J	EMPC	0.139	0.535		ND	
OCDD	0.423		0.332		EMPC	0.5	ND	
Furans								
2378-TCDF	0.276		EMPC	0.108	0.491		0.157	
12378-PeCDF	EMPC	0.17 J	EMPC	0.0868	EMPC		EMPC	0.167
23478-PeCDF	0.105 J		ND		ND		ND	
123478-HxCDF	ND		ND		ND		ND	
123678-HxCDF	ND		ND		ND		ND	
234678-HxCDF	ND		ND		0.139		ND	
123789-HxCDF	ND		ND		ND		ND	
1234678-HpCDF	ND		ND		EMPC	0.0881	ND	
1234789-HpCDF	ND		ND		ND		ND	
OCDF	ND		ND		ND		ND	
Totals								
Dioxins								
Total TCDD	ND	ND	ND	ND	ND	ND	ND	ND
Total PeCDD	ND	0.0987	ND	ND	ND	ND	ND	ND
Total HxCDD	0.5	0.721	0.112	0.112	0.751	0.932	0.168	0.452
Total HpCDD	ND	0.194	ND	0.139	0.535	0.535	ND	ND
Furans								
Total TCDF	0.276	0.276	ND	0.108	0.491	0.491	0.157	0.157
Total PeCDF	0.281	0.451	0.081	0.168	0.171	0.377	ND	0.167
Total HxCDF	0.249	0.249	ND	0.23	0.526	0.526	0.402	0.402
Total HpCDF	ND	ND	ND	ND	ND	0.0881	ND	ND
Total PCDD/Fs	**1.73**	**2.45**	**0.525**	**1.09**	**2.47**	**3.45**	**0.727**	**1.18**

pg/g: picogram per gram (10^{-12} grams of dioxins or furans per gram of whole fish)
ND: not detected at the limit indicated

NA: not applicable
J: Indicates that an analyte has a concentration below the reporting limit (lowest point of the calibration curve)
EMPC: Represents an Estimated Maximum Possible Concentration. EMPC's arise in cases where the signal/noise ratio is not sufficient for peak identification (the determined ion-abundance ratio is outside the allowed theoretical range), or where there is co-eluting interference.

Table 2: Analytical Results for Polycyclic Aromatic Hydrocarbons (PAHs) Found in the Fish Sampled

Contaminant	Black Drum Composite mg/kg	Seatrout Composite mg/kg	Whiting Composite mg/kg	Red Drum Composite mg/kg	EPA Comparison Value* mg/kg
1-Methylnaphthalene	0.00075	0.0006	0.0006	0.0006	95
2-Methylnaphthalene	0.003 J	0.00035 J	0.0003 J	0.0005	5.4
Acenaphthene	0.003	0.003	0.004	0.002	81
Anthracene	0.0003 J	0.0004 J	0.0003 J	0.00009 J	410
Benzo(b)fluoranthene	0.0003 J	0.0003 J	0.0004 J	ND	None
Benzo(e)pyrene	ND	0.0001 J	0.0002 J	ND	None
Dibenzofuran	0.0008	0.0007	0.0008	ND	None
Fluoranthene	0.0007	0.0009	0.0009	0.0002 J	54
Fluorene	0.002	0.001	0.001	0.0005 J	54
Naphthalene	0.0006	0.00045 J	0.0004 J	0.0005	27
Phenanthrene	0.001	0.001	0.001	0.0003 J	None
Pyrene	0.0003 J	0.0002 J	0.0002 J	ND	41

mg/kg = milligrams per kilogram
ND = not detected
J = estimated concentration
* Since fish CVs are not available from ATSDR, the EPA Regional Screening Levels for fish (November 2012) have been used in the screening process.
Note: Analytical detection limits ranged from 0.0000615 to 0.0000674 mg/kg for all PAHs except fluoranthene (0.000127 to 0.000139 mg/kg) and pyrene (0.000873 to 0.000957 mg/kg).

Table 3: Analytical Results of Pesticides and Polychlorinated Biphenyls (PCBs) in the Fish Sampled

Contaminant	Black Drum Composite mg/kg	Seatrout Composite mg/kg	Whiting Composite mg/kg	Red Drum Composite mg/kg	Analytical Detection Limit mg/kg	EPA Comparison Value* mg/kg
Aldrin	ND	ND	ND	ND	0.01	0.046
α-BHC	ND	ND	ND	ND	0.01	12
β-BHC	ND	ND	ND	ND	0.01	NA
γBHC	ND	ND	ND	ND	0.01	0.46
δ-BHC	ND	ND	ND	ND	0.01	NA
Chlordane	ND	ND	ND	ND	0.03	0.77
4,4,-DDD	ND	ND	ND	ND	0.01	0.77
4,4,-DDE	ND	ND	ND	ND	0.01	NA
4,4,-DDT	ND	ND	ND	ND	0.01	NA
Dieldrin	ND	ND	ND	ND	0.01	0.077
Endosulfan I	ND	ND	ND	ND	0.02	9.3
Endosulfan II	ND	ND	ND	ND	0.03	NA
Endosulfan Sulfate	ND	ND	ND	ND	0.05	NA
Endrin	ND	ND	ND	ND	0.01	0.46
Endrin Aldehyde	ND	ND	ND	ND	0.05	NA
Heptachlor	ND	ND	ND	ND	0.01	0.77
Heptachlor Epoxide	ND	ND	ND	ND	0.01	0.02
Toxaphene	ND	ND	ND	ND	0.10	NA
PCB-1016 (Aroclor)	ND	ND	ND	ND	0.03	0.11
PCB-1221 (Aroclor)	ND	ND	ND	ND	0.03	NA
PCB-1232 (Aroclor)	ND	ND	ND	ND	0.03	NA
PCB-1242 (Aroclor)	ND	ND	ND	ND	0.03	NA
PCB-1248 (Aroclor)	ND	ND	ND	ND	0.03	NA
PCB-1254 (Aroclor)	ND	ND	ND	ND	0.03	0.031
PCB-1260 (Aroclor)	ND	ND	ND	ND	0.03	NA
PCB-1268(Aroclor)	**0.113**	**0.39**	**0.20**	**0.035**	**0.03**	0.031
Methoxychlor	ND	ND	ND	ND	0.05	7.7

HCB	ND	ND	ND	ND	0.01	1.1
Mirex	ND	ND	ND	ND	0.10	0.27
Pentachloranisole	ND	ND	ND	ND	0.01	NA
Chlorpyrifos	ND	ND	ND	ND	0.05	1.4
Total Lipid	1.24%	1.0%	2.03%	0.64%	NA	

BOLD and highlighted values exceed EPA's comparison value
mg/kg: milligram per kilogram
ND: not detected at the limit indicated
NA: not available
* Since fish CVs are not available from ATSDR, the EPA Regional Screening Levels for fish (May 2014) have been used in the screening process. **Note:** The RSL used was for PCB-1254 because an RSL does not exist for the other PCBs analyzed.

Table 4: Analytical Results for Metals Found in the Fish Sampled

Contaminant	Black Drum Composite mg/kg	Seatrout Composite mg/kg	Whiting Composite mg/kg	Red Drum Composite mg/kg	Analytical Detection Limit mg/kg	EPA Comparison Value* mg/kg
Antimony	ND	ND	ND	ND	0.80	0.62
Arsenic	**1.65**	**0.68**	**0.77**	**0.95**	0.02	0.46†
Beryllium	ND	ND	ND	ND	0.1	3.1
Cadmium	ND	ND	ND	ND	0.1	1.5
Chromium, Total	0.215	ND	0.099	ND	0.1	NA
Copper	0.34	0.43	0.37	0.31	0.1	62
Lead	ND	ND	ND	ND	0.5	NA
Mercury	0.13	0.33	0.33	0.28	0.01	0.46
Nickel	0.35	0.44	0.89	0.28	0.1	31
Selenium	ND	ND	ND	ND	0.80	7.7
Silver	ND	ND	ND	ND	0.1	7.7
Thallium	ND	ND	ND	ND	0.8	0.015
Zinc	13.75	6.59	8.84	7.49	0.5	460

BOLD and highlighted values exceed EPA's comparison value
mg/kg = milligram per kilogram
ND = not detected at the limit indicated
NA = not available
* Since fish CVs are not available from ATSDR, the EPA Regional Screening Levels for fish (May 2014) have been used in the screening process
†EPA comparison value is for inorganic arsenic.
Note: The analytical detection limits for antimony and thallium were below the CVs. However, if the detection limits are used as a theoretical concentration, the estimated exposure doses for antimony and thallium from consuming one meal per week of any of the four species are below the respective EPA RfDs. In addition, ATSDR conducted health consultations and or public health assessments where sediment data from Burnett Creek was reviewed in 1998, 2000, and 2002, and both antimony and thallium were not detected.

Appendix D: Binary Weight-of-Evidence (BINWOE) Scheme for the Assessment of Chemical Interactions

	Classification	Factor
Direction of Interaction		**Direction**
= Additive		0
> Greater than additive		+1
< Less than additive		–1
? Indeterminate		0
Quality of the Data		**Weighting**
Mechanistic Understanding		
I.	Direct and Unambiguous Mechanistic Data: The mechanism(s) by which the interactions could occur has been well characterized and leads to an unambiguous interpretation of the direction of the interaction.	1.0
II.	Mechanistic Data on Related Compounds: The mechanism(s) by which the interactions could occur is not been well characterized for the chemicals of concern but structure-activity relationships, either quantitative or informal, can be used to infer the likely mechanisms(s) and the direction of the interaction.	0.71
III.	Inadequate or Ambiguous Mechanistic Data: The mechanism(s) by which the interactions could occur has not been well characterized or information on the mechanism(s) does not clearly indicate the direction that the interaction will have.	0.32
Toxicological Significance		
A.	The toxicological significance of the interaction has been directly demonstrated.	1.0
B.	The toxicological significance of the interaction can be inferred or has been demonstrated for related chemicals.	0.71
C.	The toxicological significance of the interaction is unclear.	0.32
Modifiers		
1.	Anticipated exposure duration and sequence.	1.0
2.	Different exposure duration or sequence.	0.79
a.	*In vivo* data	1.0
b.	*In vitro* data	0.79
i.	Anticipated route of exposure	1.0
ii.	Different route of exposure	0.79

Weighting Factor = Product of Weighting Scores: Maximum = 1.0, Minimum = 0.05
BINWOE = Direction Factor x Weighting Factor: Ranges from -1 through 0 to +1

Source: ATSDR. *Interaction Profile for: Persistent Chemicals Found in Fish (Chlorinated dibenzo-p- dioxins, Hexachlorobenzene, p,p'-DDE, Methylmercury, and Polychlorinated biphenyls).* May 2004.

ATSDR reviewed available data on the joint toxic action of mixtures of methylmercury, dioxin, and PCBs, and the weights of evidence were assessed regarding the mode of joint toxic action of pairs of these components [25]. In this analysis, 2,3,7,8-TCDD (TCDD) was taken as representative of CDDs in accordance with the TEF approach to assessing hazards from mixtures of CDDs. PCB mixtures were assessed as an entity in accordance with PCB MRLs which are derived for exposure to complex mixtures of PCBs.

Effects of Methylmercury on TCDD Toxicity

A direction of interaction cannot be reliably projected because of the absence of pertinent joint toxic action data, absence of information that possible pharmacokinetic interactions with methylmercury may influence TCDD toxicity, and inadequate mechanistic understanding supporting a reliable projection of the mode of possible joint action TCDD and methylmercury on other toxicity targets.

Chlorinated dibenzo-*p*-dioxins (CDDs) are postulated to produce several types of effects by binding to the intracellular Ah receptor (aryl hydrocarbon receptor) and subsequent molecular events within target organs. The Ah receptor is a protein in humans involved in the regulation of biological responses to aromatic hydrocarbons. This receptor has been shown to regulate xenobiotic[18]-metabolizing enzymes such as cytochrome P450. Cytochrome p450 enzymes transform hydrophobic molecules into water-soluble molecules that can be excreted from the body. Xenobiotic substances that bind to the Ah receptor include members of the halogenated aromatic hydrocarbon family (polychlorinated dibenzodioxins, dibenzofurans and some biphenyls) and polycyclic aromatic hydrocarbons (3-methylchoanthrene, benzo(a)pyrene, benzanthracenes and benzoflavones) [25]. Natural plant flavonoids, polyphenolics and indoles also bind to the Ah receptor. Once a xenobiotic is bound intracellularly, the bound Ah receptor in translocated from the cytoplasm into the nucleus of the cell where it combines with the Ah receptor nuclear translocator before binding to DNA. The end result is a variety of differential changes gene expression. It is an adaptive response that includes the induction of a variety of metabolizing enzymes, including the induction of a family of cytochrome P450 enzymes used in the metabolism of halogenated aromatic hydrocarbons and polycyclic aromatic hydrocarbons. Presumably, vertebrates have this function to be able to detect a wide range of chemicals, indicated by a wide range of substrates the Ah receptor is able to bind and facilitate their biotransformation and elimination [22, 26]. The Ah receptor may also signal the presence of toxic chemicals in food and cause adverse reaction to such foods [27]. Consequences of this adoptive response can be toxic responses elicited by Ah receptor activation. Toxicity results from two different ways of Ah receptor signaling. The first is a side effect of the adaptive response in which the induction of metabolizing enzymes results in the production of more highly toxic metabolites. For example, benzo(a)pyrene induces its own metabolism and bioactivation to a more highly toxic metabolite via the induction of two cytochrome P450 genes in several tissues [28]. The second approach to toxicity is the result of aberrant changes in global gene

[18] Xenobiotic-recognized as a foreign in the body.

transcription beyond those observed in the "Ah receptor gene battery." These global changes in gene expression lead to adverse changes in cellular processes and function [29].

Methylmercury toxicity is not expected to involve the Ah receptor, but whether methylmercury may interact with TCDD at other cellular or molecular sites involved in the development of TCDD health effects is unknown. Therefore, the effect of methyl mercury on TCDD has been given a BINWOE =? (0) for indeterministic evidence for additive joint action.

Effects of TCDD on Methylmercury

There is *in vitro* evidence that a synthetic mixture of CDDs, CDFs, and PCBs at concentrations that were reflective of concentrations in fish from the St. Lawrence River (that flows through Canada connecting the Atlantic Ocean to the Great Lakes) did not change the effects of methylmercury on rat lymphocyte viability and mitogenic (cell division) ability [21]. The additive direction of interaction is selected to reflect a projected lack of effect of CDDs on methylmercury immunotoxicity. For other methylmercury effects, a direction of interaction cannot be reliably projected due to the absence of pertinent joint toxic action data, absence of information that possible pharmacokinetic interactions with TCDD may influence methylmercury toxicity, and inadequate mechanistic understanding supporting a reliable projection of the mode of possible joint toxic action of TCDD and methylmercury on other toxicity targets.

CDDs are postulated to produce immunotoxic effects such as lymphoid tissue depletion and increased susceptibility to infectious agents via an initial mediation by the Ah receptor and unknown subsequent molecular events within the immune system [21]. Mercuric salts and methylmercury have been demonstrated to cause both autoimmune stimulation and a suppression of the immune system, but the mechanisms that may be involved are unknown. Pertinent molecular sites of possible interactions between TCDD and methylmercury are thus unidentified, and the limited mechanistic understanding suggests that CDDs may produce immune effects by different mechanisms than methylmercury (i.e., methylmercury immunotoxicity is not expected to involve Ah receptor mediation). Therefore, the highest uncertainty category (III) was therefore selected for mechanistic understanding.

In vitro studies of immunological endpoints in rat cultured lymphocytes found no evidence for interactions between methylmercury and a synthetic mixture of CDDs, CDFs, and PCBs at low concentrations reflective of concentrations in St. Lawrence River fish, but study design limitations preclude definitive conclusions regarding the mode of possible joint toxic actions on the immune system [22]. No other studies (*in vitro* or *in vivo*) to support or refute the results of this single study were located. A moderate confidence rating for toxicological significance (B) is selected to reflect the lack of supporting data, design limitations of the single available study, and the plausibility that the observed lack of effect of CDDs, CDFS, and PCBs on methylmercury immunotoxicity is relevant to pertinent environmental exposure levels such as fish consumption [22]. Therefore, the effect of TCDD on methylmercury has been given a BINWOE = IIIBbii (0) for a plausible additive effect on immune system suppression. See Appendix D for explanation of BINWOE codes.

Effect of PCBs on TCDD

PCBs antagonized TCDD-induced immunosuppression and developmental toxicity in mice. Intermediate-duration dietary exposure of rats to binary mixtures of TCDD plus each of three PCB congeners produced no synergism on changes in body and organ weights and levels of retinoids in liver indicating that PCB mixtures may additively act with TCDD on these endpoints, but one congener (and not the other two) synergistically acted with TCDD to increase hepatic porphyrin levels and deplete serum T4 levels [22].

Oral exposures to PCBs or CDDs such as TCDD are associated with wide arrays of health effects that show considerable overlap. Although some PCB congeners (dioxin-like congeners) have been demonstrated to produce some effects via a common initial mechanistic step with TCDD and other CDDs (binding to the Ah receptor), mechanistic understanding of ensuing processes is too incomplete to provide reliable projections of net physiological responses to joint exposure of PCB mixtures and TCDD [22]. In addition, there is evidence that other PCB congeners produce adverse effects via mechanisms that are independent of Ah receptor mediation, and some PCB congeners counteract effects of other PCB congeners and TCDD. Therefore, the highest uncertainty category (III) was therefore selected for mechanistic understanding.

PCB mixtures antagonized TCDD-induced immunosuppression (intraperitoneal exposure) and cleft palate formation (oral exposure) in mice [25]. There is evidence that individual PCB congeners vary in how they interact with TCDD in affecting these endpoints; some antagonize, some do not, and one was shown to potentiate TCDD-induced cleft palate formation. To reflect uncertainty that the observed antagonisms may occur with environmental PCB mixtures of varying composition and that antagonism will occur on other immune endpoints, a moderate data quality factor (B) was assigned to the BINWOE for immune effects (several PCB mixtures were demonstrated to antagonize TCDD inhibition of cell-mediated immune response, but one [Aroclor 1232] did not), whereas a low data quality factor (C) was assigned for developmental toxicity (the only PCB mixture examined for joint action with TCDD was Aroclor 1254) [22].

The BINWOEs were derived to assess how environmental PCB mixtures may influence TCDD toxicity. PCB mixtures are the entity of concern, because humans are exposed to complex PCB mixtures and PCB MRLs are based on data for PCB mixtures. However, there is a large degree of uncertainty in the BINWOEs, given evidence that the composition of environmental PCB mixtures can vary substantially. Evidence that PCB congeners can vary in potency, mechanisms of action, and how they interact with TCDD, and the limited number of studies that have examined how mixtures of PCBs jointly act with TCDD in influencing the wide array of shared toxicity targets exacerbates these uncertainties.

From the available evidence on the effect of PCBs on TCDD, ATSDR has assigned the following slightly less-than-additive joint action BINWOE for:

- Suppression of TCDD-induced cell-mediated immune response from acute, oral exposures; where the assigned BINWOE = <IIIB1aii (-0.20).
- Suppression of TCDD-induced cell-mediated immune response from intermediate, intraperitoneal exposures; where the assigned BINWOE=<IIIB2aii (-0.16).

- TCDD-induced developmental toxicity (cleft palate, hydronephrosis in offspring) from acute, intraperitoneal exposures; where the assigned BINWOE=<IIIC1ai (-0.10).
- TCDD-induced developmental toxicity (cleft palate, hydronephrosis in offspring) from intermediate, oral exposures; where the assigned BINWOE=<IIIC1ai (-0.10).

Effect of TCDD on PCBs

Intermediate-duration dietary exposure of rats to binary mixtures of TCDD plus each of three PCB congeners produced no synergism on changes in body and organ weights and levels of retinoids in liver indicating that PCB mixtures may additively act with TCDD on these endpoints, but one congener (and not the other two) synergistically acted with TCDD to increase hepatic porphyrin levels and deplete serum T4 levels. Available studies of joint action of PCB mixtures and TCDD on immune suppression and developmental toxicity do not discern how TCDD may influence PCB effects on these endpoints. Available data are inconclusive regarding joint action of PCB mixtures and TCDD in adversely affecting female reproductive organ development and promoting tumors [22].

Oral exposures to PCBs or CDDs such as TCDD are associated with wide arrays of health effects that show considerable overlap. Although some PCB congeners (dioxin-like congeners) have been demonstrated to produce some effects via a common initial mechanistic step with TCDD and other CDDs (binding to the Ah receptor), mechanistic understanding of ensuing processes is too incomplete to provide reliable projections of net physiological responses to joint exposure of PCB mixtures and TCDD [21]. In addition, there is evidence that other PCB congeners produce adverse effects via mechanisms that are independent of Ah receptor mediation, and some PCB congeners counteract effects of other PCB congeners and TCDD. Therefore, the highest uncertainty category (III) was therefore selected for mechanistic understanding.

A 13-week dietary exposure rat study of binary joint action of TCDD with each of three PCB congeners (expected to have various mechanisms of action) found no evidence of synergism on body or organ weight changes or vitamin A depletion in the liver [22]. Evidence for less-than-additive joint action on these endpoints (with each of the three PCB:TCDD binary mixtures examined) was found, but this may have been due to near-maximal effects occurring at the dose levels used. However, evidence was found for synergistic effects between one of the congeners (2,2',4,4',5,5'-hexachlorobiphenyl [a dioxin-like PCB congener], but not the others) and TCDD on depletion of serum T4 levels and increased accumulation of porphyrins in liver [21]. Because of this variability between PCB congeners and the lack of data examining joint action of PCB mixtures and TCDD on T4 depletion and porphyria, the direction of interaction for the BINWOE was judged to be indeterminate (?) [22].

PCB mixtures antagonized TCDD-induced immunosuppression (intraperitoneal exposure) and cleft palate formation (oral exposure) in mice [22]. At the dose levels used in these studies, toxicity of the potent TCDD masked any adverse effects the PCB mixtures may have had on these endpoints. Thus, the influence that TCDD may have on PCB effects on these endpoints is indeterminate from the available data.

The BINWOEs were derived to assess how TCDD may influence PCB toxicity of environmental PCB mixtures. However, there is a large degree of uncertainty in the BINWOEs, given evidence that the composition of environmental PCB mixtures can vary substantially. Evidence that PCB congeners can vary in potency, mechanisms of action, and how they interact with TCDD, and the limited number of studies that have examined how mixtures of PCBs jointly act with TCDD in influencing the wide array of shared toxicity targets exacerbates these uncertainties.

From the available evidence on the effect of TCDD on PCB toxicity, ATSDR has assigned the following BINWOE codes (Appendix D) for evidence that additive joint action will occur and/or for indeterministic evidence that additive joint action will occur:

- Body and thymus weight changes, hepatomegaly, and decreased hepatic retinoids from intermediate, oral exposure; where the assigned BINWOE=IIIC (0).
- Thyroid hormone disruption, porphyria, and immune suppression; from intermediate, oral exposure; where the assigned BINWOE=? (0) for indeterministic evidence for additive joint action.
- Immune suppression from intermediate, intraperitoneal exposure; where the assigned BINWOE=? (0) for indeterministic evidence for additive joint action.
- Developmental toxicity (cleft palate formation) from intermediate, oral exposure; where the assigned BINWOE=? (0) for indeterministic evidence for additive joint action.

Effect of PCBs on Methylmercury and the Effect of Methylmercury on PCBs

There is *in vitro* evidence from one study that PCBs and methylmercury may synergistically decrease dopamine levels in rat brain cells presumably via disruption of calcium homeostatic mechanisms [22], but obvious synergism or additive joint action in affecting neurobehavioral endpoints was not demonstrated in a mouse *in vivo* study [22]. A greater-than-additive joint action on neurological function or development is projected with a moderate degree of uncertainty. Additive joint action to produce hepatic porphyria is supported by evidence from a study of quails exposed to Aroclor 1260 and methylmercury in the diet [22].

Changes in neurological function or development from PCBs and methylmercury have been proposed to at least partly involve disruption of calcium homeostatic mechanisms in neural cells leading to changes in neurotransmitter release (e.g., dopamine) or cell damage. Combined *in vitro* exposure of rat striatal tissue to a methylmercury and a 1:1 mixture of Aroclor 1254/1260 appeared to synergistically deplete tissue levels of dopamine [22]. These data suggest a possible synergism between PCB mixtures and methylmercury in affecting neurological dysfunction and development. A moderate uncertainty rating (II; i.e., medium confidence rating) was selected to reflect several areas of uncertainty: (1) mechanistic linkages between changes in dopamine release and the development of PCB- or methylmercury-induced changes in neural function and development are poorly understood; (2) obvious synergism was not observed on *in vivo* endpoints of neurological function in mice exposed to mixtures of methylmercury and PCBs [22], and (3) the *in vitro* exposure of rat striatal tissue to a methylmercury and a 1:1 mixture of Aroclor 1254/1260 study had some study design and reporting limitations that prevented a formal statistical characterization of the mode of joint action on dopamine release.

Intermediate-duration exposures of quail to methylmercury or Aroclor 1260 in the diet led to accumulation of porphyrins in liver; hepatic porphyrin levels in quail exposed to both agents simultaneously were similar to levels predicted based on additivity of response [22]. To reflect uncertainty in extrapolating from quails to mammals and the lack of corroborative data, a moderate data quality factor (B) was selected for toxicological significance of the projection of additive joint action to produce hepatic porphyria.

From the available evidence on the effect of PCBs on methylmercury toxicity, or vice versa, ATSDR has assigned the following BINWOE codes (Appendix D) for evidence that additive joint action will occur:

- Impaired neurological function or development from *in vitro* evidence; where the assigned BINWOE=<IICb (+0.20).
- Hepatic porphyria from intermediate, oral exposure; where the assigned BINWOE=IIIB (0).