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DRAFT REVIEW ON

Pacific Northwest LNG Summary of the Environmental Impact Statement and Environmental Assessment of Certificate Application with specific reference to background literature on chemicals of potential concern and potential effects on marine life and human health.

Prepared for:

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EXECUTIVE SUMMARY

On behalf of United Fisheries and Allied Worker's Union-CAW (UFAWU-CAW-CAW), Biowest Environmental Research Consultants (BIOWEST), has conducted a literature review to evaluate the potential effects of chemicals of potential concern (COPCs) on marine life and human health as identified in the Pacific Northwest LNG Summary of the Environmental Impact Statement and Environmental Assessment of Certificate Application. The review was conducted to inform the UFAWU-CAW-CAW of the potential impacts to marine life and human health that may occur following dredging and disposal of contaminated marine sediment resulting from the project to construct and operate a liquefied natural gas (LNG) facility on Lelu Island in the Prince Rupert area of BC.

The Environmental Impact Statement and Environmental Assessment of Certificate Application by Pacific Northwest LNG Limited Partnership Ltd (PNW LNG) specifically addresses the dredging and disposal of approx. 8 million m³ of material from two sites: the proposed site of the Materials Offloading Facility (MOF) in Porpoise Channel to the north of Lelu Island, and the proposed marine berth dredge area located approximately 2 km southwest of Lelu Island. The probable loading site is in Brown Passage.

Sediment samples collected from the dredge sites contained metal concentrations (arsenic [As] and copper [Cu]), which exceed the Interim Sediment Quality Guidelines (ISQGs), and were below Probable Effects Levels (PELs). The concentrations of these metals indicate that they are natural background levels (and were not considered COPCs). However, further sampling needs to be performed to firmly establish this. Identified COPCs included polycyclic aromatic hydrocarbons (PAH), polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). Concentrations of PCDD/Fs from the dredge site at depths of 0-0.5 m exceeded ISQGs (0.85 pg TEQ/g for PCDD/F), but were lower than the PEL (21.5 pg TEQ/g PCDD/F). The resuspension of contaminated sediment via dredging will occur, increasing the bioavailability of these chemicals to marine organisms. This will likely increase marine organism exposures, their potential accumulation and potential food web transfer (PCDD/Fs bioconcentrate and biomagnify). Sediments with measured chemical concentrations between the national ISQG and the PEL (PAH and PCDD/Fs in this regard) are considered to represent potential hazards to exposed organisms. Effects of PCDD/Fs on marine ecological receptors (invertebrates, fish, birds and mammals) include immunological, developmental, reproductive, and cardiotoxic effects. Effects of PAH include immunological, developmental, reproductive, and behavioural effects mainly to benthic organisms due to the low biomagnification potential of this group of chemicals. The propensity of PCDD/Fs to biomagnify in the food web is cause for concern for humans consuming contaminated marine organisms from this area, particularly those organisms from higher trophic levels. The potential effects of PCDD/Fs in humans include biochemical alterations, oxidative stress, endocrine disruption, reproductive and developmental effects, chloracne and cancer. The sediment concentrations of PAH and PCDD/Fs exceeding ISQGs, the potential for PCDD/F biomagnification, and the myriad of toxic effects in both ecological and human receptors suggest that there is potential hazard associated with these proposed dredging activities. Of particular concern are sensitive habitats in this area such as Flora Bank that is critical juvenile salmonid habitat which will be affected by dredging. In order to mitigate the potential effects of contaminated sediments on wildlife and in humans, alternatives to the application should be explored.

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1.0 INTRODUCTION

On behalf of United Fisheries and Allied Worker's Union-CAW (UFAWU-CAW), Biowest Environmental Research Consultants (BIOWEST), has conducted a literature review to evaluate the potential effects of chemicals of potential concern (COPCs) to both marine life and human health related dredging activities proposed by Pacific Northwest LNG in preparation of constructing and operating a liquefied natural gas (LNG) facility on Lelu Island, BC. At full build-out, the facility will receive approximately 3.2 billion standard cubic feet per day of pipeline grade natural gas, and produce up to 19.2 million tonnes per annum of LNG. The key components of the proposed Project include a natural gas reception system, gas pretreatment, three 6.4 million tonnes per annum natural gas liquefaction trains, three full containment 180,000 m³ LNG storage tanks, a marine terminal and berths with a trestle, trestle control room, two LNG carrier berths, shipping LNG (between the terminal and Triple Island pilotage station), a materials off-loading facility, pioneer dock, bridge, and pipeline. The proposed Project will be located on Lelu Island in northwest British Columbia, Canada. Lelu Island and surrounding waters are federal lands and waters within the boundaries of the PRPA, 15 km southwest of the City of Prince Rupert, BC. The review has been conducted to inform the UFAWU-CAW of the potential impacts of COPCs to marine life (directly) or human health (through consumption of contaminated seafood) resulting from two marine components of the project which include construction of: 1) the materials off-loading facility (MOF) and the approaches to the facility (vessel turning basin for safe navigation), located on Porpoise Channel, and 2) the marine terminal, including the berths, trestle, trestle control room, berths, cryogenic piping, and loading arms required to load LNG, located on Agnew Bank and Flora Bank.

Existing data and information were obtained from a number of sources including the primary literature, electronic resources (e.g., websites), and publicly available reports. The review was guided by 6 topic areas posed in the April 4, 2014 *Proposed Statement of Work (SOW)* from Ms. Luanne Roth:

- Topic #1. Information identifying the main chemicals of potential concern (COPCs) in dredging material as determined from the Environmental Impact Statement and Environmental Assessment of Certificate Application of PNW LNG.
- Topic #2. Information on the chemical and physical properties of COPCs identified.
- Topic #3. Identification of potential exposure pathways for COPCs from dredge materials to marine organisms and humans, including background information on bioaccumulation and biomagnification processes.
- Topic #4. Information on the potential toxic effects of COPCs to marine life.
- Topic #5. Information on the potential toxic effects of COPCs to humans.

2.0 SUMMARY OF DREDGING AND ASSESSMENT ACTIVITIES

Pacific NorthWest LNG Limited Partnership (PNW LNG) is proposing to construct and operate a liquefied natural gas (LNG) facility within the District of Port Edward, British Columbia. The marine components of the Project important to this review include: 1) a materials off-loading facility (MOF) and the approaches to the facility located on Porpoise Channel, and 2) marine terminals, including the berths, trestle, trestle control room, berths, cryogenic piping, and loading arms required to load LNG, located on Agnew Bank and Flora Bank.

There are two main dredging sites that will operate during the construction phase of this project. The first location is the proposed site of the MOF in Porpoise Channel to the north of Lelu Island. The second location is the proposed marine berth dredge area located approximately 2 km southwest of Lelu Island. Dredging at the MOF will include the removal of approximately

690,000 m³ of dredge material to a depth of 12.5 m below chart datum. The marine berth dredge area will include the removal of approximately 7 million m³ of dredge material to a depth of 15.6 m below chart datum.

Changes in sediment or water quality that could lead to toxicological concerns were assessed. Canada's Fisheries Act, 1985, and SARA, 2002, administered by Fisheries and Oceans Canada (DFO), are the primary laws providing protection for fish and fish habitat and marine mammals in the project boundaries. The CEPA, 1999, administered by Environment Canada, regulates the disposal of dredged material at sea. This regulation and the Canadian Council of Ministers of the Environment (CCME) sediment and water quality guidelines (WQG) for protection of marine life were used to assess potential effects of contaminants in sediment and water. Changes in sediment or water quality was assessed by comparing baseline project-related chemical concentrations to CCME and BC water and sediment quality guidelines for the protection of marine life and to Environment Canada screening criteria for disposal of sediment at sea.

Physical and chemical characteristics of intertidal and subtidal sediment and water quality were identified through field studies to assess the potential for release of contaminants during dredging at the MOF and disposal of the sediment. Marine sediment samples were collected around Lelu Island, but focused on the MOF dredge area only. This was explained in the PNW LNG report because the MOF is closer to Porpoise Harbour (4 km) than the marine berth dredge area (7 km). Porpoise Harbour is a historical disposal at sea site, which was the receiving environment for wastes generated by past industrial activities including the disposal of locally dredged materials (e.g., mud, silt and wood) and effluent from the kraft pulp and paper mill. In addition, the marine berth dredge area is situated in the open ocean on the southwest side of Lelu Island, which is more exposed. The field program was developed through consultation with Environment Canada.

Sediment was sampled from the proposed dredge area within the MOF and turning basin in May, July, and October 2013 at a variety of depths at 36 locations. Parameters of interest were polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), metals, dioxins and furans, particle size and total organic carbon.

BC Ministry of Environment guidance was used to assess contaminated sediments under the BC Contaminated Sites Regulation. For cadmium, lead, mercury, PCBs and PAHs, sediment quality was assessed in relation to the Disposal at Sea National Action List and the Canadian Council of Ministers of Environment (CCME) 2001 guidelines for the protection of aquatic life. These include Interim Sediment Quality Guidelines (ISQGs) and Probable Effects Levels (PELs). All other metals and polychlorinated dibenzo-dioxins (PCDDs) and polychlorinated dibenzo-furans (PCDFs) were assessed on the CCME ISQG and PEL.

Sediment samples were collected to establish a horizontal and vertical (area and depth) profile of chemicals contained in the sediment at the proposed MOF. Chemicals of interest included metals, polycyclic aromatic hydrocarbons (PAH), polychlorinated biphenyls (PCB), and polychlorinated dibenzo-p-dioxins and furans (PCDD/F) from historical human activities and presumed naturally occurring events. Chemical concentrations were compared to CCME SQGs for the protection of aquatic life and Canadian disposal at sea guidelines to address the potential to dispose dredged materials at the Brown Passage disposal site.

For the assessment of contamination at the marine berth dredge area, only 5 surface and 1.0 meter core sediments were collected to the southwest of Lelu Island, within 5 km of the marine berth dredge area. Sediments present at the disposal site in Brown Passage were screened for

contaminants by Environment Canada in April and October 2011.

3.0 CHEMICALS OF POTENTIAL CONCERN (COPCS)

Contaminant concentrations in sediment and water in the proposed project area have been affected by historical and current industrial activities such as a pulp mill which is no longer in operation, terminals and port facilities, fish processing facilities, a log dump, and releases of sanitary waste and storm water from developed areas. There are 3 areas of consideration with respect to present (baseline) and future sediment contamination: 1) the MOF dredge area, 2) the marine berth dredge area, and 3) the proposed loading site at Brown Passage.

Information on existing contaminant levels is from samples taken in the MOF dredge area which include a total of 82 sediment samples that were collected at 5 different spatial depth profiles within the MOF. The sample depth and number of samples include:

6 Intertidal surface grab samples (top 7.5 cm); 8 subtidal surface grab samples (top 7.5 cm); 13 surface core samples (0 – 1.5 m); 29 mid-core samples (1.5 – 5.5 m); and 26 deep core samples (5.5 – 12.0 m).

For metals, arsenic (As) concentrations ranged from 1.74 to 12.8 mg/kg with an average concentration of 7.47 mg/kg. Concentrations of As were higher than the ISQG (7.24 mg/kg) in 45 of 82 samples, and below the PEL of 41.6 mg/kg. Arsenic concentrations that exceeded the ISQG occurred at all depth profiles from 0 to 12.0 m. The proponents suggest that As is naturally occurring due to the depth and consistency of As concentrations measured in samples.

Copper (Cu) concentrations ranged from 11.0 to 40.7 mg/kg with an average concentration of 23.9 mg/kg. Concentrations of Cu were higher than the ISQG (18.7 mg/kg) in 56 of 82 samples, and below the PEL of 108.0 mg/kg. Cu concentrations that exceeded the ISQG occurred at all depth profiles from 0 to 12.0 m, therefore the project proponents suggest that these Cu concentrations are naturally occurring.

For PAH concentrations, 78 of 82 sediment samples were below the laboratory detection limit. PAHs were only detected in 3 surface sediments to a maximum depth of 1.5 m. All core samples deeper than 1.5 m showed PAH concentrations below the detection limit. The total PAH concentration of all sediment samples were below the disposal at sea criteria (2.5 mg/kg), while one intertidal surface sample had concentrations of individual PAHs (i.e., benzo[a]pyrene, benz[a]anthracene and chrysene) above the CCME ISQG. No samples were above the CCME PEL for individual PAHs. Therefore, the data presented in the PNW LNG report leads to the categorization of PAH as COPCs. A description of PAHs, along with their physical-chemical properties and potential effects, are presented in following sections.

For PCB concentrations, 85 sediment samples were analyzed for nine PCB congeners. Concentrations of individual congeners were below the laboratory detection limits in all samples except one. In this sample, PCB-1254 was 0.059 mg/kg and total PCB in the sample was below the disposal at sea screening criteria (total PCB < 0.1 mg/kg).

PCDD/Fs were analyzed in a subset of the 82 sediment samples. The initial sampling program in May to July 2013 included seven intertidal and five subtidal surface grabs and composite samples at 0 - 0.5 m and 0.5 - 1.0 m within two deep cores. The sampling program was expanded in October 2013 to include 24 samples from three pairs of cores to establish PCDD/F concentrations at 0.2 m intervals reaching depths of 1.0 to 1.4 m. Dioxin and furan concentrations are reported as toxic equivalencies (TEQ) calculated using toxic equivalency factors (TEF) for fish based on the World Health Organization 1998 guidelines (CCME 2001;

Van den Berg et al. 1998) to allow comparison with the CCME ISQG (0.85 pg/g TEQ) and PEL (21.5 pg/g TEQ).

PCDD/Fs were detected in surface sediments up to a depth of 1.5 m. From 1.5 m to 12.0 m, all samples were below the laboratory detection limit for PCDD/Fs. The intertidal surface samples had measurable concentrations ranging from 0.4 to 0.90 ng/kg TEQ with only one sample exceeding the ISQG of 0.85 ng/kg TEQ. Subtidal and surface core PCDD/F concentrations ranged from 0.06 to 2.64 ng/kg TEQ. These concentrations are above the ISQG and below the PEL of 21.5 ng/kg TEQ. Therefore, the data presented in the PNW LNG report leads to the categorization of PCDD/Fs as COPCs. A description of PCDD/Fs, along with their physical-chemical properties and potential effects, are presented in following sections.

Information on existing contaminant levels found in samples taken in the marine berth dredge area southeast of Agnew Bank came as part of a data-sharing agreement with the Prince Rupert Gas Transmission Project. Several surface and three 1.0 m core sediments were collected to the southwest of Lelu Island, within 5 km of the marine berth dredge area.

Total PAH concentrations were below detection limits (0.02 mg/kg for individual parameters) and the disposal at sea screening criterion of 2.5 mg/kg in all samples. PCB concentrations were below the detection limit (0.02 mg/kg) and the disposal at sea screening criterion of 0.1 mg/kg in all samples except one (0.120 mg/kg). Arsenic concentrations exceeded the ISQG in all 14 samples, with a maximum of 12.7 mg/kg, and did not exceed the PEL. Cu concentrations exceeded the ISQG in 12 of 14 samples, with a maximum of 35.6 mg/kg, and did not exceed the PEL. Mercury, cadmium, chromium, lead, and zinc concentrations were below the screening criteria in all samples.

Dioxins and furans were measurable in 19 surface, core (0 to 0.5 m depth) and detailed core (0.2 m increments to 1.0 m) samples. Concentrations were lower than the ISQG, ranging from 0.080 to 0.234 pg/g TEQ and with the majority of compounds present at levels below the detection limits. PCDD/F concentrations in these samples had an average of 0.11 ng TEQ/kg dw.

Sediments present at the disposal site in Brown Passage were also screened for contaminants by Environment Canada in April and October 2011. Sediment that meets disposal at sea screening criteria has been deposited at Brown Passage several times in the past decades. Results for the 55 samples collected in 2011 were generally similar to those collected from around Lelu Island, with the exception of lower As, Cu and dioxin/furan levels at Brown Passage.

The sediment characteristics reported were: Total PAHs: less than 0.02 mg/kg to 1.86 mg/kg, all below the disposal at sea screening criterion As: less than 5.0 mg/kg to 7.7 mg/kg, with one sample higher than the screening criterion Cu: 3.1 mg/kg to 24.3 mg/kg, with nine samples higher than the screening criterion Cd: 0.06 mg/kg to 0.67 mg/kg, with one sample higher than the screening criterion (0.6 mg/kg) Hg: 0.006 mg/kg to 0.064 mg/kg, all below the screening criterion (0.75 mg/kg) Dioxins and furans: 0.026 pg/g to 0.509 pg/g TEQ.

In summary, the PNW LNG report data shows that sediment characteristics within the MOF dredge area are typical of the Prince Rupert area and suggest that localized contaminant accumulations do not occur (these results are similar to those from other locations around Lelu Island and from the Fairview Phase II and Canpotex programs) and that widespread contamination of the area has occurred. PCDD/Fs were detected in sediments down to a depth of 1.5 m, with the highest concentrations in the surface sediment layers. Common dioxin

sources include atmospheric releases from combustion, waste incineration, chemical manufacturing, petroleum refining, wood burning, metallurgical processes, vehicle emissions, historic pulp and paper mill effluents (CCME 2001). Dioxins and furans are most likely a legacy of historical discharges at the former Skeena Cellulose pulp and paper mill on Watson Island, about 3 km from the MOF. As well, sawmills, wood treatment facilities and chlorophenol-treated wood chip storage areas, diesel emissions, coal combustion, municipal solid waste and other incineration stack emissions may be contributors. These chemicals are of concern because they are taken up by biota and bioconcentrate and biomagnify in the food chain, which can lead to toxicological risks (mainly in fish, marine mammals, and humans).

Chemical concentrations at the marine berth dredge area were measured from three boreholes drilled for geotechnical surveys. Characteristics of subtidal sediment to the southeast of Agnew Bank reported are that concentrations of dioxins and furans were lower than the ISQG, ranging from 0.080 to 0.234 pg/g TEQ and with the majority of compounds present at levels below the detection limits. However, it should be noted that sampling was highly inadequate to determine if these levels accurately represent the contamination present in this area, and are not useful in conclusions drawn on potential risk to ecological or human receptors.

Results for the 55 samples collected in 2011 at Brown Passage were generally similar to those collected from around Lelu Island, with the exception of lower As, Cu and dioxin/furan concentrations.

The exceedances of ISQG for Cu and As at all three sites likely reflect the baseline and natural conditions for the area since sampling shows a consistency in contamination across the area at all depths of sediment. A further and more extensive sampling regimen should be used to ascertain the accuracy of this conclusion. PAHs were undetectable in most sampling areas and samples were all below the CCME PEL but several were higher than the ISQG. PCBs were undetectable in the vast majority of samples.

Assuming the results of the sediment sampling program, as presented in the PNW LNG report, and related reports (e.g. Canpotex disposal at sea application [Stantec 2014]) are accurate, it is likely that the maximum concentrations of PCDD/Fs are not currently located at the immediate surface (0-0.1 m), and thus, many aquatic receptors are not currently being exposed to maximum concentrations. There is the potential that following dumping at the load site, that the sediments with the highest concentrations could be present at shallower depths than they are currently at the dredge site. Conclusions regarding the potential effects to ecological and human receptors appear to be based on the assumption that current marine organisms are now exposed to the highest COPC concentrations (Canpotex sampling disputes this [Stantec 2014]) and that dredging will reduce concentrations and bioavailability. More sampling with finer depth profiles would aid in assessing this, and potentially altering the positions taken on the potential magnitude of effects.

Although not presented in the PNW LNG report, in addition to predicting concentrations for comparison to benchmarks protective of aquatic organisms/fish, consideration must be given to the potential for the human health exposures. Although there is low potential for humans to be directly exposed to the sediments at any dredge or loading site, as will be further discussed in subsequent sections of this report, PCDD/Fs bioaccumulate and biomagnify in the food chain, and thus, there is the potential for humans to be indirectly exposed to PCDD/Fs via consumption of fish and shellfish from the load site. This is of particular concern based on the First Nations communities in the area, and their reliance on fish and shellfish (i.e., subsistence fishing). Given the potential for the higher concentrations and bioavailability of PCDD/F to be exposed during dumping of the dredgeate at the load site, and the use of the area for

subsistence fishing, further evaluation of human exposures via this pathway is recommended prior to approval of the proposed project.

3.1 Polyaromatic hydrocarbons (PAH)

Polyaromatic hydrocarbons, also known as polycyclic aromatic hydrocarbons (PAH) or polynuclear aromatic hydrocarbons, are compounds that consist of 2 or more fused aromatic rings and do not contain heteroatoms or carry substituents. The resulting structure is a molecule where all carbon and hydrogen atoms lie in one plane. Naphthalene ($C_{10}H_8$; MW = 128.16 g), the simplest example of a PAH, is formed from two benzene rings fused together, and has the lowest molecular weight of all PAHs. The environmentally significant PAHs are those molecules that contain two (e.g., naphthalene) to seven benzene rings (e.g. coronene with a chemical formula $C_{24}H_{12}$; MW = 300.36 g). In this range, there are a large number of PAHs that differ in number of aromatic rings, position at which aromatic rings are fused to one another, and number, chemistry, and position of substituents on the basic ring system.

The chemical properties, and hence the environmental fate, of a PAH molecule are dependent in part upon both molecular size (i.e., the number of aromatic rings) and molecule topology or the pattern of ring linkage. Ring linkage patterns in PAHs may occur such that the tertiary carbon atoms are centers of two or three interlinked rings, as in the linear kata-annelated PAH anthracene or the pericondensed PAH pyrene. However, most PAHs occur as hybrids encompassing various structural components, such as in the PAH benzo[a]pyrene (B[a]P).

Generally, an increase in the size and angularity of a PAH molecule results in a concomitant increase in hydrophobicity and electrochemical stability (NRCC 1983). PAH molecule stability and hydrophobicity are two primary factors that contribute to the persistence of HMW PAHs in the environment.

Vapor pressure characteristics determine the persistence of PAHs in the aquatic environment. Two- to 3-ring PAHs are very volatile, while PAHs with 4 or more rings show insignificant volatilization loss under all environmental conditions (Moore and Ramamoorthy 1984).

In addition, PAHs are non-polar, hydrophobic compounds, which do not ionize. As a result, they are only slightly soluble in water, which limits their distribution in aquatic environments and potential bioavailability to aquatic organisms. In general, PAH solubility in water decreases as the molecular weight increases. Alkyl (i.e., CH_2 - group) substitution of the aromatic ring results in an overall decrease in the PAH solubility, although there are some exceptions to this rule. Molecules with a linear arrangement tend to be less soluble than angular or perfused molecules.

The solubility of PAHs in water is enhanced 3- to 4-fold by a rise in temperature from 5 to 30 °C. Dissolved and colloidal organic fractions also enhance the solubility of PAHs which are incorporated into micelles (a micelle is composed of an aggregate of surface-active molecules, or surfactants, each possessing a hydrophobic hydrocarbon chain and an ionizable hydrophilic group) (Neff 1979). Due to their hydrophobic nature, PAHs entering the aquatic environment also exhibit a high affinity for suspended particulates in the water column. As PAHs tend to sorb to these particles, they are eventually settled out of the water column onto the bottom sediments. Thus, the PAH concentrations in water are usually quite low relative to the concentrations in the bottom sediments (Moore and Ramamoorthy 1984).

Physical and chemical characteristics of PAHs vary with molecular weight. As a result, PAHs differ in their behaviour, distribution in the environment, and their effects on biological systems.

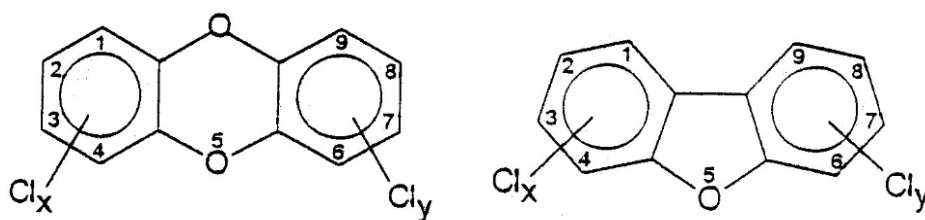
PAHs can be divided into two groups based on their physical, chemical, and biological characteristics. The lower molecular weight PAHs (e.g., 2 to 3 ring group of PAHs such as naphthalenes, fluorenes, phenanthrenes, and anthracenes) have significant acute toxicity to aquatic organisms, whereas the high molecular weight PAHs, 4 to 7 ring (from chrysenes to coronenes) do not. However, several members of the high molecular weight PAHs have been known to be carcinogenic or have chronic toxicity associated with exposure (NRCC 1983).

3.2 Polychlorinated dibenzodioxins and polychlorinated dibenzofurans (PCDD/Fs)

Dioxins encompass two main structural families, the polychlorinated dibenzo-*p*-dioxins (PCDDs) and the polychlorodibenzofurans (PCDFs), which are members of class of chemicals known as the halogenated polycyclic aromatic hydrocarbons (HPAHs). PCDFs and PCDDs are distinct compounds, however they have similar molecular structures, physical, and chemical properties (Inserm 2000).

PCDDs are a group of 75 congeners, and PCDFs are a group of 135 congeners (Shiu et al. 1988). Both classes of compounds contain a triple ring structure; an oxygenated ring connected to two benzene rings. PCDDs are connected by a pair of oxygen atoms (1,4-dioxin ring) and contain between 1 and 8 chlorine atoms ($C_{12}H_8O_2Cl_x$) (WHO 2000). PCDFs are connected by a single oxygen atom (a furan ring) and contain between 2 and 8 chlorine atoms ($C_{12}H_8OCl_x$) (WHO 2000).

Figure 1. Structure of basic PCDD/Fs. Numbers represent positions of substitution of chlorine atoms. The structure on the left is a PCDD, the one on the right is a PCDF.



The physical and chemical properties of each congener vary depending on the location and number of chlorine atoms (US EPA 2000). Due to the high variability in chemical characteristics and high toxicity of the PCDD and PCDF congeners, there is limited research available to determine their individual physical and chemical properties. The majority of research has been conducted on the 2,3,7,8-tetrachlorodibenzodioxin (TCDD) congener as it is the most toxic, and the majority of reported chemical properties is based on those for TCDD (WHO 2000).

PCDDs and PCDFs are only slightly water-soluble; while there is a broad range of available solubilities, the accepted solubility value for 2,3,7,8-TCDD is 19.3 ng/L (Marple et al. 1987). They are only slightly volatile as well, with a recommended vapor pressure for 2,3,7,8-TCDD of 1.50×10^{-9} mm Hg at 25 °C (Mackay et al. 1992a). Directly measured Henry's Law constants range from 1.99×10^{-5} to 9.4×10^{-5} atm-m³/mol (Santl et al. 1994; Dunnivant and Elzerman 1988). Log octanol-water partition coefficients (log K_{ow}) are congener specific, and range from 5.6 to 8.0 for both PCDDs and PCDFs (US EPA 2000). The accepted value of the log organic carbon partition coefficient (log K_{oc}) is 7.39 (Marple et al, 1987).

Once released into the atmosphere, PCDDs and PCFS become widely dispersed via atmospheric transport and deposition (Hites and Harless 1991). Due to the low water solubility and high hydrophobicity, PCDDs and PCDFs associate with particulate and organic matter in sediment, soil and particulates in the water column (Muir et al. 1992). Congeners with more chlorine atoms (4+) are very stable under most environmental conditions, although they may undergo atmospheric photo-oxidation and photolysis. Certain species of bacteria and fungi can degrade PCDDs and PCDFs, typically via dechlorination (Field and Sierra-Alvarez 2008).

Both PCDDs and PCDFs are not produced commercially; rather they are formed as trace impurities in the manufacture of other chemicals (e.g. polychlorinated biphenyls [PCBs], chlorinated phenols). They can also be produced during combustion (waste incineration), during the production of certain metals (iron and steel) and in the bleaching of pulp and papers (Ballschmiter 1986; Beck et al. 1989; Rappe 1994). They can also be formed naturally, for example in garden composting and in sewage (Öberg et al. 1992; Öberg et al. 1993).

4.0 FATE OF DREDGE MATERIAL AND ENVIRONMENTAL PARTITIONING

Once a dredge occurs, sediment is transported and degraded according to numerous physical, chemical, and biological processes. The sediment characteristics (e.g. composition and size) will determine its environmental fate. Moreover, other factors such as location of the deposition and weather conditions (e.g. temperature, wind speed) will impact the distribution and fate of sediments. The general factors involved in the transport, distribution, and the fate of deposited sediments that may occur at the deposition site in the application are described below.

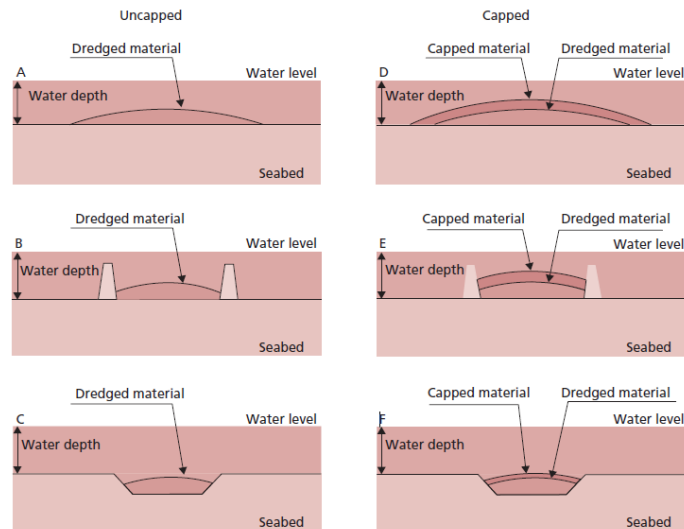
There are two major facets to sediment fate during dredging and deposition. The first is the production of a sediment plume in the region where dredging activities are occurring. Depending on the dredging technique being used, a plume of sediments is generated which may spread depending on local currents. This plume can be restricted to an area as small <100 hundred metres to an area in excess of several hundred, depending on dredging physical parameters of dredged sediments, dredging techniques and currents. These sediments will eventually settle, proximity to site of dredging dependent on particulate size and local current regimes (e.g. tidal v. unidirectional). Currents may eventually return these sediments to their origin (Bray 2008; Wasserman et al. 2013).

Dredging at the MOF is expected to occur using a clamshell dredge, which typically releases up to 1% of sediment due to bottom wake (disturbance of the sediment-water interface) from: capturing sediment in the clamshell bucket; release during closing; loss of sediments from the shovel while rising through the water column; draining during slewing and washing from descent through the water column; and debris captured in the dredge (Schroeder and Ziegler 2004). About half of the sediment release occurs at the bottom 5 m of the water column (Hayes et al. 2007). Dredging at the MOF and disposal of dredged material at Brown Passage will result in resuspension of marine sediment and potential for dispersal of contaminants from sediment.

Dredging at the marine berths during construction will removing approximately 7 million m³ of sediment, which is expected to be of somewhat similar chemical composition to sediment found in the MOF. Dredging is expected to occur using a cutter-suction dredge, which has similar sediment release rates to a clamshell dredge.

The second facet of sediment fate in dredging operations occurs when the sediments are deposited into the receiving environment. Three types of aquatic placement exist; unconfined, semi-confined and confined. The first will be explored further as it remains the most common due to ease and cost (Bray 2008) and is to be used in the application by Canpotex.

Figure 2. Unconfined, semi-confined and confined aquatic placement (Adapted from Bray et al. 1997). A: unconfined (unrestricted aquatic placement); B and C: Semi-confined aquatic placement (with lateral containment); D to F: confined (contained) aquatic placement.



Unconfined aquatic placement is the proposed method in the application. It is a reintroduction of dredged sediments in the water system from which they originate such that the returned sediments become again part of the natural sediment cycle. This involves placement of sediments on flat or gently sloping waterbed in the form of a mound (see Figure 3A). The placement sites can be dispersive or non-dispersive (retentive) depending on whether the sediment is transported out of the site by currents and/or wave action (Bray 2008).

While the type of placement influences the long-term fate of dredged sediments in the receiving environment several short-term behaviours occur during and shortly after sediment discharge. These are convective descent, collapse and mound formation, and passive dispersion.

Convective descent describes the fall of the concentrated sediment cloud (Bray 2008). The sediments fall as a high-density plume with nearly all sediments contained within the cloud; descent velocity is determined by negative plume buoyancy, drag and momentum (ERDC 2008). Some sediment enters the water column, the exact quantity depends on sediment type, plume density, descent velocity and distance between release and sediment bed (ERDC 2008). When the descent plume reaches the bottom it forms a horizontal surge as the vertical momentum is transferred to horizontal momentum called dynamic collapse (Bray 2008). The surge head will slow as it expands across the sediment bed, depositing sediments to the bed and entraining some into the water column due to currents and turbulence caused by the surge head (ERDC 2008). As discharge continues a mound develops, characteristics of which depend upon sediment type. Passive dispersion describes the motion of the sediments entrained in the water column. Advection and diffusion by currents and particle settling govern sediment motion and sedimentation rates (ERDC 2008). Only a small portion of suspended particles, 5-20%, is exposed to currents capable of transporting sediments outside of designated discharge areas (Bray 2008).

Descent characteristics are highly dependent upon discharge characteristics (Figure 2). The material from direct pipeline discharge as in the application is a liquid slurry which may contain clay balls, gravel or coarse sand material. The coarse material quickly settles, while the mixture of process water and fine particles descends to the bed to form a fluid mud mound. Some fine material may remain in suspension as a turbidity plume.

Sediment plume modelling was conducted at the proposed MOF dredge site to evaluate the short- term and long-term distribution of disturbed sediments. Plume modelling of the dredging area by ASL Environmental Services predicted that up to 2 mm of sediment will re-settle in the majority of the surrounding area up to a radius of 3 km from the MOF. Maximum sediment deposition could reach 11.3 mm within 500 m of the dredge site (excluding redeposition at the MOF where dredging occurs) in areas of low water current along Lelu Island. A plume dispersion model for the marine berth dredge area was not available. It is anticipated that the resulting plume range, concentration of suspended solids and subsequent sediment deposition upon completion of dredging activities will be greater.

Longterm behaviour of sediments occurs over months or years and includes mound consolidation, resuspension and erosion, and the transport and deposition of eroded materials. Mound consolidation occurs as the self-weight of the sediments expel pore-water and mound elevation decreases. Reduction in mound size depends upon sediment characteristics; fine-grained sediments can face upwards of a 50% reduction in deposit thickness (Bray 2008).

Resuspension and erosion are influenced by bottom current velocity, potential for wave-induced currents, sediment grain size and cohesion. Wave-induced pore pressures can destabilise mounds resulting in large submarine slides occasionally exceeding a kilometre. Sediment dwelling biota, such as crabs, lobsters and fish, can colonize the mound and cause bio-erosion of the sediments. Extent of erosion may be self-limiting as fine-grained sediments are eroded away it leaves behind coarser sediments that are more resistant to erosion (Bray 2008).

Transport and deposition depends largely upon size of the eroded materials and hydrodynamic processes in the area. High current, shallow areas allow for long-distance transport of the sediments. Depending on current regime it may be taken back towards the origin of dredged sediments (Bray 2008).

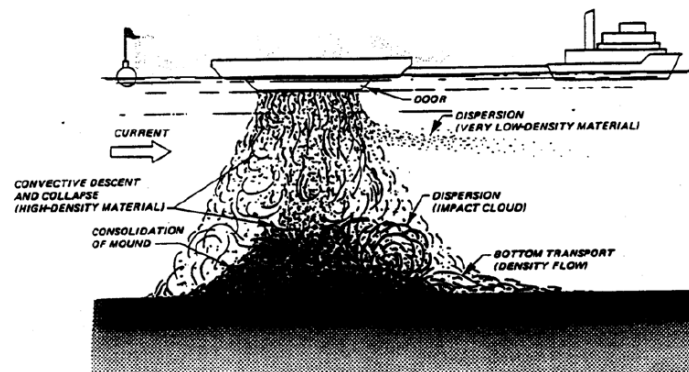


Figure 3. ERDC (2008). Potential fate of sediments released from a barge deposition operation.

5.0 BIOAVAILABILITY

Sediments within aquatic environments act as physical and biological repositories and can act as a sink for organic and inorganic contaminants (Zoumis et al. 2001). Multiple physical (rate of mixing, rate of sedimentation, diffusion, resuspension), chemical (pH, equilibration time with sediment, dissolved organic content) and biological (biotransformation, behavior, diet) factors may influence the bioavailability of contaminants in sediment. In general, high hydrophobicity, lower polarity and larger organic contaminants such as PCDD/Fs have higher likelihood to adhere to sediments and sediment organic content (EPA 2000). Specifically, moderate to high levels of clay or organic carbon within sediments tend to adsorb dissolved contaminants, which can continue to be a source of contamination after the source is depleted (EPA 2000).

Organic pollutants may associate temporarily with particulate matter and establish a water–sediment equilibrium interface (Perelo 2010). Contaminants within the aquatic environment may

be immobilized and accumulated, or possibly subjected to activation and transformation within sediments (Martinez-Jeronimo et al. 2008). According to Voie et al. (2002), organic pollutants such as PCBs and PCDDs are only available as a small fraction dissolved in water due to their hydrophobic nature. Intrinsic properties of these organic particles and the particulates suspended within the water column determine the partitioning between the dissolved phase and particulates (Voie et al. 2002). One major route of contaminant uptake in organisms is sediment ingestion (Lamoureaux and Brownawell 1999). Organic contaminants dissolved in water are considered as another important exposure source by other authors (Schrap and Opperhuizen 1990). Uptake of dissolved hydrophobic chemicals is thought to occur through the gills or skin of aquatic organisms (Randall et al. 1998).

Contaminant remobilization may occur due to changes in sediment chemistry or seabed disturbance (Eggleton and Thomas 2004). For example, through dechlorination reactions in anoxic conditions in some sediments, organic contaminants may be degraded and transferred to a more mobile form (Zoumis et al. 2001). Seabed disturbances include activities such as dredging which can result in the remobilization of buried sediment and associated contaminants (Eggleton and Thomas 2004). Dredging is often performed to remove contaminated sediments and to remediate the environment (Bridges et al. 2008) by the physical removal of contaminated sediment layers. This can be problematic as sediments must be localized elsewhere and may require further management (Perelo 2010).

According to the NRC (1997), environmental dredging is often used to minimize the spread of contaminants to the surrounding environment by removing sediment contaminated above certain levels. The resuspension of sediment via dredging can occur as dredge operations dislodge sediment particles which are dispersed into the water column (Bridges et al. 2008). The resuspended particulates may be redeposited or transported to other locations in the same water body (Bridges et al. 2008). Dissolution may occur with some contaminants into the water column and be made available for uptake by biota, an environmental exposure of some concern (Bridges et al. 2008). Several completed environmental dredging project field results indicate that post-dredging residual contaminant concentrations (expressed as contaminant concentration from surface sediments) are greater than pre-cleanup levels. Continuing or short-term risk at the site can occur as a result of this resuspension (Bridges et al. 2008). High concentrations of particulates and associated contaminants can also result from resuspension in the water column (Bridges et al. 2008). One example is that of Duwamish river remediation (Werth et al. 2012) where deep residual layers of sediment were modeled for dredging. This report indicated that the dissolved phase released was of highest importance as PCBs would be more bioavailable to fish as the contaminant dissolves into the water column (Werth et al. 2012).

The baseline study in the PNW LNG reports states that marine sediments at the MOF contain historical deposits of chemicals (i.e., PAH, PCB, PCDD/F) that are primarily within the upper 1.5 m. PCDD/F concentrations were highest in the upper 0 to 0.2 m sediment layer with gradually decreasing concentrations to a depth of 1.5 m, based on sediment core intervals of 0.2 and 0.5 m. The underlying sediment from a depth of 1.5 m to 12.5 m is relatively free of PAH, PCBs and PCDD/Fs. Data from the Canpotex application for disposal at sea (Stantec 2014) indicate that the highest concentrations of PCDD/Fs in the sediments sampled occur in the top 0.1-0.2m, therefore chemical concentrations in deeper sediment layers are higher than concentrations found in immediate surface layers. Under this scenario, deep sediments containing higher concentrations of contaminants will settle over less contaminated surface sediments. These concentrations are in some cases above ISQGs and lower than PELs. Dredging will remove the surface sediments, and potentially expose and disturb relatively cleaner sediments. Under this scenario, chemical concentrations in the sediment could potentially increase or remain similar to existing conditions. Therefore, the statement in the PNW LNG report that [“the sediment plume](#)

resulting from dredging activities will contain similar or lower concentrations of anthropogenic chemicals than the existing surface sediments' is inaccurate and many assumptions regarding ecological impacts may not be correct. Therefore, the same assumption made in the PNW LNG report as being applicable to the sediment plume resulting from the dredging activities at the marine berth site may also be inaccurate.

This information suggests that the dredging operations outlined in the application of sediments contaminated with PAH and PCDD/Fs may increase the bioavailability of these compounds at the dredge and loading sites and lead to increases in marine life exposures, their potential accumulation and effects, and (for PCDD/Fs) potential food web transfer to humans consuming impacted seafood.

5.1 Bioaccumulation and biomagnification

The purpose of this section is to detail the potential for PCDD/Fs to bioconcentrate, bioaccumulate, and biomagnify in a marine environment such as that outlined in the dredging application. PAH are not likely to biomagnify in food webs, as most of this class of chemicals are biotransformed relatively easily by organisms, particularly by those at higher trophic levels (e.g. fish, birds, mammals). These 3 processes, alone or in conjunction, can result in body burdens of xenobiotics in aquatic organisms far in excess of environmental concentrations. Thus, understanding these processes is crucial in assessing the potential adverse outcomes posed to biota in aquatic environments that are acting as a receiving body for PCDD/F contaminated sediments. The definitions for each process, and corresponding means of measuring them, are below.

Bioconcentration is the process by which a chemical is taken up into an organism from the ambient environment (abiotic sources) to higher concentrations. It is the net result of absorptive uptake and chemical elimination from the organism (Arnot and Gobas 2006). The degree to which bioconcentration occurs is termed the bioconcentration factor (BCF), and is defined as the ratio of chemical concentration in the organism to the chemical concentration in the environment.

Bioaccumulation is the process by which a chemical is taken up into an organism from the ambient environment (abiotic sources) and dietary sources (biotic sources). It is the net result of absorptive uptake, ingestion and chemical elimination from the organism. The degree to which bioaccumulation occurs is termed the bioaccumulation factor (BAF), and is defined as the ratio of chemical concentration in the organism to the chemical concentration in the environment.

Both BCFs and BAFs of organic compounds are commonly compared to the log octanol-water partitioning coefficient ($\log K_{ow}$). $\log K_{ow}$ is a measure of a chemical's hydrophobicity and lipophilicity and how it thermodynamically distributes between aqueous and organic phases (Arnot and Gobas 2006). Values between 3 and 7 (many PCDD/F congeners) tend to have a high tendency for biomagnification in aquatic organisms (Kelly et al. 2007; Barber 2008).

Biomagnification is the process by which the thermodynamic activity of a chemical in an organism exceeds that of its diet. It can be thought of an organism acquiring a higher body burden of a chemical relative to the body burden of the food it eats. The degree to which biomagnification occurs is termed the biomagnification factor (BMF), and is defined as the ratio of chemical concentration in an organism to the chemical concentration in its diet.

Bioconcentration, bioaccumulation, and biomagnification cause body-burden levels of a chemical such as PCDD/Fs to far exceed ambient concentrations in aquatic environments. This

may illicit adverse effects in affected environments despite a low environmental concentration of a chemical. Thus they are essential processes in assessing and understanding the impacts of low concentrations of PCDD/Fs. Table 1 contains known log K_{ow} , BCF, BAF, and BMF values for select common congeners of dioxins and furans.

Several studies have explored the bioaccumulation of PCDD/Fs in aquatic biota. Recently, Wan et al. (2010) conducted a study on two rivers that were subjected to heavy historical industrial use (Tittabawasee and Saginaw rivers). Addressing seven dioxin and ten furan compounds, 13 fish species (primary to tertiary consumers) were sampled. The results showed the extent of bioaccumulation to be species- and chemical-specific with all species showing a higher tendency to accumulate furans over dioxins (with regard to sum concentration of congener totals). Similar to previous studies, a positive correlation between tissue concentrations and lipid content/size of the specimen was also found (Kidd et al. 1998; Kidd et al. 2001), but a negative correlation between trophic level and chemical accumulation in tissues (Naito et al. 2003; Wan et al. 2005; Ruus et al. 2006).

Trophic dilution is not a characteristic of all dioxins and furans, rather it has been proposed to be a characteristic of particular congeners. Higher chlorinated congeners of dioxins and furans show generally less accumulation in higher trophic level organisms (Naito et al. 2003; Okumura et al. 2003; Wan et al. 2005; Ruus et al., 2006). This is reasoned to be due to reduced membrane permeability of the larger, highly chlorinated congeners (Naito et al. 2003; Okumura et al. 2003; Ruus et al. 2006) or higher metabolic transformation rates (Wan et al. 2005). Consequently, lower trophic level organisms tend to exhibit a congener profile similar to that of the pollution source whereas higher trophic level accumulation will show a preference for low chlorination congeners (Lyytikäinen et al. 2003; Okumura et al. 2003; Ruus et al. 2006).

Clearly, predicting whether a dioxin or furan congener will bioaccumulate and biomagnify should not be based solely on log K_{ow} values. While log K_{ow} provides evidence for how a chemical will partition between two phases it cannot account for actual accumulation in all organisms due to complexity of chemical-organism interactions and inter-species differences. It is generally agreed that dioxins and furans are bioaccumulative (with many BCF values >5000), these studies highlight the importance of ecologically relevant values such as BCFs, BAFs, BMFs in assessing the environmental impacts of dioxins and furans.

5.2 Exposure pathways

Once released into the environment, ecological receptors can be exposed to PCDD/Fs through four possible pathways. These routes of exposure include direct contact with contaminated sediment, ingestion of contaminated sediment, exposure to contaminated water or air (unlikely for PCDD/Fs), and the consumption of contaminated prey or food.

Bioaccumulative PCDD/Fs partition primarily into sediments in the marine environment and so aquatic-dependent wildlife species, and other organisms may be exposed to these chemicals through several pathways. For aquatic organisms, such as microbiota, aquatic algae, sediment-dwelling organisms (e.g. amphipods), and benthic fish (e.g. starry flounder), direct contact with contaminated sediment and/or contaminated pore water represents the most important route of exposure to toxic substances that partition into sediments. Direct contact with contaminated water or sediment can result in the uptake of these chemicals over the general organism body surface. However, ingestion of contaminated sediments can also represent an important exposure pathway for certain species [(e.g. organisms that process sediments to obtain food (e.g. polychaetes) and/or organisms that incidentally ingest sediments during feeding activities (e.g. benthic fish)]. Of the wildlife species that occur in the vicinity of the proposed dredge site,

sediment-probing birds (e.g. sandpipers) and omnivorous mammals (e.g. raccoons) are the most likely to be exposed through this pathway.

For aquatic-dependent wildlife species, ingestion of contaminated prey species represents the principal route of exposure to bioaccumulative substances (biomagnification). The groups of wildlife species that are likely to be exposed to PCDD/Fs through this pathway include sediment-probing birds (e.g. sandpipers; black oyster catcher), carnivorous-wading birds (e.g. great-blue herons), piscivorous birds (e.g. belted kingfishers; osprey; double-crested cormorant), carnivorous birds (e.g. surf scoter; bald eagle), omnivorous mammals (e.g. raccoons), carnivorous mammals (e.g. river otters, mink), and piscivorous mammals (e.g. harbour seals; orcas).

Low water solubility of PCDD/F congeners make it unlikely that aquatic organisms and aquatic-dependent wildlife species will be exposed to these substances to any major degree through partitioning into the surface water. However, for some organisms, such as microbiota, aquatic algae, and aquatic invertebrates, or fish, direct contact with contaminated water (likely containing dredge particulates) in the water column may represent a route of exposure as these chemicals partition into surface water. This exposure route involves uptake through the general body surface or gills. This exposure pathway is likely to be important for benthic invertebrates and benthic fish due to desorption of PCDD/Fs from bottom sediments (i.e., through exposure to near-bottom water).

For aquatic-dependent wildlife species, ingestion of contaminated water represents a very minimal route of exposure to PCDD/Fs that partition into surface water. While virtually all aquatic-dependent wildlife species are exposed to toxic substances that partition into surface water, this pathway is likely to account for a minor proportion of the total exposure for most of these species.

Since PCDD/Fs are unlikely to partition into the surface microlayer (i.e. the layer of water that is present at the water-air interface), aquatic organisms and aquatic-dependent wildlife species, direct contact with the surface microlayer will not represent a likely route of exposure and will be of relatively minor importance under these circumstances.

The PNW LNG report states that ‘there is minimal potential for surface sediments to increase in concentrations of PCDD/Fs because the highest concentrations are already in the surface layers, which decrease in concentration with depth. Dredging would mix surface sediment layers with the underlying layers with lower PCDD/F levels. The pathway where PCDD/Fs increase in marine biota from interactions with sediment, and subsequent biomagnification of PCDD/Fs of higher trophic organisms from the diet is minor.’ This statement is unlikely, as data indicate that the close sub-surface layer, but not the surface sediments may contain the highest PCDD/F concentrations (Canpotex disposal at sea application [Stantec 2014]). More importantly however, is that in the process of buried contaminated sediment resuspension, bioavailability of chemicals to organisms may increase over present conditions. In fact, the PNW LNG report contradicts itself and also states that ‘The sediment plume from dredging is a new exposure pathway for gilled and filter feeding marine organisms that could absorb PCDD/Fs from particles of suspended solids. Changes to PCDD/F concentrations in marine tissues could progress in the food chain and affect higher trophic level marine mammals and marine birds’.

The PNW LNG report also states that ‘based on low bioavailability of PCDD/Fs to organisms when exposed to sediment plumes and the absence of PCDD/F inputs to the environment from project activities. The residual effects on health risks to ecological health, from direct exposure to sediment plumes containing PCDD/Fs or subsequent trophic uptake by marine vertebrates,

[are not significant for all project phases](#)' is not supported for reasons described above. The resuspension of contaminated sediment would make PCDD/Fs more bioavailable to many organisms, and could act as a new source of these contaminants to ecological receptors in this area.

As an exposure pathway, human receptors generally do not interact with marine benthic sediments and the PNW LNG report states that '[project activity interactions with sediment quality represent an incomplete exposure pathway to human health and no further analysis is warranted](#)'. Human receptors may frequently use the marine environment for recreational uses such as fishing, kayaking or swimming, however, there would be minimal to no contact with deep ocean sediments in the dredging area. Exposure to suspended sediment particles in the water column would be short in duration and infrequent. PCDD/Fs also have poor solubility in the water column and uptake rates would be negligible given the short exposure duration and frequency for humans.

In this review, marine foods are described as local marine organisms that are harvested and consumed for nutritional or medicinal purposes by local people. Marine foods include various algae, crab, shrimp, shellfish, groundfish and pelagic fish species that are harvested by local residents in the area, First Nations, recreational users and commercial harvesting industries. As described above, the concentrations of PCDD/F made bioavailable by resuspending and uncovering contaminated sediments may increase from the proposed dredging activities, and the resulting sediment plume may result in increased exposure durations to many marine organisms as well. Suspended sediments in the water column from the plume could contain PAH and PCDD/Fs that could be taken up by a variety of marine organisms (e.g., fish, prawns and crabs, shellfish). These could be directly consumed by humans, exposing them to increased PCDD/F concentrations, or could be biomagnified (only PCDD/F) through the food chain into other species consumed by humans.

The PNW LNG report states '[that the ecological implications of introducing dioxins and furans at Brown Passage are limited. At the disposal site, water is about 200 m deep, where the diversity and abundance of organisms is expected to be lower than in shallower water \(Fairview Phase II environmental assessment \[Stantec 2010\]\)](#)'. The PNW LNG report states that '[important marine benthos species exist in the general area \(e.g., Dungeness crab, tanner crab and shrimp\), however, the important habitat areas do not appear to overlap with the Brown Passage disposal site even though the disposal site is located adjacent to the boundary for important Dungeness crab habitat](#)'. The assumption that it is unlikely to be used extensively by Dungeness crab, as this species inhabits maximum depths of 180 m (DFO 2013) and is typically found at depths shallower than 50 m (DFO 2001) is speculation only. Some fisheries overlap with the Brown Passage site as well. The fact that there is relatively low effort/catch in the disposal site for the main fisheries, compared to adjacent areas does not mean it will not be used. The main fisheries, based on publicly available spatial catch data are shrimp, salmon, and groundfish including rockfish (DFO 2011). The dispersal of sediment outside of the designated Brown Passage disposal site could introduce contaminants such as dioxins and furans into these other areas including the sensitive habitat of Flora Bank.

The PNW LNG report states that species of marine foods would only experience temporary exposures to PCDD/Fs in the plume and that PCDD/F concentrations in the tissues of marine foods are expected to remain at concentrations similar to baseline levels. This is based on the assumption that there is minimal potential for dredging to increase the bioavailability of PCDD/Fs in the surface or resuspended sediments with which marine food organisms can interact, an assumption that has not been established. The PNW LNG report does concede that there remains some uncertainty regarding the change in physical dynamics of the potential

sediment plume in the marine berth dredging site. While the sediment plume model indicates very localized sediment deposition around the MOF dredge site, the marine berth in the open ocean is more exposed with a larger dredge volume and is of longer duration, potentially leading to more dispersion of contaminated sediment. Confidence in the predictions of marine food quality would be increased with the availability of accurate plume modelling data in the marine berth area.

The PNW LNG report suggests that there are multiple lines of evidence and supporting conditions suggesting that the overall potential for PCDD/Fs in marine foods to increase in concentrations is negligible to low. The assumptions underlying this conclusion does not take into account the potential increase in the bioavailability of dioxins and furans due to dredging (nor their biomagnification potential following) and therefore conclusions that the consumption of marine foods is not expected to change substantially from existing baseline conditions cannot be made.

6.0 IDENTIFICATION OF RECEPTORS POTENTIALLY AT RISK

6.1 Introduction

A critical element for understanding potential impacts of PAH and PCDD/Fs due to dredging is the identification of the receptors at risk. Guidance is available from several sources to help identify receptors at risk in the vicinity of contaminated sites (CCME 1996; USEPA 1989; 1992; 1997; SAB 2004). Guidance from these sources generally indicates that receptors at risk include: resident species or communities exposed to the highest chemical concentrations in sediments, in surface water, and in prey species; species or functional groups that are essential to, or indicative of, the normal functioning of the affected habitat; and species at risk (i.e., extirpated species, endangered species, threatened species, or species of special concern).

The ecological receptors potentially at risk include the plants and animals that utilize aquatic habitats within the dredge areas and the potential loading area, as well as areas that may receive resuspended contaminated sediments such as Flora Bank. These groups of organisms include microbiota, aquatic algae, aquatic invertebrates, fish, birds, and mammals. The groups of ecological receptors identified in the PNW LNG report that occur within the dredge site areas, intermittently, seasonally, or continuously, are briefly described in the following sections.

Several areas of particular interest in terms of ecological receptors include Flora Bank (mainly eelgrass beds) which are ecologically valuable to the region (Department of the Environment 1973b) and provide important and crucial rearing habitat for out-migrating salmon, predominantly from the Skeena River. As well, it supports healthy populations of invertebrates including Dungeness crab and *Pandalus* shrimp (DFO 1985). Eelgrass beds are restricted to the intertidal areas of Flora Bank because the high TSS influence of the Skeena River limits the photic zone, impairing subtidal plant growth (Faggetter 2009, 2013).

Agnew Bank has relatively lower species diversity, with the main fauna consisting of sparsely distributed invertebrates, such as orange sea pens (*Ptilosarcus gurneyi*), *Pandalus* shrimp, tunicates, sponges, and various mollusc species.

Rocky subtidal areas support diverse seaweed communities, including numerous species of kelp. These plants provide food and shelter for mobile and sessile invertebrates and fish. Soft sediments rarely support seaweeds but provide suitable habitat for burrowing invertebrates, crabs (e.g., Dungeness, *Metacarcinus magister*), shrimp (*Pandalus* spp.) and flatfish (family Pleuronectidae).

6.2 Microbial Community

Microbial communities, consisting of bacteria, protozoans, and fungi, play several essential roles in marine ecosystems. First, the microbial community represents an important food source for many benthic organisms, such as worms, bivalves, and snails (Apple et al. 2001). In addition, microbial communities also play a number of key roles in the cycling and transformation of nutrients in sediments and the water column (Odum 1975). The microbial community also supports primary productivity by transforming phosphorus into forms that can be readily used by aquatic plants. Finally, carbon cycling in aquatic ecosystems is dependent on the microbial community. Although specific information on the composition of microbial communities in the study areas were not available, it is certain that the microbial community plays an essential ecological role in this area. However, these organisms were not identified as ecological receptors which may be impacted by the dredging activities or PCDD/Fs.

6.3 Plant Communities

The plant communities in the proposed dredge areas and loading area will consist of phytoplankton, periphyton, and potentially aquatic macrophytes. Phytoplankton, the small non-vascular plants that are suspended in the water column, are comprised of several types of algae. While periphyton are also non-vascular plants, they tend to be larger than the planktonic forms of algae and grow on other aquatic plants or on the bottom of the water body. Aquatic macrophytes is the general term applied to either large vascular or non-vascular plants that grow in aquatic systems (including both submergent and emergent plants).

As primary producers, aquatic plants represent a primary food source for a variety of plant-eating invertebrates (i.e., herbivores, or primary consumers). In addition, aquatic plants provide habitats for a wide variety of species, including aquatic invertebrates. Submergent and emergent aquatic plants provide critical spawning and rearing habitats for many estuarine fish species. Many aquatic-dependent wildlife species, such as ducks and geese, rely on habitats created by aquatic vegetation for reproduction and other life history stages. Hence, aquatic plants represent essential components of aquatic ecosystems. The aquatic plants that are known to occur within the proposed dredging or sediment deposition area are the eelgrass beds of Flora Bank which provide critical habitat for juvenile salmonids (Faggetter 2014).

6.4 Zooplankton Communities

Zooplankton communities in marine ecosystems can be comprised of a wide variety of animals. Some of the groups of animals that are commonly found in the water column include protozoa (which are single-celled animals) and the early life history stages of many marine invertebrates (e.g. crabs, barnacles, mollusks). In addition, several classes of arthropods are commonly encountered in zooplankton communities, such as isopods, ostracods, and copepods. Finally, the early larval stages of certain fish species are often planktonic; this group of animals is commonly referred to as nekton. The zooplankton that are known to occur within the proposed dredging areas are unknown and were not identified as ecological receptors which may be impacted by the dredging, PAH, or PCDD/Fs.

6.5 Benthic Macroinvertebrate Community

Benthic invertebrates are the animals that live in and on the sediments in marine ecosystems. Benthic animals are extremely diverse and are represented by nearly all taxonomic groups from protozoa to large invertebrates. The groups of organisms that are commonly associated with benthic communities include protozoa, sponges (i.e. Porifera), coelenterates, bryozoans,

aquatic worms (i.e. polychaetes), crustaceans (such as ostracods, isopods, and amphipods), and mollusks (such as oysters and clams). Because benthic invertebrate communities are difficult to study in a comprehensive manner, benthic ecologists often focus on the relatively large members of benthic invertebrate communities, which are known as benthic macroinvertebrates. These organisms are usually operationally defined, for example, as those that are retained on a 0.5 mm sieve.

Benthic macroinvertebrates represent key elements of aquatic food webs because they consume aquatic plants (i.e., such as algae and aquatic macrophytes) and detritus. In this way, these organisms facilitate energy transfer to fish, birds, and other organisms that consume aquatic invertebrates. Information on the benthic macroinvertebrate communities that occur in the vicinity of the proposed dredging activities include taxa such as mollusks, annelids, crustaceans, and echinoderms which were identified during a macro-invertebrate study in the area (Canpotex Disposal at Sea, Stantec 2014).

6.6 Invertebrate Communities

The invertebrate communities in marine ecosystems consist primarily of zooplankton and benthic macroinvertebrate communities. Zooplankton is the term used to describe the small animals that remain suspended in the water column in aquatic systems. In contrast, benthic macroinvertebrates are the small animals that live in (i.e. infaunal species) or on (i.e. epibenthic species) the sediments in aquatic systems. Aquatic invertebrates (i.e. primary consumers) represent essential elements of aquatic food webs because they consume aquatic plants (i.e., primary producers) and provide an important food source for fish and many other aquatic organisms. The aquatic invertebrates that are known to occur within the proposed dredging area are unknown and were not identified as ecological receptors which may be impacted by the dredging activities, PAH, or PCDD/Fs.

6.7 Fish Communities

Fish are key elements of marine ecosystems for a number of reasons. As one of the most diverse groups of vertebrates, fish are able to occupy a wide range of ecological niches and habitats (Hoese and Moore 1998). As such, fish represent important components of aquatic food webs by processing energy from aquatic plants (i.e. primary producers), zooplankton and benthic macroinvertebrate species (i.e. primary consumers), or detritivores. Fish represent important prey species for piscivorous (fish-eating) wildlife, including birds, and mammals.

Various fish species are relevant to the physical, psychological and cultural health and well-being of Aboriginal people and other land users. The waters around the project area support commercial, recreational and Aboriginal food, social and ceremonial fisheries. The key fish species used for these purposes include Pacific salmon, halibut, Pacific herring, rockfish, lingcod, sole, and eulachon. The most important commercial and recreational type of fish includes various flatfish species (Pleuronectidae) (Stantec 2014).

The marine environment surrounding the proposed facility is characterized by nutrient-rich waters, which support a diverse assemblage of fish. A variety of fish species utilize habitats within the proposed dredging areas, and likely include Pacific salmon, lingcod, rockfish, greenling, perch, flounder, herring, eulachon, Pacific salmon, and green sturgeon. These fish can be classified into three main categories based on the position in the food web and habitat use, including forage fish (e.g. herring), benthic fish (e.g. halibut), and carnivorous fish (e.g. lingcod). Although the proposed dredging areas were reported as fish habitat, species that

utilize the area are unknown and were not identified as ecological receptors which may be impacted by the dredging activities, PAH or PCDD/Fs.

The importance of Flora Bank to potential fish receptors is unquestionable. Approximately 377 million juvenile salmon exit the Skeena River on an annual basis, the composition of which is roughly 72% pink, 21% sockeye, 3% coho, 2% chinook, 1% chum, and 1% steelhead (Faggetter 2014). Juvenile Pacific salmon migrating along the BC coast turn northward as they exit and begin a migration along the coast to the Gulf of Alaska. Sampling data for juvenile salmon in Chatham Sound (Carr-Harris and Moore 2013; Gottesfeld et al. 2008), it has been estimated that approximately 88% of the juvenile salmon out-migrating from the Skeena River turn north into Inverness Passage with the remaining 12% travelling through Telegraph Passage before turning north. Those juveniles traveling through Inverness Passage will pass over Flora Bank or around the shores of Lelu and Ridley Islands. Juveniles of species which forage in epibenthic habitats will remain in these areas until they are large enough to feed in the neritic environment. Flora Bank is a high quality habitat for juvenile salmon and is in the direct path of approximately 332 million juvenile salmon, most of which (approximately 297 million) being epibenthic feeders who will use it.

6.8 Birds and Mammals

Although most avian and mammalian species are primarily terrestrial, many utilize marine and foreshore habitats through portions or all of their life history. These species consume a variety of aquatic organisms and, hence, are often termed aquatic-dependent wildlife species. These birds and mammals process energy from aquatic plants, invertebrates, and fish. In turn, these species may be consumed by other avian or mammalian predator species. As such, birds and mammals represent critical components of ecological systems.

Avian receptors that could be impacted by PCDD/Fs likely in this area can be classified into three main categories: first, piscivorous birds are considered to include those avian species that feed primarily on fish (e.g. belted kingfisher, double-crested cormorant, osprey). Sediment-probing birds are considered to include those species that consume primarily invertebrates obtained from soft sediments (e.g., black oyster catcher). Carnivorous-wading birds include those species that forage in intertidal and shallow subtidal habitats for both fish and invertebrates (e.g. great blue herons). Finally, carnivorous birds include those top-level predators that feed on fish, other birds, and carrion (e.g., Surf scoter, bald eagles).

Mammalian receptors that could be impacted by PCDD/Fs likely in this area can be classified into two main categories: piscivorous mammals which are considered to include those species that feed primarily on fish (e.g. harbour seals). Carnivorous mammals, such as river otters and mink, feed primarily on fish and aquatic invertebrates. Omnivorous mammals are considered to include those species that consume a wide variety of food items, including both plant and animal matter (e.g. raccoons, black bear). Of the most commonly observed species, humpback whale (*Megaptera novaeangliae*), northern resident and Bigg's killer whale (*Orcinus orca*) and harbour porpoise (*Phocoena phocoena*) have been identified by the Province of British Columbia, COSEWIC, and SARA as species of conservation concern. Dall's porpoise (*Phocoenoides dalli*), Pacific white-sided dolphin (*Lagenorhynchus obliquidens*), and harbour seal (*Phoca vitulina richardsi*) also frequent the area. Other species less common in the area include Loughlin's northern sea lion (*Eumetopias jubatus monteriensis*), fin whale (*Balaenoptera physalus*), gray whale (*Eschrichtius robustus*), minke whale (*Balaenoptera acutorostrata scammonii*), and sea otter (*Enhydra lutris*). Marine mammals in the Prince Rupert

area generally increase in numbers during the summer months, coinciding with the seasonally migrating fish (e.g., salmon, Pacific herring).

6.9 Species at Risk

Species at Risk, as defined under the Species at Risk Act (SARA), are receptors that require special consideration. In Canada, both the federal and provincial governments have conventions for classifying aquatic organisms and wildlife species relative to their status.

SARA provides federal legislative authority to list a species as threatened or endangered species. The purpose of the Act is to: protect these endangered and threatened species and to provide a means to conserve the ecosystems of which they are a part. The current status of listed species indicates that they may be more vulnerable than other species to the presence of contaminants in environmental media and/or to other stressors. Species at risk that may be present within the waters of the disposal site include humpback whales, marbled murrelet, northern resident killer whale, transient killer whale, grey whale, Stellar sea lion, harbor porpoise, and green sturgeon. Several federally or provincially listed avian species may also occur at the site and include the northern goshawk (*Accipiter gentilis laingi*), cassins auklet (*Ptychoramphus aleuticus*), common murre (*Uria aalge*), harlequin duck (*Histrionicus histrionicus*), surf scoter (*Melanitta perspicillata*), great blue heron (*Ardea herodias*), double-crested cormorant (*Phalacrocorax auritus*), pelagic cormorant (*Phalacrocorax pelagicus*), and short-billed dowitcher (*Limnodromus griseus*).

6.10 Humans

Humans harvesting fish/shellfish from the area have the potential to be exposed to bioaccumulated PCDD/Fs. PCDD/Fs bioaccumulate in the fatty tissues of fish and mammals, and are biomagnified in the food chain; humans, represented at the top of the food chain, therefore have the potential to be exposed. First nations communities in the area are of particular concern, based on their reliance on fish/shellfish (i.e., subsistence fishing). Although the data presented as part of the PNW LNG report is not adequate to allow for the prediction of human exposures, given the potential for the highest concentrations of PCDD/Fs in sediments to become more bioavailable and biomagnify, further evaluation of the potential for human exposures and associated risks is recommended prior to approval of the project.

7.0 POTENTIAL EFFECTS FOR ECOLOGICAL AND HUMAN RECEPTORS

7.1 Aquatic ecological receptors

Sediments provide habitat for many benthic and epibenthic organisms. They also influence the environmental fate of many chemical substances in aquatic ecosystems by acting as both sinks and subsequently sources of substances that have entered the aquatic environment. Many aquatic organisms may be exposed to chemical substances through their immediate interactions with sediments; therefore, benchmarks of environmental quality (such as sediment quality guidelines [SQGs]) are required to support protection and management strategies for freshwater, estuarine, and marine ecosystems. These SQGs can be used to assess sediment quality, to help set targets for sediment quality that will sustain aquatic ecosystem health for the long term, and to develop site-specific objectives. SQGs for the protection of aquatic life are derived from the available toxicological information on the biological effects of sediment-associated chemicals on aquatic organisms.

In using SQGs as benchmarks, adverse biological effects are not predicted when the measured concentrations of sediment-associated chemicals at a site are at or below the national SQGs. Further investigation of sediment quality at the site is usually not necessary, but may be warranted under some circumstances (e.g. when sediments at the site have low levels of TOC, when other variables (e.g. dredging operations) are suspected to be increasing the bioavailability of chemicals. The potential for observing adverse biological effects is recognized when the concentration of one or more sediment-associated chemicals is greater than the SQG, with the incidence and severity of these effects generally increasing with increasing chemical concentrations (Long et al. 1994).

A second sediment quality assessment value, the PEL represents the lower limit of the range of chemical concentrations that are usually or always associated with adverse biological effects. The national SQG and the PEL are used to define three ranges of chemical concentrations for a particular chemical, those that are rarely (<SQG), occasionally (between the SQG and the PEL), and frequently (>PEL) associated with adverse biological effects (MacDonald 1993; Long et al. 1994). The quantification of the incidence of biological effects within each of these concentration ranges provides a useful tool for estimating the probability of observing similar adverse effects within the defined concentration ranges of particular chemicals. Therefore, the frequency with which and degree to which measured sediment chemical concentrations at a site fall within each of these concentration ranges are useful to distinguish sites and chemicals of little toxicological concern, of potential toxicological concern, or significantly hazardous to exposed organisms.

The PNW LNG report states that ‘[sediment and water quality guidelines indicate levels below which adverse effects on marine life are not expected. Due to the conservative methods in which they are derived \(use of large safety factors, mix of toxicity endpoints in laboratory tests\) and their generic nature, the guidelines do not define levels at which adverse effects could occur](#)’. However, sediments with measured chemical concentrations between the national SQG and the PEL are considered to represent potential hazards to exposed organisms. Although adverse biological effects are possible within this range of concentrations, their occurrence, nature, and severity are difficult to reliably predict on an a priori basis. Specific conditions at these sites are likely to control the expression of toxic effects. Further investigations on these sediments are needed to determine whether sediment-associated chemicals (PAH and PCDD/F) represent significant hazards to aquatic organisms. Such investigations may include the determination of background concentrations for naturally occurring substances and/or a suite of biological tests designed to evaluate the toxicological significance of particular chemicals (with respect to key species of aquatic biota and factors at the site that may be influencing the bioavailability of the chemical).

The report states that ‘[on this basis, deposition of sediment contaminants at the Brown Passage disposal site does not appear to pose a risk to health of fish, marine mammals or humans: existing levels at Brown Passage and in the dredge material are similar for metals, PAHs and PCBs \(well below the PEL and most are below the ISQG\)](#)’. This statement cannot be validated for PAH as some samples were found to be above ISQGs, and according to the definition of SQG and PEL and the concentrations reported, some deleterious effects may occur in marine organisms due to exposure, however, it is unlikely that PAH would affect mammals, avian, or human receptors due to their low biomagnification ability.

There is still some question as to whether the levels of As and Cu in this sediment is of concern. Sediment quality guidelines formulated on the basis of biological-effect data of sediment-associated chemicals are intended to be used as nationally consistent benchmarks. During their implementation, however, allowance must be made for the incidence of natural inorganic and organic substances in sediments. Adverse biological effects may be observed below measured

chemical concentrations that are attributable to natural enrichment. However, management concerns over the potential for adverse effects of sediment-associated chemicals (particularly trace metals) must be practically focused on those chemicals whose concentrations have been augmented above those that would be expected to occur naturally. Therefore, the potential for adverse biological effects as indicated by the exceedances of SQGs must be evaluated in conjunction with other information such as the natural background concentrations of substances.

The PNW LNG report states definitively that the As and Cu are natural background levels. More sampling needs to be performed to clearly establish this fact. An interpretive tool has been developed that provides an effective means of distinguishing the probable origin (i.e., natural vs. anthropogenic) of many metals in marine sediments (Schropp and Windom 1988; Schropp et al. 1989; Loring 1990, 1991; Schropp et al. 1990; Loring and Rantala 1992; MacDonald 1993). This method involves determining the ratio of measured trace element concentrations to that of a reference element at a number of uncontaminated sites (such ratios are relatively constant in the earth's crust). Although normalizations to a reference element can be accomplished using a number of naturally occurring elements (e.g., aluminum, iron, lithium), lithium appears to be the most appropriate for redox positive sediments in marine systems in eastern Canada (Loring 1990, 1991).

7.2 Avian and mammalian ecological receptors

In September 2013, samples of crab (*Metacampus magister*), clam (*Macoma sp.*, *Mya arenaria*) and prawn (*Pandalus hypsinotus*) were collected within 3 km of the MOF based on the anticipated sediment plume distribution and the southern end of Lelu Island. Samples were analyzed for all congener classes of PCDD/F. These samples included 16 crab muscle, 16 composites mixtures of *Macoma sp.* and *Mya arenaria*, and 8 prawn samples. The lower-bound and mid-point average concentration of PCDD/F were compared to the tissue residue guidelines (TRGs) to protect mammals and birds that consume aquatic biota.

Overall, average concentrations of PCDD/F measured in crabs, prawns and clams were below concentrations that would adversely affect mammals and birds that consume aquatic biota. For mammalian consumers of aquatic biota, the average concentration of PCDD/Fs in the muscle tissues was 0.33 ng TEQ/kg wet weight (ww) compared to the tissue residue guideline of 0.71 ng TEQ/kg ww. For avian consumers of aquatic biota, the average concentration of PCDD/Fs in the muscle tissues was 0.59 ng TEQ/kg ww compared to the tissue residue guideline (TRG) of 4.75 ng TEQ/kg ww. This is true of present conditions, however, if the bioavailability of these compounds increases as expected, then TRGs in prey items may increase and mammalian and avian consumers of aquatic biota may be at risk.

7.3 Human receptors

Although not presented in the PNW LNG report, in addition to predicting concentrations for comparison to benchmarks protective of aquatic organisms, consideration must be given to the potential for the human health exposures. Although there is low potential for humans to be directly exposed to the sediments at the dredge or load sites, as will be further discussed in subsequent sections of this report, PCDD/Fs bioaccumulate and biomagnify in the food chain, and thus, there is the potential for humans to be indirectly exposed to PCDD/Fs via consumption of fish and shellfish from the load site. This is of particular concern based on the First Nations communities in the area, and their reliance on fish and shellfish (i.e., subsistence fishing). Given the potential for increased bioavailability of these chemicals during dredging and dumping of the dredgate at the load site, and the use of the area for subsistence fishing, further

evaluation of human exposures via this pathway is recommended prior to approval of the project.

Estimations of human exposure and associated risks related to consumption of fish/shellfish from the area requires the completion of a human health risk assessment (HHRA); an HHRA would consider the bioaccumulation of PCDD/F in fish/shellfish, and subsequent consumption of fish/shellfish by local consumers, including First Nations subsistence consumers. The HHRA would then include the estimation of daily intakes of PCDD/F on an mg/kg bw/day basis, and the subsequent estimation of health risks to consumers. The WHO (2005) toxic equivalency factors (TEFs) for mammals and humans would be applied in the estimation of human health risks. Based on the conservatism and uncertainty in bioaccumulation and food chain models, the use of measured tissue concentrations (over modeled) is preferred. We understand that tissue concentrations of crab, clams and prawns in the area of the dredge site were determined as part of the application; as previously discussed, based on the potential for increased bioavailability of PCDD/F during dredging and dumping, there is the potential that these concentrations will underestimate bioavailability. Furthermore, tissue concentrations were not determined for fish species.

In the PNW LNG report, the concentrations of PCDD/Fs reported for the sediment samples were calculated using the WHO 1998 toxic equivalency factors (TEFs) for fish; the estimated toxic equivalents (TEQs) were then compared to the CCME Interim Sediment Quality Guidelines (ISQGs) and Probable Effect Levels (PELs), guidelines that have been derived to be protective of aquatic receptors. The WHO (2005) TEFs for mammals and human have not been considered in the PNW LNG report in this regard. The application of the WHO (2005) TEFs to an abiotic medium, such as sediment, has limited toxicological significance. Furthermore, we are not aware of sediment guidelines/benchmarks for PCDD/F from Canadian agencies that have been derived to be protective of human health. The Oregon Department of Environmental Quality has derived a guideline for the protection of human consumers; the guideline is intended as a screening level value to determine the need for subsequent bioaccumulation modeling/testing (i.e., tissue sampling), and ranges from 0.0011 pg TEQ/g PCDD/F for subsistence consumers to 1.1 pg TEQ/g PCDD/F for the general population. Given the TEQs (based on the WHO, 1998 TEFs) reported in the PNW LNG report and the relationship between the WHO, 1998 and WHO, 2005, TEFs, it is anticipated that if the WHO, 2005 TEFs were applied to the sediment data included in the PNW LNG report, that concentrations would exceed the Oregon DEQ screening level for subsistence consumers by a minimum of three orders of magnitude. This further supports the recommendation for further assessment of potential human health risks prior to approval of the application.

8.0 EFFECTS OF DIOXINS AND FURANS ON MARINE LIFE

2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) is the most toxic chemical of the group of hydrophobic, halogenated aromatic compounds that include similarly structured PCDDs, PCDFs, and polychlorinated biphenyls (PCBs) (Poland et al. 1982; Safe 1986). As a result of its toxicity and association with aquatic sediments, biota, and the organic carbon fraction of ambient waters, TCDD poses a potential risk to aquatic organisms such as fish, invertebrates, marine mammals, waterfowl and humans from the proposed dredging operation.

8.1 Invertebrates

Bivalves have been used extensively to mainly study the bioaccumulation of TCDD. For example, the soft-shelled clam, the eastern oyster, and the blue mussel are able to bioaccumulate TCDD as well as other dioxins and furans from the sediments and suspended

sediments (Brown 1991; Pruell et al. 1993; Rappe et al. 1991; Rhodes et al. 1997). Oysters, and in particular the eastern oyster (*Crassostrea virginica*), are useful biomonitors for the effects of compounds such as PCDD/Fs as they are sessile, in continual contact with the water and sediment, filter large quantities of water allowing for chemical bioaccumulation, and are sensitive to many xenobiotics (Wintermyer et al. 2005). These accumulations are seasonally variable as bivalves will undergo a seasonal change in the percent body lipid during gametogenesis and gonadal development; this increase in lipid affects the concentration of dioxin present during gonadogenesis as a result of TCDD's mobilization into lipid produced for gamete maturation (Cappuzzo et al. 1989; Haddad et al. 2000; Saucedo et al. 2002; Wintermyer et al. 2005). Cooper and Wintermyer (2009) demonstrated that TCDD preferentially accumulates in the gonads of the bivalve mollusks, eastern oyster (*C. virginica*) and soft shell clam (*Mya arenaria*).

8.1.1 Reproductive toxicity

In Miller et al. (1973), a significant decrease in reproduction was demonstrated in snails (*Physa sp.*) and annelids (*Paranais sp.*) following 36 and 55 days of exposure, respectively, to 200 ppt of TCDD (Miller et al. 1973).

TCDD exposure also resulted in abnormal gametogenesis in female and male oysters (*C. virginica*) which included: (i) incomplete oocyte division, (ii) inhibition of oocyte growth and maturation, (iii) unsynchronized sperm development, and (iv) inhibition of spermatogenesis (Wintermyer and Cooper (2003)). The results presented in this review provide evidence that reproduction in bivalve mollusks is highly sensitive to TCDD exposure (2 to 20 pg/g wet weight). The TCDD body burdens that resulted in altered gonad development were comparable to those observed in field populations (*M. arenaria* 0.5–20 pg/g wet weight and *C. virginica* 0.15–3.2 pg/g wet weight) (Brown et al. 1994; Wintermyer and Cooper 2003).

Daphnia magna exposed to a range of concentrations of TCDD (0.1 to 1000 ng/ml) altered reproduction of *D. magna* at different times of exposures to TCDD from 0 to 16 days (Wu et al. 2001). There were 3 very clear reproductive peaks on exposure day 2, 5, and 7 in all test and control groups. The reproductive capacity for all groups treated with TCDD significantly decreased after day 8.

8.2 Fish

8.2.1 Developmental toxicity

Lake trout (*Salvelinus namaycush*) eggs demonstrated significantly reduced hatchability at TCDD concentrations \geq 226 ppt and that the greatest TCDD-related mortality occurred during the sac fry stage (Walker et al. 1991). In all TCDD-exposed groups (34–302 ppt), sac fry that died developed subcutaneous yolk sac edema prior to death, resembling blue-sac disease. This disease affects the fry of salmon and trout before the yolk sac is absorbed; it usually appears 1 or 2 days after the fry have hatched. The first symptom is an enlargement of the yolk sac which becomes so heavy that the fish are unable to rise to the surface and can no longer maintain an upright position. The enlargement of the yolk sac is due to the accumulation of a serous fluid (often a bluish tinge, which has given rise to the name commonly applied to the disease) in the abdominal cavity between the inner and outer walls of the yolk sac (Davis 1953; Brinkworth et al. 2003). Other characteristics of blue-sac disease are pericardial edema, hemorrhaging, skeletal deformities, fin rot and cardiovascular dysfunction (Billiard et al. 1999; Scott and Hodson 2008)

In the study by Dong et al. (2001), the effects of TCDD on cell death in zebrafish embryos (*Danio rerio*) during the early stage of development were investigated. As shown by terminal transferase-mediated nick-end-labeling staining, TCDD exposure significantly increased the occurrence of pycnotic cell death, especially in the dorsal midbrain (optic tectum). The ultrastructures of these pycnotic cells showed apoptotic features such as condensation and cleavage of chromatin. Therefore, TCDD can induce apoptosis in the central nervous system during development.

Developmental abnormalities that included hemorrhaging, loss of vascular integrity, edema, stunted development and death were found in mummichog (*Fundulus heteroclitus*) embryos following exposure (nanoinjection or water bath) to TCDD (Toomey et al. 2001). To identify a possible cause for these developmental abnormalities, the effects of TCDD on apoptotic cell death were examined. TCDD exposure increased apoptotic cell death in several tissues including brain, eye, gill, kidney, tail, intestine, heart, and vascular tissue.

Similar developmental abnormalities were reported in Yamauchi et al. (2006). They characterized the early life stage toxicity related to the expression of aryl hydrocarbon receptors and cytochrome P450 1A in red seabream (*Pagrus major*). Embryos at 10 h post-fertilization (hpf) were treated with 0 to 100 µg/L TCDD for 80 min via a waterborne exposure. TCDD, in a concentration-dependent manner elicited developmental toxicities including mortality, yolk sac edema, retarded body growth, spinal deformity, reduced heart rate, shortened snout, underdeveloped fins, heart, and lower jaw.

8.2.2 Reproductive toxicity

Successful reproduction is a necessary process for the survival of any species. Compounds that interfere with normal male and female gonad development and/or post-fertilization development have the potential to alter population dynamics.

The reproductive toxicity of TCDD in fish is of significant interest since this dioxin is known as an endocrine disrupting compound. It inhibits estrogen biosynthesis in fish most likely by inhibiting steroidogenic enzyme activity (Hutz et al. 2006). Estrogens coordinate processes that are vital for population viability including the regulation of embryonic development and all aspects of reproductive development from sex differentiation to gonad maturation, reproductive cycling and behavior. In fish, periods of gonad differentiation and reproduction in mature adults offer two “windows” of enhanced susceptibility to disruption by TCDD. Since endocrine signaling is important for the regulation of early development and gonad differentiation, exposure to TCDD during critical ontogenetic periods may cause permanent functional changes that result in reduced fitness and reproductive capacity later in life (Guillette et al. 1995; Bigsby et al. 1999; Segner 2006). This may occur by TCDD exposure altering reproductive behavior, sexual selection and/or population sex ratio along with decreasing reproductive capacity. These alterations can greatly affect genetic diversity and community structure as well as decrease long-term fitness of feral fish populations (Keller and Waller 2002; Guinand et al. 2003).

Reproductive toxicity of TCDD in zebrafish (*Brachydanio rerio*) has been intensely studied, and consists of reduced fitness as both reproductive capacity and the recruitment of offspring is reduced. Most of the research on TCDD reproductive toxicity in zebrafish has examined the effects on female reproduction and demonstrated that ovarian development and egg release is significantly impaired. In adult zebrafish, dietary exposure to acutely toxic levels of TCDD led to dose-dependent reductions in egg production and a complete suppression of spawning activity, corresponding with arrested gonad development and oocyte atresia (Wannemacher et al. 1992).

Exposure to sublethal concentrations of TCDD have been linked to the impairment of female reproduction. Adults exposed via the diet demonstrated reduced egg release along with altered follicular development, decreased serum 17β -estradiol and decreased vitellogenin concentrations, all of which correlate with reduced reproductive capacity (King-Heiden et al. 2005; King-Heiden et al. 2006). The mechanisms by which TCDD modulates follicular development within a mature ovary are not understood. Histopathological analyses of the ovary also show that the adverse effects of TCDD on follicular development and vitellogenesis probably result from a direct effect on the ovary by modulating follicular development and inducing follicular atresia (King-Heiden et al. 2006; King-Heiden et al. 2009).

Although TCDD has known effects on the adult ovary, little is known about its effects on the developing gonad. Since reproductive hormones regulate gonad development, there is potential for TCDD to impact sex ratios and sexual differentiation as well as cause organizational effects that alter reproductive success later in life. Waterborne exposure of zebrafish to TCDD from the embryo stage of development through sex differentiation impairs reproductive capacity of not only the TCDD-exposed zebrafish when they reached adulthood, but also their offspring (King-Heiden et al. 2009; King-Heiden et al. 2011). Some TCDD-exposed females had ovarian lesions (King-Heiden et al. 2011) while TCDD exposed males showed no lesions in the testis. Approximately half of the TCDD-exposed females produced only slightly fewer eggs per spawn compared to control females, while the other half exhibited complete reproductive failure (< 20 eggs/spawn) (King-Heiden et al. 2009). While the study by King-Heiden et al. (2009) did not directly correlate ovary histopathology to reduced reproductive capacity of the TCDD-exposed females, the results revealed that ovaries from these TCDD-exposed females contained significantly more atretic follicles, smaller vitellogenic follicles and around half of the TCDD-exposed females exhibited malformed ovaries (King-Heiden et al. 2011). Since other aspects of reproduction (i.e., fertility, gamete quality and recruitment) were also impaired in zebrafish, this implies that exposure to low concentrations of dioxins during early life stage development and sexual differentiation in fish could pose a threat to the sustainability of TCDD-exposed feral fish populations (King-Heiden et al. 2009).

There are insufficient studies that have looked at the effects of TCDD exposure on male fish reproduction. Aside from a lack of observed pathology in the testis, male zebrafish exposed to TCDD during early stages of development appear to be a larger contributor to reduced female egg release during spawning than TCDD-exposed female zebrafish (King-Heiden et al. 2009; King-Heiden et al. 2011). This finding in male zebrafish necessitates a more careful evaluation of the impacts of TCDD exposure on the testis, sperm count, sperm motility and male spawning behavior.

While direct impacts of TCDD on gonad development, egg production and fertilization success have obvious impacts on reproductive capacity, it is also important to consider the effects of TCDD exposure on the recruitment of offspring. Maternal transfer of TCDD to eggs can induce the typical signs of larval toxicity (e.g. blue sac disease), decrease offspring survival and reduce offspring recruitment (Peterson et al. 1993; Cook et al. 2003; King-Heiden et al. 2005; Tillitt et al. 2008). Nevertheless, some effects on recruitment may not be only related to the maternal transfer of TCDD to the eggs and subsequent uptake of TCDD from the eggs by the developing embryos and larvae. This point is illustrated in the study by King-Heiden et al. (2009) by the first filial (F1) generation offspring, derived from initial parent (F0) generation zebrafish exposed to TCDD during early stages of development, who also showed reduced reproductive success and ovarian malformations. The F1 generation zebrafish were only exposed to TCDD as a germ cell. That is, germ cells giving rise to the F1 generation were directly exposed to TCDD during early development of the F0 generation. There was no other exposure of the F1 generation to TCDD. Yet the F1 generation still exhibited reproductive toxicity which demonstrates that their

germ cell-only exposure to TCDD was sufficient to cause reproductive toxicity (King-Heiden et al., 2009). The significance of this finding is that it raises the possibility for TCDD reproductive toxicity in fish to be trans-generational in nature.

In adult female rainbow trout (*Oncorhynchus mykiss*), exposure to dietary TCDD (1.8 ng/kg) during the reproductive season resulted in an accumulation of TCDD into tissues and eggs, decreased fry survival, and decreased adult survival (Giesy et al. 2002).

Eggs from sexually mature female lake trout (*Salvelinus namaycush*) were found to be non-viable following exposure to TCDD concentrations of 233-387 ng/kg, while concentrations of 50-152 ng/kg resulted in a dose-related increase in sac fry mortality associated with yolk sac edema, craniofacial alterations, and arrested development, resembling blue-sac disease (Walker et al. 1994).

The sexual ratio of rare minnow (*Gobiocypris rarus*) exposed to TCDD (as well as 17 β -estradiol) from embryo to sexually mature revealed feminization and overdevelopment of connective tissue in male fish gonad in the 2 to 30 pg/L TCDD concentration range (Wu et al. 2001).

8.2.3 Cardiotoxicity

While toxic effects on some organ systems may not result in mortality, the cardiovascular system is perhaps the most evident example of a close association between organ dysfunction and mortality (Heideman et al. 2005). The first published study of TCDD developmental toxicity in lake trout in 1991 identified the cardiovascular system as the initial tissue affected in both the TCDD toxicity syndrome and in blue sac disease of developing lake trout (Spitsbergen et al. 1991). Six years later, it was demonstrated that TCDD exposure also adversely affected the cardiovascular system of developing zebrafish (Henry et al. 1997). Zebrafish embryos treated with TCDD shortly after fertilization developed malformed hearts and pericardial edema at 72 h post-fertilization (hpf), followed by the onset of yolk sac edema (96 hpf) and mortality (132 hpf). Reduced blood flow in vascular beds of the trunk, head and gills and slowed heart rate also occurred in TCDD-treated larvae prior to, or concurrently with, the onset of other signs of toxicity (Henry et al. 1997). This was the first report in zebrafish to demonstrate that the cardiovascular system was a target of TCDD developmental toxicity. This conclusion was reinforced by a subsequent study on TCDD developmental toxicity in rainbow trout, where reduced perfusion of tissues with blood and arrested heart development were identified as playing a major role in TCDD-associated developmental toxicity (Hornung et al. 1999).

Delayed regression of the common cardinal vein was found in zebrafish and red seabream larvae exposed to TCDD (Bello et al. 2004; Yamauchi et al. 2006) and malformation of the mesencephalic vein and prosencephalic artery was discovered in the head of zebrafish larvae exposed to dioxin (Teraoka et al. 2010). The mechanism of the TCDD-induced decrease in midbrain regional blood flow in zebrafish larvae has been the focus of recent research. It has been shown that a TCDD-induced increase in cyclooxygenase 2 (COX-2) is involved in the mesencephalic vein circulation failure, which can be prevented by selective COX-2 inhibitors and rescued by knocking down COX-2 activity (Teraoka et al. (2009).

Results from two different TCDD dosing paradigms suggest that the TCDD-induced decrease in cardiac output is the cause of reduced peripheral blood flow in zebrafish rather than a consequence of it. First of all, in embryos treated shortly after fertilization, a decrease in cardiac output was detected at 60 hpf, prior to, or concomitant with, the onset of pericardial edema and decrease in peripheral blood flow (Antkiewicz et al. 2006). Secondly, larvae treated at 72 hpf

exhibited a reduction in stroke volume and cardiac output at 80 hpf, prior to the decline in peripheral blood flow at 84 hpf (Antkiewicz et al. 2005; Carney et al. 2006). Other TCDD-induced effects on the zebrafish heart include a reduction in heart size at 72 hpf and altered heart morphology (Antkiewicz et al. 2005). At 96 hpf, the heart of TCDD-treated embryos was no longer looped, with the ventricle small and compacted and the atrium thin and elongated. These effects were AhR-dependent (Antkiewicz et al. 2006). TCDD also inhibited heart valve development (King-Heiden et al. 2011). This was evidenced by a failure of valve cushion and subsequent valve leaflet formation at the atrio-ventricular and bulbo-ventricular valve junctions, resulting in blood regurgitation between heart chambers (Mehta et al. 2008; King-Heiden et al. 2011). TCDD exposed larvae also exhibited abnormal development of the bulbus arteriosus (Mehta et al. 2008).

The cardiotoxic responses elicited by TCDD exposure suggest that signaling pathways responsible for normal heart development are disrupted (King-Heiden et al. 2011). By evaluating the TCDD-induced gene expression changes underlying the cardiotoxicity in larval zebrafish, its mechanism of action in causing this cardiotoxicity is becoming illuminated. Carney et al. (2006) performed a time course analysis of the transcriptional response to TCDD exposure that began at 72 hpf in the hearts of zebrafish larvae 1, 2, 4 and 12 h later. TCDD induced rapid expression changes of 42 genes (within 1 h), the majority of which were involved in xenobiotic metabolism, proliferation, contractility and regulation of heart development (Carney et al. 2006). These rapidly induced gene expression changes preceded signs of TCDD-associated cardiovascular toxicity, making them strong candidates as downstream targets of aryl hydrocarbon receptor signaling that contribute and/or mediate TCDD-induced cardiovascular toxicity. This immediate transcriptional response in the heart of zebrafish larvae suggests that the heart may be direct target of TCDD toxicity. Specifically, a cluster of genes that includes genes that promote cell division and growth by functioning in DNA replication, DNA repair, cell division, transcription and chromosome assembly and maintenance are down regulated preceding TCDD-induced attenuation of heart function (Carney et al., 2006; Chen et al. 2008). Taken together, down-regulation of this cluster of genes correlated with the cell cycle is considered responsible for the reduction in number of cardiomyocytes in the TCDD-exposed zebrafish embryo heart (King-Heiden et al. 2011). Furthermore, TCDD affects the expression of genes in zebrafish embryos that are important for heart function, and suggests altered expression of genes for both sarcomeric components and mitochondrial reactive oxygen species production may play a role in TCDD-induced cardiac toxicity (Handley-Goldstone et al. 2005). Finally, the failure of valve formation in the heart of zebrafish embryos exposed to TCDD is associated with increased ectopic expression, as well as mislocalized expression, of *Bmp4* and *Notch1b* in areas of the heart where valves would normally form (Mehta et al. 2008; King-Heiden et al. 2011).

In the study by Cantrell et al. (1998), medaka embryos were treated with a single 2 h waterbath exposure of TCDD within 5 hpf. The egg concentrations of TCDD were 0, 1.5, 2.9, 5.8, 11.7, and 17.8 ng TCDD/g egg. Pericardial sac edema and vascular hemorrhaging were the predominant concentration-dependent morphologically visible lesions at all of the developmental stages examined [stage 26 (this stage is identified by development of vitelline vasculature), stage 33 (embryos at this stage are characterized by development of the gill and digestive organs), stage 40 (embryos at this stage are characterized by hatching of the embryo from the chorionic membrane and by a marked increase in utilization of yolk sac reserves), and at 3 d post-hatch)].

In addition to zebrafish, red seabream and medaka, other studies with other fish have shown that the cardiovascular system is adversely affected early in development following TCDD exposure. These adverse effects include (i) edema (yolk-sac, pericardial, and meningeal), (ii)

craniofacial malformations, (iii) slowed blood flow in vascular beds of trunk, head and fills, and (iv) hemorrhaging in lake trout (Spitsbergen et al. 1991; Walker et al. 1991; Guiney et al. 1997), rainbow trout (Walker et al. 1992) and fathead minnows (Olivieri and Cooper 1996).

8.2.4 Histopathology

In Henry et al. (1997), the histopathology and toxicity of TCDD in the early life stages of zebrafish (*Danio rerio*) was characterized from 12 to 240 h post-fertilization (hpf) following water-borne exposure of newly fertilized eggs. Egg doses of ≥ 1.5 ng [3 H]TCDD/g wet weight elicited toxic responses in zebrafish larvae with pericardial edema and craniofacial malformations first observed at 72 hpf, followed by the onset of yolk sac edema (96 hpf) and mortality (132 hpf). Histological examination of TCDD-treated zebrafish revealed a variety of epithelial tissue lesions including arrested gill development and ballooning degeneration and/or necrosis of the renal tubules, hepatocytes, pancreas, and all major brain regions. In addition, mesenchymal tissue lesions were discovered, which included subcutaneous edema in the head, trunk, and yolk sac, edema of the pericardium and skeletal muscle, and underdevelopment of the swim bladder.

8.2.5 Immunotoxicity

Paper and pulp mill effluents are complex mixtures of myriad organic and inorganic toxic compounds, including PCDDs, PCDFs, heavy metals and pentachlorophenol (Oikari and Niittyala 1985; Suntio et al. 1988; Servos et al. 1994). In a study conducted by Fatima et al. (2001), the effects on the humoral immune response (also known as antibody-mediated specific immunity) in the snakehead fish, *Channa punctatus* (Bloch) exposed to 1% concentration (v/v in water) of paper and pulp mill effluent found significant decreases in the splenic and pronephric (head kidney) cellularity. Fatima et al. (2001) also looked at the effects of the length of exposure (15, 30, 60, and 90 d) to paper and pulp mill effluent on a number of parameters in snakehead fish. Short-term exposure for 15 d induced an elevated PFC response, but change was not statistically significant. On the other hand, exposure for 30, 60, and 90 d significantly reduced PFC response. Furthermore, long-term exposure caused significant reduction in the weights of lymphoid organs (spleen, head kidney, and total kidney). As a whole, these results demonstrated that the chemical constituents of paper and pulp mill effluent (which includes dioxins and furans) have an immunosuppressive effect in fish.

8.3 Marine mammals

8.3.1 Immunotoxicity

Impaired immune functions such as (i) lower natural killer cell activity, (ii) decreased mitogen- and antigen-induced lymphocyte proliferation in peripheral blood, (iii) decreased mixed lymphocyte reaction, and (iv) suppression of *in vivo* delayed-type hypersensitivity and antibody responses were found in captive harbor seals (*Phoca vitulina*) fed fish contaminated from the Baltic Sea with high levels of organochlorines (which include dioxins and furans) compared to seals that fed on less contaminated fish from the open waters of the Atlantic Ocean (Ross et al. 1995; De Swart et al. 1996, Ross et al. 1997). Most of these effects on the immune system were correlated with dioxin-like polychlorinated biphenyls and other dioxin-like compounds in the blubber of the seals (De Swart et al. 1996).

8.4 Waterfowl

The avian egg is frequently used to investigate pollutant effects on wild birds and to monitor pollutant trends in the environment (Peakall and Boyd 1987; Furness 1993). Eggs are an important route of chemical elimination for female birds, particularly for highly lipophilic compounds (such as dioxins and furans), and measurement of contaminants in eggs allows comparison of maternally deposited doses to those associated with toxicological effects in field and lab investigations (Augspurger et al. 2008). Colonial waterbirds are frequent subjects of such assessments because of their high trophic status and the ease with which large numbers of eggs and corresponding data on productivity and health of sibling embryos can be collected. The value of using colonial waterbird reproductive outcomes, deformity rates, and contaminant accumulation in integrated pollutant assessments has been demonstrated in the Great Lakes (Fox et al. 1991), the North American Atlantic and Gulf coasts (Blus 1982; Custer et al. 1983), and Europe (Bosveld et al. 1995).

8.4.1 Reproductive toxicity

White and Seginak (1994) studied the effects of the PCDD/F contamination on nesting wood ducks (*Aix sponsa*) during 1988 to 1990 in a central Arkansas wetland. Dioxin and furan residues in wood duck eggs were found to be 50 times higher in eggs near the point source of exposure than in eggs from an uncontaminated reference site. Moreover, parameters such as nesting success, hatching success, and duckling production were significantly suppressed in wood ducks inhabiting the contaminated environments.

A year later, a study by White and Hoffman (1995) found significantly higher levels of oxidative stress and increased teratogenic effects in wood ducklings at a more contaminated nesting sites along Bayou Meto downstream from a hazardous waste site in central Arkansas compared to other sites.

9.0 EFFECTS OF DIOXINS AND FURANS IN HUMANS

9.1 Biochemistry

The effects of dioxins and dioxin-like compounds involve the interaction of dioxin with the dioxin receptor, which is also known as the Ah receptor (AhR) (Bock and Köhle 2006). The AhR is a ligand-activated transcription factor. The AhR is involved in organogenesis, in detoxification of endo- and xenobiotics, and in mediating organ-specific dioxin toxic responses (Bock and Köhle 2006).

One of the major functions of the AhR could be the elicitation of cellular stress responses (reviewed in Matsumura 2003). Toxicity is linked to the occupancy of the AhR by dioxins, but occupancy is not solely responsible for potential carcinogenicity (reviewed in Cole et al. 2003). Furans can also induce changes in the expression of various genes associated with oxidative stress, DNA damage and cell cycle control in rat liver cells (Hickling et al. 2010). Furans induce oxidative stress and DNA damage via oxygen radicals in these rat liver cells (Hickling et al. 2010).

Dioxins and furans induce enzymes involved in drug and xenobiotic metabolism, including phase I biotransformation enzymes, such as the cytochromes P450. In particular, members of the CYP1A family and CYP1B1 are induced (Schechter et al. 2003). Several phase II conjugation enzymes, including glucuronyl transferases, glutathione transferases and aldehyde

dehydrogenases are also induced by dioxin. Other proteins involved in DNA synthesis and transcriptional control may also be induced by dioxin (Schechter et al. 2003).

Furans are rapidly metabolized by cytochrome P450, and the resultant metabolites are linked to toxicity and carcinogenicity (Bakhiya and Appel 2010; Peterson et al. 2005). These furan metabolites react readily with protein and DNA nucleophiles and has been shown to be a bacterial mutagen (Peterson 2006). Protein alkylation by a furan metabolite is thought to trigger the cytotoxic effects of furan, and the consequences of these cytotoxic effects give rise to the carcinogenic properties of furan (Peterson 2006). This includes induction of cell proliferation in rodent livers and the lack of induction of DNA repair responses in the rat liver (Peterson 2006).

One of the typical stresses of dioxin poisoning is oxidative stress caused by cytochrome P450 induction (Matsumura 2003). One of the major means by which dioxin triggers the stress response in cells is via "stress-activated kinase pathways". These pathways stimulate cellular production of cytokines and autocrines, particularly growth factors (Matsumura 2003). The multiple growth factor systems affected by dioxins include the retinoid signalling systems (Schechter et al. 2003; Nilsson and Håkansson 2002) and multiple cytokines (Pande et al. 2005; Schechter et al. 2003). Interleukin 1 (IL1)-like cytokines may provide protection from dioxin-induced liver inflammation (Pande et al. 2005) and liver cell apoptosis (Pande et al. 2005). However, interleukins do not appear to protect against damage to the thymus, induction of enzymes CYP1a2, or enlarged liver (Pande et al. 2005). Furans are also shown to affect cytokine expression. Furan toxicity increases the expression of cytokines and other inflammation-associated genes, such as IL-6 and IL-10 (Hamadeh et al. 2004; Hickling et al. 2010; reviewed in Moro et al. 2012).

Dioxins also affect the retinoid signalling systems, which are involved in patterning and specification of cells in fetal development, and support normal growth, vision and maintenance of numerous tissues (Nilsson and Håkansson 2002). Dioxins decrease retinoic levels in liver cells (Fletcher et al 2001) and extrahepatic tissues (Nilsson and Håkansson 2002) in rodent studies. Decreased retinoid levels may lead to impaired growth, reduced reproduction, developmental abnormalities, impaired immune function and lesions of epithelial linings (Nilsson and Håkansson 2002). Attempts to correct retinoid levels with dietary vitamin A intake only provided limited protection from toxicity (Nilsson and Håkansson 2002).

9.2 Endocrine effects

Dioxins are also potent endocrine disrupters, resulting in alterations in both protein and steroid hormone systems (Schechter et al. 2003). There are three possible routes of action: at the hormone receptor itself, during synthesis or breakdown of the hormone, or during transport of the hormone via the blood (Schechter et al. 2003).

Dioxins also interfere with the estrogen signalling pathway. In a study of human ovarian carcinoma BG-1 cells, Rogers and Denison (2002) determined that a dioxin-induced protein interferes with signalling at the step after the estrogen receptor complex binds to the DNA, most likely at the level of gene-transcription (Rogers and Denison 2002).

Results are inconsistent regarding changes in reproductive hormone levels in humans due to dioxins and furans, but include decreased testosterone and increased gonadotrophin concentrations (Kogevinas 2001). In human luteinized granulosa cells (hLGC), dioxins inhibit expression of the P450c17, and decrease in 17,20-lyase activity, both of which are steroidogenic enzymes (Morán et al. 2003). The decreases in P450c17 and 17,20-lyase were

proportional to the inhibition of estradiol secretion (Morán et al 2003), suggesting that this may explain the decrease in reproductive hormone levels in exposed humans.

Thyroid hormones and thyroid binding globulin have both demonstrated a positive relationship with dioxin concentrations in the blood (Kogevinas 2001). However, a more recent study demonstrates that total thyroxine (T4) levels decrease with exposure to dioxin-like toxic equivalents (TEQs) similar to those found in the general US population (Turyk et al. 2007). These effects were also stronger in women, suggesting that older adults with a high risk of thyroid disease may be at higher risk for changes in thyroid hormone levels by dioxin-like organochlorines than younger adults (Turyk et al. 2007). Thyroid-stimulating hormone was also found to be elevated in neonates born from mothers with elevated dioxin levels in plasma, even 30 years after exposure (Baccarelli et al. 2008).

9.3 Reproductive effects

A variety of reproductive effects of dioxins and furans have been characterized in humans. In specific cohorts, excessive risks were observed for reproductive cancers, including breast female, endometrium, breast male and testis cancers (Kogevinas 2001).

Dioxin exposure during infancy in males in the residents of Seveso, Italy was linked to decreased sperm count and decreased sperm mobility at adulthood (Mocarelli et al. 2008). However, exposure during puberty led to increased total sperm count and increased total motile sperm count (Mocarelli et al. 2008). Exposure during infancy and puberty both were linked to decreased estradiol and increased follicular stimulating hormone levels (Mocarelli et al. 2008).

Additionally, there is some evidence that there is a change in sex ratio at birth with an excess of females over males in the population in Seveso exposed to dioxins (Mocarelli et al. 2001).

9.4 Developmental effects

Several developmental effects of dioxins and furans have been characterized in humans. Firstly, there is some evidence that pre-natal exposure to dioxins may result in impaired motor, but not mental, development in 6-month old Japanese infants (Nakajima et al. 2006). Potential neurotoxic effects of dioxin exposure may persist into childhood, resulting in subtle cognitive and motor developmental delays, but these effects may be counteracted by advantageous parental and home environments (Vreugdenhil et al. 2002).

There is some evidence that dioxin exposure may result in cleft palate, due to expression of cytokines at an inappropriate time (Thomae et al. 2005; reviewed in Bock and Köhle, 2006). Addition of cytokine TGF β 3 to dioxin-exposed organ cultures restored fusion of palatal shelves, preventing cleft palate (Thomae et al. 2005).

9.5 Other effects

Dioxin exposure is also linked to chloracne, which is characterized as a hyperkeratotic skin disorder that affects the hair follicles, sebaceous glands and interfollicular epidermis (Bock and Köhle 2006). These effects were consistently observed in exposed humans since 1957 when dioxins were first linked to chloracne, due to sustained and inappropriate AhR activation by dioxin (Bock and Köhle 2006).

Dioxin toxicity has also been linked to immunosuppression, as B and T cell responses as well as host resistance to bacterial and viral infection are affected by dioxin exposure (Bock and

Köhle 2006). This is not surprising, as previously mentioned, dioxin toxicity results in involution of the thymus.

Oral exposure to furan (via food consumption) has been shown to cause liver cell death, liver inflammation and elevated liver enzyme activities within 24 h in rodents (Moro et al. 2012). Two weeks of oral exposure to furan in rodents resulted in decreased body weights, increased liver weights and significant increases in serum transaminases, alkaline phosphatases, cholesterol, triglycerides and total bilirubin (Hamadeh et al. 2004). Furthermore, blood urea nitrogen and serum creatinine were also increased, indicative of impaired hepatic and renal function (Hamadeh et al. 2004).

Dioxins can be stored in adipose tissue in both infants and adults (Thoma et al. 1990). The levels were generally lower in infants than adults (Thoma et al. 1990). Furan induces liver cell necrosis and marked increase in plasma liver enzymes following oral administration in mice (Wilson et al. 1992). This is followed by an increase in compensatory cell proliferation 48 h after treatment (Wilson et al. 1992).

Dioxins may also shift the sensitivity of adults to type-II diabetes. In the early 1990s, a decrease in glucose tolerance was seen in one cohort (Sweeney et al. 1997), and an increase in diabetes was seen in Vietnam veterans serving in areas sprayed with Agent Orange (Michalek et al. 1999; Longnecker and Michalek 2000). An increase in diabetes was also seen in the Italian population exposed to dioxin (Bertazzi et al. 2001). It is thought that dioxins shifts the distribution of sensitivity, putting people at risk at younger ages or with less weight, by altering lipid metabolism (Sweeney et al. 1997).

9.6 Longterm effects of exposure and cancer

Fifteen years after exposure to dioxins in Sevesco, Italy, the mortality amongst men due to all cancers combined has increased (Bertazzi et al. 2001). Mortality also increased in men due to rectal cancer and lung cancers alone, respectively (Bertazzi et al. 2001). There was also an excess of lymphohemopoietic neoplasms in both genders, and increase in Hodgkins disease risk in the first ten years following exposure (Bertazzi et al. 2001). The highest increase for non-Hodgkin's lymphoma and myeloid leukemia occurred 15 years after exposure (Bertazzi et al. 2001). However, Cole et al (2003) argue that this long-term study was case-controlled and has not been replicated since.

Male chemical production workers in a long-term American study were examined 10 years after exposure to substantial levels of dioxins. There was no increased risk of mortality, and the incidence of all cancers combined and lung cancer were at or below expected levels (Bodner et al. 2003). Rates for soft tissue sarcoma and non-Hodgkins lymphoma were greater than predicted, but below expectation in the update period (Bodner et al. 2003). There was also no trend of increasing risk with increasing exposure for these cancers (Bodner et al, 2003).

Effects of longterm exposure to dioxin were also assessed in Vietnam War veterans that served in areas where dioxin-contaminated herbicides were sprayed. These veterans were compared with those soldiers that had served in areas of Southeast Asia that were not sprayed during the same time. The incidence of melanoma and prostate cancer were increased among veterans who served in sprayed areas relative to those who served in unsprayed areas (Akhtar et al. 2004). These results are consistent with the link between cancer and dioxin exposure.

Further, exposure to dioxins increased the risk for breast cancer in both women and men, endometrial cancer and testicular cancer (Kogevinas et al. 1997). There was an increase in mortality in breast cancer in women in a cohort in Germany (Kogevinas et al. 1997).

Oral consumption of furan increased the incidences of liver cancers in rodents shortly after exposure (Moro et al. 2012). However, 2 years after furan exposure, there were higher incidences of bile duct cancers in rodents (Moro et al. 2012). Furan has the potential to be a human genotoxic carcinogen based on previous rodent studies. Furan induces liver tumors in both adult and infant mice (Fransson-Steen et al. 1997; Johansson et al. 1997). Oral administration of furan leads to cancer of the bile duct in rodents at higher doses (30 mg/kg bwt and more) (Elmore and Sirica 1991).

10.0 EFFECTS OF POLYCYCLIC AROMATIC HYDROCARBONS (PAH) ON MARINE LIFE

Polycyclic aromatic hydrocarbons (PAHs) constitute a group of organic pollutants formed through natural and industrial processes that are ubiquitous in the environment (Neff 1979; Varanasi 1989). They enter the aquatic ecosystem through atmospheric deposition, surface runoff, effluent discharge, and oil spills, and can persist in the environment for long periods of time. In aquatic ecosystems, hydrophobic PAH are relatively insoluble in water and are mainly found associated with particulate matter.

The toxicity of PAHs to aquatic organisms is determined by several factors which include: (a) the PAH type (e.g., molecular weight, alkyl substitution, etc.), (b) the species of the organism exposed, and (c) the duration and the type of exposure to a given PAH. In general, fish appear to be the most sensitive of the aquatic organisms to PAHs. Most of the literature on acute and lethal toxic effects in estuarine and marine environments is related to the lower molecular weight PAH (LPAH), containing 2-3 benzene rings in their structure. These compounds are relatively more soluble in water than the higher molecular weight PAHs (HPAH); at saturation, their concentrations in water can exceed LC_{50} values, unlike the HPAH compounds (e.g. benz[a]anthracene and benzo[a]pyrene) which have limited water solubility. In addition, alkyl homologues of PAHs are generally more toxic to aquatic life than the parent compound.

10.1 Invertebrates

10.1.1 Immunotoxicity

In an experiment designed to identify individual effects of several PAH compounds (naphthalene, pyrene, and benzo(a)pyrene) on hemocyte viability and phagocytic activity in the eastern oyster (*Crassostrea virginica*), it was shown that the most-toxic compound, benzo(a)pyrene, at the highest concentration, stimulated an increase in agranular hemocyte counts. A follow-up experiment examining the effects of benzo(a)pyrene on hemocyte viability, adhesion and phagocytosis showed that the ability of this benthic diatom to transport PAHs to the eastern oyster causes immunomodulation (Croxtton et al. (2012). Bivalve molluscs (*Cerastoderma edule* and *Ensis siliqua*) exposed to a range of phenanthrene concentrations showed a significant reduction in phagocytic haemocytes 14 d following exposure to >100 µg L⁻¹ phenanthrene (Wootton et al. 2003).

The study by looked at Lysosomal destabilization (range from 34 to 81%) in the hemocytes of eastern oysters (*C. virginica*) collected along a chemical concentration gradient in Galveston Bay, Texas, USA was found and compared to concentrations of organic compounds. A significant positive correlation was observed between lysosomal destabilization and body burden of PAHs and other organics, while no significant correlation was found with metal

concentrations (Hwang et al. 2002). The immunotoxic effects of PAHs were investigated in marine mussels (*Mytilus edulis*) by means of in vivo exposure to a PAH cocktail of anthracene, fluoranthene and phenanthrene. PAHs were found to inhibit phagocytosis and damage lysosomes Grundy et al. (1996a). These results were confirmed (Grundy et al. 1996b) these results, as PAHs were also found to inhibit phagocytosis and disrupt the ability of lysosomes to take up or retain neutral red dye, suggesting that membrane permeability was affected. Many other studies have demonstrated that exposure to PAH affected the immune system of marine invertebrate species (Hannam et al. 2010; Auffret et al. 2004; Mayrand et al. 2005).

10.1.2 Genotoxicity

Genotoxic end-points are routinely measured in various sentinel organisms in aquatic environments in order to monitor the impact of water pollution on organisms (Michel and Vincent-Hubert 2012). Two of the more sensitive, reliable and simple techniques to examine DNA damage are comet assay and the micronucleus assay (MN test). In Siu et al. (2004), green-lipped mussels (*Perna viridis*) exposed to water-borne B[a]P indicated that an increase in the proportion of strand breaks occurred generally with increasing B[a]P concentration. Seabob shrimp (*Xiphopenaeus kroyeri*) exposed for 96 h to B[a]P revealed that DNA damage significantly increased as a function of B[a]P exposure concentrations (Silva Rocha et al. 2012). The Comet assay and MN test were carried out in Binelli et al. (2008) using zebra mussel (*Dreissena polymorpha*) to evaluate the potential genotoxicity of ultra low B[a]P concentrations (0.1-10 µg/L). A clear genotoxic effect on zebra mussel hemocytes in the presence of all B[a]P exposure concentrations was found. A similar genotoxic effect of B[a]P exposure on zebra mussel (*D. polymorpha*) was also found (Michel et al. 2012).

10.1.3 Oxidative stress

During the detoxification process, PAHs or their metabolites can undergo biotransformation reactions within lysosomes and penetrate membranes, which in turn can lead to alterations in lysosomal integrity, as well as membrane fluidity and ionic pumps (Baussant et al. 2009; Hannam et al. 2009a). Moreover, by-products of their metabolism, such as diol epoxides, radical cations and redox active o-quinones are characterised by high redox potential, thus leading to the induction of severe oxidative damage (Penning et al. 1996; Livingstone 2003), probably through the generation of reactive oxygen species (ROS). Many studies have reported increased levels of ROS or other indicators of oxidative stress upon exposure to various PAH; for example in the traditional aquatic invertebrate ecotoxicological model (*Daphnia magna*) (Feldmannová et al. 2006), the temperate scallop (*Pecten maximus*) (Hannam et al. 2010), Mediterranean mussels (*Mytilus galloprovincialis*) (Giannapas et al. 2011), and the shell clam, (*Mya arenaria*) (Frouin et al. 2007).

10.1.4 Reproductive toxicity

In Feldmannová et al. (2006), all tested N-PAHs were found to significantly suppress reproduction of *Daphnia magna* following a 21-day exposure. Frouin et al. (2007) demonstrated that a significant delay in gametogenesis occurred in all exposed shell clam (*M. arenaria*) males and in females contaminated with dietary PAHs. Furthermore, these researchers observed that males were more sensitive to PAHs than females. Mazurová et al. (2008) highlighted that Lake Pilnok sediment that was highly contaminated with powdered waste coal affected the fecundity of the Prosobranchian euryhaline mud snail, *Potamopyrgus antipodarum*. In Sese et al. (2009), it was reported that acenaphthene, phenanthrene, anthracene, fluoranthene, pyrene, and B[a]P were reproductively toxic to *Caenorhabditis elegans*. Results showed that reproduction, in

addition to growth, were much more sensitive parameters of adverse response than lethality, and consequently may be more useful in assessing PAH toxicity using *C. elegans*.

10.1.5 Phototoxicity

There is a growing body of evidence to suggest that certain polycyclic aromatic hydrocarbons (PAHs) pose a greater hazard to aquatic organisms than previously demonstrated, due to their potential to cause photo-induced toxicity when exposed to ultraviolet (UV) radiation. This toxicity may occur through two mechanisms: photosensitization and photomodification. Photosensitization generally leads to the production of singlet oxygen, a reactive oxygen species that is highly damaging to biological molecules. Photomodification of PAHs, usually via oxidation, results in the formation of new compounds and can occur under environmentally relevant levels of actinic radiation (electromagnetic radiation that can produce photochemical reactions).

For example, Lampi et al. (2006) examining the toxicities of 16 PAHs to *D. magna* were under the presence and absence of full-spectrum simulated solar radiation. Showed the importance of the role of photomodification since several oxy-PAHs were found to be highly toxic to *D. magna*. Another study also found that photosensitization of bioaccumulated PAHs, namely anthracene and pyrene, appeared to be the primary mechanism for acute photoinduced toxicity in *D. magna* (Huovinen et al. 2001). Sublethal effects, such as reduced feeding efficiency due to fluoranthene phototoxicity, have been shown in *D. magna* (Hatch and Burton, 1999). The consequences of photo-induced toxicity were reported for embryo-larval stages of the pacific oyster, *Crassostrea gigas*, following exposure to pyrene and B[a]P (Lyons et al. 2002). Significant increases in toxicity were observed in the presence of environmentally attainable levels of ultraviolet-radiation, compared with embryos exposed to PAH alone, at levels previously deemed to have little acute biological effect.

Photoactivation of PAHs bioaccumulated in the blackworm, *Lumbriculus variegatus*, and freshwater amphipod, *Hyaella Azteca*, from contaminated field sediments can cause increased mortality (Ankley et al. 1994; Monson et al. 1995). UV exposure can increase the toxicity of PAH-contaminated sediments to the infaunal amphipods, *Rhepoxynius abronius* and *Leptocheirus plumulosus*, decreasing survival and ability to rebury (Boese et al. 2000). Exposure via water to fluoranthene and subsequently to UV radiation demonstrated increased mortality in *L. variegatus* as a function of both PAH dose in tissue and UV intensity (Ankley et al. 1995). The exposure to PAHs and UV radiation resulted in mortality of marine crab larvae (Peachey 2005). Increased mortality due to phototoxicity of fluoranthene has been demonstrated in glochidal larvae of the freshwater paper pondshell, *Utterbackia imbecillis*, exposed to waterborne PAH (Weinstein 2001) and in embryos of the marine dwarf surf clam, *Mulinia lateralis*, with body burden of PAH through maternal transfer from benthic adults (Pelletier et al. 2000).

10.2 Salmonids

10.2.1 Acute toxicity

Most HPAH are not acutely toxic at concentrations that reflect their water solubilities. However, as mentioned, 2- and 3-ringed PAH do have acute toxicity at concentrations that would be encountered in water.

10.2.2 Biochemical indicators

PAHs are known to interact with the Aryl Hydrocarbon Receptor (AhR), which is a nuclear transcription factor present in most species examined, including fish. Interaction of PAH (or other ligands such as dioxin) with the AhR leads to transcription of various genes that contain a dioxin- or xenobiotic-response element (DRE or XRE). Some of these genes are important for developmental processes or adaptive responses to xenobiotic exposure such as biotransformation (Beishlag et al. 2008; Zhou et al. 2010).

Cytochrome P450s (CYP) are enzymes responsible for Phase I metabolism reactions in fish (and other species), which add functional groups to both endogenous and exogenous (xenobiotic) compounds to increase their polarity and allow these chemicals to be excreted more readily. CYP1A1 is one of these enzymes that is induced following AhR interaction with DRE in the gene. Since CYP1A1 activity, which can be measured using the ethoxyresorufin-O-deethylation (EROD) assay, is induced following exposure to AhR ligands (such as PAHs), it is frequently used as a biomarker of exposure to these chemicals in the environment.

Indeed, there have been a number of studies that have looked at CYP1A1 activity in either gill or liver tissue following collection of wild Pacific salmon from PAH-contaminated aquatic environments (field studies), following caging of salmonids in potentially PAH-contaminated areas (*in situ* studies) in the Pacific Northwest, or in laboratory studies using contaminated sediments. Gill or liver EROD activity was induced in these fish, even with low levels of dissolved (aquatic) or sediment-associated PAHs (Stehr 2000; Fragoso 2006; Blanc et al. 2010; Bravo et al. 2011).

Carls et al. (2005) demonstrated in their study and meta-analysis with pink salmon that CYP1A1 induction can be used as a good biomarker for predicting other sublethal effects of PAHs, including poor marine survival, reduced growth and developmental abnormalities.

10.2.3 Growth impairments and somatic indicators of toxicity

Moles et al. (1981) conducted a study to assess the impact of toluene (MAH) and naphthalene (PAH) exposure in freshwater on coho salmon growth. They report that dry weights, wet weights and lengths of fry exposed to naphthalene at concentrations of 3.2 µL/L or more for toluene or 0.7 mg/L or more for naphthalene is decreased, as is daily growth rate.

Meador et al. (2006) conducted a 56 day study in which juvenile Chinook salmon were fed a diet contaminated with a mixture of both low and high molecular weight PAHs that was intended to mimic the types and concentrations of these PAHs that the fish would encounter in the natural aquatic environment. The feeding of contaminated food was intended to mimic contaminated prey items that the fish would consume and was based on a previous study examining PAH levels in stomach contents of field-collected fish (Varanasi et al. 1993). They found that fish had accumulated PAHs in their tissues to a limited degree (concentrations in tissue were lower than in food) and that fish were able to metabolize the PAHs for excretion through the bile. Exposure to dietary PAHs also led to decreased growth of fish (both wet and dry weight measurements), decreased the overall whole-body lipid content, influenced the distribution of different lipid classes (specifically decreasing the triacylglycerol (TAG) content) and altered various plasma chemistry parameters (e.g. albumin, amylase, cholesterol, creatinine, glucose, lipase). Overall this study demonstrated the detrimental effects of PAH on growth and metabolism, which the authors termed “toxicant-induced starvation”, since the effects were similar as what would be observed in starved fish.

10.2.4 Immunotoxicity

Field-collected salmonids (Chinook) that spend time in contaminated estuaries have been shown to be more susceptible to infectious diseases caused by bacterial disease such as *Listonella anguillarum* (Arkoosh et al. 1998). However, one of the difficulties in interpretation of field studies is that contaminants and other factors do not occur individually or in isolation, so it can be difficult to assign causation to particular chemicals or other factors.

Bravo et al. (2011) had similar findings of increased mortality following disease challenge (with *Aeromonas salmonicida*) in the lab following feeding of juvenile rainbow trout with food contaminated with predominantly HPAHs. In contrast, Palm et al. (2003) found that Chinook salmon fed a diet contaminated containing both LPAH and HPAH had no changes in disease susceptibility (*Listonella anguillarum*) relative to control groups. The study done by Bravo et al. (2011) was double the duration (50 d) compared to the Palm et al. (2003) study (28 d), and effects on disease susceptibility were not apparent until day 50 of exposure, suggesting that both duration of exposure and type of PAHs may affect the overall immunotoxicity.

10.2.5 Genotoxicity

An additional biomarker of PAH exposure that may be useful in field settings is based on the measurement of genomic damage. Once metabolized in the liver, some PAHs, particularly those with higher molecular weight (4 or more rings), can form reactive intermediates or metabolites. These metabolites can react with DNA to form DNA adducts which can lead to DNA strand breaks, mutations and ultimately cancer or tumors if not repaired. Measurement of the various types of DNA damage (strand breaks or fragmentation, DNA content changes, and micronuclei) can be a biomarker for PAH exposure, uptake and metabolism.

For example, Barbee et al. (2008) demonstrated that juvenile coho salmon, caged *in situ* in an area with sediment PAH contamination, had higher chromosomal damage in both peripheral blood and liver measured using several different assay methods. The level of damage correlated with the sediment PAH concentration, but not the aquatic PAH concentration. This is consistent with the preferential partitioning of higher molecular weight PAHs to the sediment, which are more typically associated with genomic damage.

10.2.6 Reproductive toxicity

No studies could be located on the effects of PAHs as a class on reproductive endpoints in salmonids. The effects on non-salmonid cold water fish are summarized in the next subsection.

10.2.7 Developmental toxicity

Causation of developmental effects have not yet been attributed to PAH directly in many studies, as most PAH sources in studies have been with mixtures. However, studies that examine the toxicity of mixtures of hydrocarbons such as PAH have been invaluable in assessing which component(s) of mixtures are causing the observed effects. For example, an experiment done by Sundberg et al. (2006) used special separation columns to investigate the effects of 3 different sediment-derived crude oil fractions (aliphatic hydrocarbons/MAHs, dicyclic aromatic hydrocarbons and PAHs) on fertilized egg development and larval deformities in rainbow trout. Exposure (through micro-injections, to mimic maternal transfer and environmental uptake in the egg) to the PAH fraction led to increased mortality in the developing eggs and elevation of deformities such as assymetrical yolk-sacs in embryos and haemorrhaging in larvae. This suggests that PAHs, and particularly high-molecular weight PAHs, can contribute to embryotoxicity.

Billiard et al. (1999) demonstrated that exposure of rainbow trout to retene (32 – 320 µg/L) during egg and post-hatch stages resulted in increased incidence of blue-sac disease. Some symptoms were observed at the lowest concentration tested and effects included induction of CYP1A1, edema, haemorrhaging, craniofacial malformation as well as reduced growth, mortality, fin erosion and opercular sloughing.

In a followup study to the Billiard et al. (1999) investigation, a study with rainbow trout was done to evaluate retene (320 µg/L) for its ability to produce blue-sac disease through an oxidative stress mechanism (Bauder et al. 2005). Retene-exposed fish had increased prevalence of blue-sac disease and decreased Vitamin E and glutathione concentrations in the tissues. Co-exposures with retene and Vitamin E resulted in reduced incidence of blue-sac disease and increased tissue concentrations of Vitamine E, but did not affect glutathione concentrations. The authors concluded that a portion of the effects of retene are related to oxidative stress, but there may be additional mechanisms of toxicity such as formation of retene adducts in DNA, lipids or protein.

The minimum concentrations of lower molecular weight PAHs: naphthalene, acridine, and phenanthrene, causing gross developmental anomalies in rainbow trout, were found to be much higher than B[a]P (Black et al. 1983).

Teratogenic effects during organogenesis (7- to 24-d post fertilization) were studied by Hannah et al. (1982) and Hose et al. (1984) in rainbow trout (*Oncorhynchus mykiss*) exposed to B[a]P-contaminated sand. Gross anomalies (e.g., microphthalmia) were noted in a significant population of fish exposed to the contaminated sand (Hose et al., 1984).

10.2.8 Neurotoxicity

In a series of studies, Gesto and colleagues demonstrated that naphthalene is neurotoxic to rainbow trout. Gesto et al. (2009) showed that both naphthalene and benzo(a)pyrene, following intraperitoneal injections, can disrupt the functioning of the pineal gland, altering the release of melatonin and other hormones responsible for regulation of biological rhythms. Additionally, exposure to naphthalene for up to 5 days (via injection or implants) altered the levels of the monoaminergic neurotransmitters (dopamine, serotonin, noradrenalin) and metabolites in the brain of immature rainbow trout (Gesto et al. 2006). An earlier study had demonstrated that naphthalene also decreases plasma cortisol and other plasma analytes in rainbow trout (Gesto et al. 2008). Taken together, these studies suggest that naphthalene (and potentially other PAHs) may modify neuroendocrine interactions, which may have widespread physiological implications for fish.

10.2.9 Behavioural toxicity

Purdy (1989) conducted a study using a 24-h aquatic exposure to a mixture of low and high molecular weight PAHs in coho salmon. Effects on feeding and avoidance behaviour were evaluated at the end of the 24-h exposure period and periodically following recovery from that exposure. It was found that exposure to the mixture of PAHs resulted in impaired feeding behaviour, as well as loss of a learned avoidance response. In addition, the time taken for fish to respond in the avoidance assay increased, indicating reaction times were slowed. These effects persisted for 1 to 10 days after the exposure was withdrawn.

10.3 Other Fish

10.3.1 Biochemical or somatic indicators of toxicity

A 4-month study was done of winter flounder using sediments contaminated with petroleum-derived PAHs, containing a mix of low and high molecular weight PAHs (Payne et al. 1988). These fish live in close contact with sediments in the aquatic environment and so could be at risk from PAHs that might partition to the sediments. The authors found that general health indices, such as liver somatic index and spleen somatic index were altered (increased and decreased, respectively). Muscle protein content decreased, while glycogen stores in the liver increased. EROD activity, a measure of CYP1A1 induction, and liver lipids were elevated in livers of exposed fish. Some effects on fish were notable at total PAH concentrations as low as 1 µg/g in the sediment, which would be expected to occur in a variety of natural aquatic environments that are impacted by anthropogenic activities.

10.3.2 Histopathological

The most sensitive chronic effects of naphthalene were observed by DiMichele and Taylor (1978) while studying histopathological and physiological responses in mummichog (*Fundulus heteroclitus*). These investigators found gill hyperplasia in 80% of the fish after a 15-d exposure to 2 µg/L naphthalene; only 30% of the control fish showed the effect. All of the fish exposed to 20 µg/L demonstrated necrosis of tastebuds, a change not observed in the control fish.

10.3.3 Immunotoxicity

A review of the immunotoxicity of PAH in fish is available, which summarizes the diverse impacts on both the innate and adaptive immune system (Reynaud and Deschaux, 2006). Effects on enzyme and cellular functions may vary depending on species, chemical(s) used in exposures, and method of exposure; and some studies have found that susceptibility to pathogens is increased following PAH exposure.

10.3.4 Genotoxicity

A study by Wessel et al. (2010) looked at the relationship in juvenile sole between various biomarkers commonly used to measure PAHs exposure in the environment: bioavailability (CYP1A1 activity/ EROD), biotransformation (bile PAH metabolites) and genotoxicity (DNA strand breaks). Fish were given food containing a mixture of 3 high molecular weight PAHs (>4 rings; benzo[a]pyrene, fluoranthene and pyrene), followed by 1 week of depuration. The production of metabolites confirmed the biotransformation of PAHs. EROD activity was slightly elevated, while formation of DNA strand breaks was significantly elevated in PAH-exposed fish. There was good correlation between the concentration of metabolites in bile and the formation of DNA strand breaks (genotoxicity), but not for EROD. This study highlights the importance of using multiple biomarkers when assessing PAH exposure, and the correlation between bile metabolites and DNA damage further strengthens the hypothesis that metabolism of PAHs is required in order to generate reactive intermediates that can form DNA adducts.

10.3.5 Reproductive toxicity

Hall et al. (1991) conducted a study using adult fathead minnows to assess the effects of anthracene (3 ring PAH) exposure on reproduction and larval/fry survivability. Anthracene is one of the PAHs that are associated with potential for phototoxicity or photosensitization. Adult fish were exposed to anthracene for up to 6 weeks. Eggs that were laid were collected and transferred to fresh, clean (no anthracene) water. Some of the eggs were exposed to solar ultraviolet radiation, while others were not. Eggs were monitored for hatchability, survivorship and abnormalities/deformities. The authors report that anthracene bioconcentrated in eggs, gonads and carcasses of exposed fish. Fewer eggs were laid by anthracene-exposed fish. Egg

survivorship was impaired in all groups and and teratogenicity (larval/fry deformities) was increased by maternal anthracene exposure when eggs were subsequently exposed to UV radiation. This study demonstrates the potential for maternal transfer of contaminant effects, indicating that PAH exposure does not need to occur to the eggs themselves or during developmental stages of the egg, larvae or fry before detrimental effects are observed.

HPAH can also be chronically toxic to sand sole (*P. melanostichus*) (Hose et al. 1982). These investigators observed that the average hatching success in sand sole exposed to 0.10 µg/L B[a]P was reduced by about 29% compared to the control.

10.3.6 Developmental toxicity

The zebrafish (warm water species) is commonly used as a model organism for assessing developmental consequences of many chemicals, since the zebrafish are small, readily grow and reproduce in the lab setting, and have a short lifecycle allowing full lifecycle assessments. Using zebrafish, Incardona et al. (2004) demonstrated that different PAHs cause different effects in developing embryos. In this experiment, embryos were exposed to 7 different individual PAHs (naphthalene, fluorene, dibenzothiophene, phenanthrene, anthracene, pyrene or chrysene), or 2 mixtures of those PAHs. All except pyrene and anthracene are commonly found components of Alaskan North Slope crude oil. Exposures to dibenzothiophene or phenanthrene alone were enough to cause the PAH-associated blue-sac disease previously described. The relative toxicity of the mixtures was proportional to either the amount of phenanthrene or dibenzothiophene+phenanthrene present in the mixture. Pyrene, a higher molecular weight PAH (4 rings) was found to cause a different suite of symptoms which included anemia, peripheral vascular defects and neuronal cell death. The change in effects between the lower molecular weight PAHs and the high molecular weight PAHs has implications for oil-contaminated aquatic environments, since crude oil will typically undergo a weathering process in which the lower molecular weight PAHs are lost, leaving the higher molecular weight compounds behind. This shift in PAH composition may have consequences for fish embryos in terms of the types of effects that might be observed.

Also using zebrafish embryos, Carls et al. (2008) investigated the effects of either dissolved PAHs only, or total PAHs (dissolved plus oil droplets) derived from Alaskan North Slope crude oil. Embryos were assessed for physiological effects including pericardial edema, abnormal heart development and intracranial haemorrhaging following 2 days of exposure and all parameters were altered by exposures. The authors found that the effects of total PAHs (including oil droplets) were not different than the effects of dissolved PAHs (no oil droplets) on any of the physiological alterations assessed, indicating that it the embryos do not need to come into direct contact with oil droplets in order for toxicological consequences to occur.

Japanese medaka is a warm water fish species that are also a commonly used fish model in toxicological studies. Fallahtaffi et al. (2011) used this species to examine the effects of a series of alkyl-phenanthrenes and their hydroxylated metabolites on the development and severity of blue-sac disease (edema, heart defects, deformities, haemorrhaging) in early life stages following a 17 d exposure period. Generally, the metabolites of the alkyl-phenanthrenes were found to cause more severe blue-sac disease symptoms, confirming the importance of metabolism in the generation of more toxic PAH intermediates.

Among PAHs studied, B[a]P is typically found to be the most toxic. For example, 5% of sand sole (*Psettichthys melanostichus*) eggs exposed to B[a]P at 0.1 µg/L in water showed gross anomalies such as overgrowth of tissue originating from the somatic musculature, and arrested

development (as compared to 0% in control fish) (Hose et al., 1982). Also, the hatching success of eggs exposed to 0.1 µg B[a]P/L was significantly lower than that of controls.

10.3.7 Behavioural toxicity

Goncalves et al. (2008) carried out a study using gilthead seabream (warm water fish species) to examine the effects of 3 PAHs (fluorene, pyrene and phenanthracene) individually and in mixtures. Each of the PAHs impaired swim performance and increased lethargy. Phenanthracene exposure also impaired social performance of the fish. Pyrene was the most potent and measurement of lethargy was the most sensitive endpoint. The mixture of the 3 PAHs had similar effects and the chemicals in mixture appears to be additive in their effects.

Farr et al. (1995) used fathead minnow to demonstrate that fish are able to detect, respond to and avoid plumes of fluoranthene at high concentrations, but not at lower concentrations. Thus fish might be unable to detect fluoranthene at environmentally relevant concentrations, resulting in further exposure of the fish to an environment where toxic effects may occur.

11.0 SUMMARY AND CONCLUSIONS

The Environmental Impact Statement and Environmental Assessment of Certificate Application by Pacific Northwest LNG Limited Partnership Ltd (PNW LNG) specifically addresses the dredging and disposal of approximately 8 million m³ of material from two sites: the proposed site of the Materials Offloading Facility (MOF) in Porpoise Channel to the north of Lelu Island, and the proposed marine berth dredge area located approximately 2 km southwest of Lelu Island. The probable loading site of the dredgate is in Brown Passage. This document was reviewed and the interpretations on the potential for effects in marine biota and humans were assessed.

- The assumption that As and Cu are background contamination and should not be considered COPCs is likely, however, a strict sampling program needs to be implemented to establish that the concentrations of these metals are background and natural;
- PAH, PCDDs and PCDFs are COPCs in the proposed operation areas;
- The mean concentration of PCDD/F in sediments in the most contaminated area near the proposed MOF is highest in the 0-0.5 m depth, and is as high as 2.64 ng/kg TEQ PCDD/F;
- The concentrations of PCDD/Fs in several cases are between the ISQG and the PEL, indicating that there is a high potential for effects in marine organisms. The assumption in the document that no adverse effects will occur is not warranted;
- Movement of contaminated sediment by dredging will uncover and distribute contaminated sediments and may increase concentrations near the sediment surface in the dredging and loading areas;
- The bioavailability of PAH and PCDD/Fs will increase with the dredging and loading of contaminated sediments to the proposed loading site;
- Many dioxin and furan congeners are bioaccumulative and will bioconcentrate and biomagnify in the food web;
- The potential effects of PCDD/F exposure may occur in several ecological receptors (e.g. microorganisms, algae, zooplankton, benthic invertebrates, invertebrates, fish, mammals and birds), as well as in humans;

- The potential effects of PAH exposure may occur in several ecological receptors closely associated with contaminated sediments (e.g. microorganisms, benthic invertebrates, benthic fish);
- Effects of PCDD/Fs in ecological receptors may include changes in biochemistry, developmental and reproductive effects, endocrine disruption, immunotoxicity, and histopathology;
- Sensitive habitats including Flora Bank and dependent organisms (e.g. juvenile salmonids) are at particular risk from the proposed dredging operation;
- Effects of PAHs in ecological receptors may include changes in biochemistry, developmental and reproductive effects, immunotoxicity, and behavioral effects;
- Humans may consume contaminated marine organisms in which bioconcentration and/or biomagnification of dioxins and furans have occurred;
- Potential effects in humans from dioxin and furan exposure include biochemical alterations, developmental toxicity, reproductive effects, endocrine disruption, chloracne, and potentially cancer.
- In order to mitigate the potential effects of contaminated sediments on wildlife and in humans, alternatives to the application should be explored.

12.0 STATEMENT OF LIMITATIONS

This review has been prepared and the work referred to in this review has been undertaken by Biowest Research Consultants for the United Fishermen and Allied Workers' Union (UFAWU-CAW). It is intended for the sole and exclusive use of the UFAWU-CAW and its authorized agents for the purpose(s) set out in this review. Any use of, reliance on or decision made based on this report by any person other than UFAWU-CAW for any purpose, or by UFAWU-CAW for a purpose other than the purpose(s) set out in this review, is the sole responsibility of such other person or UFAWU-CAW. UFAWU-CAW and BIOWEST RESEARCH CONSULTANTS make no representation or warranty to any other person with regard to this review and the work referred to in this review and they accept no duty of care to any other person or any liability or responsibility whatsoever for any losses, expenses, damages, fines, penalties or other harm that may be suffered or incurred by any other person as a result of the use of, reliance on, any decision made or any action taken based on this review or the work referred to in this review.

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The evaluation and conclusions reported herein do not preclude the identification of additional literature pertinent to the compounds discussed in this review. If new literature/studies become available, modifications to the findings, conclusions and recommendations in this review may be necessary.

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13.0 REFERENCES

- Åkerblom N, Goedkoop W, Nilsson T, Kylin H. 2010. Particle-specific sorption/desorption properties determine test compound fate and bioavailability in toxicity tests with *Chironomus riparius* – high-resolution studies with lindane. *Environ. Toxicol. Chem.* 29:1520–1528.
- Akhtar FZ, Garabrant DH, Ketchum NS, Michalek JE. 2004. Cancer in US Air Force veterans of the Vietnam War. *J. Occup. Environ. Med.* 46:123-136.
- Akkanen J, Slootweg T, Mäenpää K, Leppänen MT, Agbo S, Gallampois C, Kukkonen JVK. 2012. Bioavailability of organic contaminants in freshwater environments, emerging and priority pollutants in rivers. Springer-Verlag, Heidelberg.
- Ankley GT, Collyard SA, Monson PD, Kosian PA. 1994. Influence of ultra-violet light on the toxicity of sediments contaminated with polycyclic aromatic hydrocarbons. *Environ. Toxicol. Chem.* 13:1791-1796.
- Ankley GT, Erickson RJ, Phipps GL, Mattson VR, Kosian PA, Sheedy BR, Cox JS. 1995. Effects of light intensity on the phototoxicity of fluoranthene to a benthic macroinvertebrate. *Environ. Sci. Technol.* 29:2828-2833.
- Antkiewicz DS, Burns CG, Carney SA, Peterson RE, Heideman W. 2005. Heart malformation is an early response to TCDD in embryonic zebrafish. *Toxicol. Sci.* 84(2):368-377.
- Antkiewicz DS, Peterson RE, Heideman W. 2006. Blocking expression of AHR2 and ARNT1 in zebrafish larvae protects against cardiac toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Toxicol. Sci.* 94(1):175-182.
- Apple J, del Giorgio P, Newell R. 2001. Use of microbial community metabolism to assess saltmarsh ecological function. North Carolina National Estuarine Research Reserve Technical Paper Series. Horn Point Laboratory, University of Maryland. Horn Point, Maryland.
- Arkoosh MR, Casillas E, Huffman P, Clemons E, Evered J., Stein JE, Varanasi U. 1998. Increased susceptibility of juvenile Chinook salmon from a contaminated estuary to *Vibrio anguillarum*. *Trans. Am. Fish. Soc.*
- Arnot JA, Gobas FAPC. 2006. A review of bioconcentration factor (BCF) and bioaccumulation factor (BAF) assessments for organic chemicals in aquatic organisms. *Env. Rev.* 14(4): 257-297.
- Auffret, M, Duchemin M, Rousseau S, Boutet I, Tanguy A, Moraga D, Marhic A. 2004. Monitoring of immunotoxic responses in oysters reared in areas contaminated by the Erika oil spill. *Aquat. Living Resour.* 17:297–302.
- Augspurger TP, Echols KR, Peterman PH, May TW, Orazio CE, Tillitt DE, Di Giulio RT. 2008. Accumulation of environmental contaminants in wood duck (*Aix sponsa*) eggs, with emphasis on polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans. *Arch. Environ. Contam. Toxicol.* 55:670-682.

- Baccarelli A, Giacomini SM, Corbetta C, Landi MT, Bonzini M, Consonni D, Grillo P, Patterson, DG, Pesatori AC, Bertaazi, PA. 2008. Neonatal thyroid function in Sevesco 25 years after maternal exposure to dioxin. *PLoS Med.* 5(7):e161.
- Bakhiya N, Appel KE. 2010. Toxicity and carcinogenicity of furan in human diet. *Arch. Toxicol.* 84:563-578.
- Ballschmiter K, Buchert H, Niemczyk R, Munder A, Swerev M. 1986. Automobile exhausts versus municipal waste incineration as sources of the polychlorodibenzodioxins (PCDD) and -furans (PCDF) found in the environment. *Chemosphere.* 15:901-915.
- Barbee GC, Barich J, Duncan B, Bickham JW, Matson CW, Hintze CJ, Autenrieth RL, Zhou G-D, McDonald TJ, Cizmas L, Norton D, Donnelly KC. 2008. In situ biomonitoring of PAH-contaminated sediments using juvenile coho salmon (*Oncorhynchus kisutch*). *Ecotox. Environ. Safe.* 71:454-464.
- Barber CM. Dietary uptake models used for modeling the bioaccumulation of organic contaminants in fish. *Environ. Toxicol. Chem.* 27(4):755-777.
- Bauder MB, Palace VP, Hodson PV. 2005. Is oxidative stress the mechanism of blue sac disease in retene-exposed trout larvae? *Environ. Toxicol. Chem.* 24:694-702.
- Baumard P, Budzinky H, Michon Q, Garrigues P, Burgeot T, Bellocq J. 1998. Origin and bioavailability of PAHs in the Mediterranean Sea from mussel and sediment records. *Est. Coast. Shelf Sci.* 47:77-90.
- Baussant T, Bechmann RK, Taban IC, Larsen BK, Tandberg AH, Bjornstad A, Torgrimsen S, Naevdal A, Oysaed KB, Jonsson G, Sanni S. 2009. Enzymatic and cellular responses in relation to body burden of PAHs in bivalve molluscs: a case study with chronic levels of North Sea and Barents Sea dispersed oil. *Mar. Pollut. Bull.* 58:1796-1807.
- Beck H, Dross A, Eckart K, Mathar W, Wittkowski R. 1989. PCDDs and PCDFs and related compounds in paper products. *Chemosphere.* 19:655-660.
- Beischlag TV, Morales JL, Hollingshead BD, Perdew GH. 2008. The aryl hydrocarbon receptor complex and the control of gene expression. *Crit. Rev. Eukaryot. Gene Expr.* 18:207-250.
- Bello SM, Heideman W, Peterson RE. 2004. 2,3,7,8-Tetrachlorodibenzo-p-dioxin inhibits regression of the common cardinal vein in developing zebrafish. *Toxicol. Sci.* 78(2):258-266.
- Bertazzi PA, Consonni D, Bachetti S, Rubagotti M, Baccarelli A, Zocchetti C, Pesatori AC. 2001. Health effects of dioxin exposure: a 20-year mortality study. *Am. J. Epidemiol.* 153(11):1031-1044.
- Bigsby R, Chapin RE, Daston GP, Davis BJ, Gorski J, Gray LE, Howdeshell KL, Zoeller RT, vom Saal FS. 1999. Evaluating the effects of endocrine disruptors on endocrine function during development. *Environ. Health Perspect.* 107 Suppl 4:613-618.
- Billiard SM, Querbach K, Hodson PV. 1999. Toxicity of retene to early life stages of two freshwater fish species. *Environ. Toxicol. Chem.* 18:2070-2077.

- Billiard SM, Querbach K, Hodson PV. 1999. Toxicity of retene to early life stages of two freshwater fish species. *Environ. Toxicol. Chem.* 18:2070-2077.
- Binelli A, Riva C, Cogni D, Provini A. 2008. Assessment of the genotoxic potential of benzo(a)pyrene and pp-dichlorodiphenyldichloroethylene in zebra mussel (*Dreissena polymorpha*). *Mutat. Res. Genet. Toxicol. Environ. Mutagenesis.* 649(1-2):135-145.
- Black JA, Birge WJ, Westerman, A.G. (1983). Comparative aquatic toxicology of aromatic hydrocarbons. *Fundam. Appl. Toxicol.* 3: 353–358.
- Blanc AM, Holland LG, Rice SD, Kennedy CJ. 2010. Anthropogenically sourced low concentration PAHs: In situ bioavailability to juvenile Pacific salmon. *Ecotox. Environ. Safe.* 73:849-857.
- Blus LJ. 1982. Further interpretations of the relation of organochlorine residues in brown pelican eggs to reproductive success. *Environ. Pollut. A.* 28:15–33.
- Bock KW, Köhle C. 2006. Ah receptor: Dioxin-mediated toxic responses as hints to deregulated physiologic functions. *Biochem. Pharmacol.* 72:393-404.
- Bodner KM, Collins JJ, Bloemen LJ, Carson ML. 2003. Cancer risk for chemical workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Occup. Environ. Med.* 60:672-675.
- Boese BL, Ozretich RJ, Lamberson JO, Cole FA, Swartz RC , Ferraro SP. 2000. Phototoxic evaluation of marine sediments collected from a PAH-contaminated site. *Arch. Environ. Contam. Toxicol.* 38:274-282.
- Bosveld ATC, Gradener J, Murk AJ, Brouwer A, van Kampen M, Evers EHG, Van den Berg M. 1995. Effects of PCDDs, PCDFs, and PCBs in common tern (*Sterna hirundo*) breeding in estuarine and coastal colonies in the Netherlands and Belgium. *Environ. Toxicol. Chem.* 14:99–115.
- Box A, Sureda A, Galgani F, Pons A, Deudero S. 2007. Assessment of environmental pollution at Balearic Islands applying oxidative stress biomarkers in the mussel *Mytilus galloprovincialis*. *Comp. Biochem. Physiol.* 146C:531-539.
- Bravo CF, Curtis LR, Myers MS, Meador JP, Johnson LL, Buzitis J, Collier TK, Morrow JD, Laetz CA, Loge FJ, Arhoosh MR. 2011. Biomarker responses and disease susceptibility in juvenile rainbow trout *Oncorhynchus mykiss* fed a high molecular weight PAH mixture. *Environ. Toxicol. Chem.* 30:704-714.
- Bray RN (Ed.). 2008. Chapter 7- Reuse, Recycle or Relocate, “Environmental Aspects of Dredging”. Taylor & Francis.
- Bridges T, Ells S, Hayes D, Mount D, Nadeau S, Palermo M, Patmont C, Schroeder P. 2008. The Four Rs of Environmental Dredging: Resuspension, Release, Residual, and Risk. Dredging Operations and Environmental Research Program.
- Brinkworth LC, Hodson PV, Tabash S, Lee P. 2003. CYP1A induction and blue sac disease in early developmental stages of rainbow trout (*Oncorhynchus mykiss*) exposed to retene. *J. Toxicol. Environ. Health A.* 66(7):627-646.

- Brown RP, Cooper KR, Christini A, Rappe C, Bergqvist PA. 1994. Polychlorinated dibenzo-p-dioxins and dibenzofurans in *Mya arenaria* in the Newark/Raritan Bay estuary. *Environ. Toxicol. Chem.* 13:523–528.
- Brown RP. 1991. The toxicokinetics and histological effects of 2,3,7,8-Tetrachlorodibenzo-p-dioxin on the soft-shell clam, *Mya arenaria*. PhD thesis, Rutgers University, New Brunswick, NJ, USA.
- Cantrell SM, Joy-Schlezing J, Stegemann JJ, Tillitt DE, Hannink M. 1998. Correlation of 2,3,7,8-tetrachlorodibenzo-p-dioxin-induced apoptotic cell death in the embryonic vasculature with embryotoxicity. *Toxicol. Appl. Pharmacol.* 48(1):24-34.
- Cappuzzo J, Farrington M, Rantamaki P, Clifford C, Lancaster B, Leavitt D, Jia X. 1989. The relationship between lipid composition and seasonal differences in the distribution of PCBs in *Mytilus edulis*. *Mar. Environ. Res.* 14:201–228.
- Carls MG, Heintz RA, Marty GD, Rice SD. 2005. Cytochrome P4501A induction in oil-exposed pink salmon *Oncorhynchus gorbuscha* embryos predicts reduced survival potential. *Mar. Ecol. Prog. Ser.* 301:253-265.
- Carls MG, Holland L, Larsen M, Collier TK, Scholz NL, Incardona JP. 2008. Fish embryos are damaged by dissolved PAHs, not oil particles. *Aquat. Toxicol.* 88:121-127.
- Carney SA, Chen J, Burns CG, Xiong KM, Peterson RE, Heideman W. 2006. Aryl hydrocarbon receptor activation produces heart-specific transcriptional and toxic responses in developing zebrafish. *Mol. Pharmacol.* 70(2):549-561.
- Carr- Harris, C., Moore, J.W. 2013. Juvenile Salmonid Habitat Utilization in the Skeena River Estuary. Earth to Ocean Research Group, Department of Biological Sciences, Simon Fraser University. Prepared for: Skeena Wild Conservation Trust.
- CCME (Canadian Council of Ministers of the Environment). 1996. A framework for ecological risk assessment : General guidance. The National Contaminated Sites Remediation Program. Winnipeg, Manitoba.
- CCME. 2001. Canadian Sediment Quality Guidelines for Protection of Aquatic Life. Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzo furans (PCDD/Fs), using toxicity equivalency factors for fish. C. C. o. M. o. t. Environment. Winnipeg.
- Chen J, Carney SA, Peterson RE, Heideman W. 2008. Comparative genomics identifies genes mediating cardiotoxicity in the embryonic zebrafish heart. *Physiol. Genomics.* 33(2):148-158.
- Cheng, T.C., 1996. Hemocytes: forms and functions. In: Kennedy, V.S., Newell, R.I.E., Eble, A.F. (Eds.), *The Eastern Oyster Crassostrea virginica*. Maryland Sea Grant College, College Park.
- Cole P, Trichopoulos D, Pastides H, Starr T, Mandel JS. 2003. Dioxin and cancer: a critical review. *Regul. Toxicol. Pharm.* 38:378-388.
- Cook PM, Robbins JA, Endicott DD, Lodge KB, Guiney PD, Walker MK, Zabel EW, Peterson RE. 2003. Effects of aryl hydrocarbon receptor-mediated early life stage toxicity on lake trout

- populations in Lake Ontario during the 20th century. *Environ. Sci. Technol.* 37(17):3864-3877.
- Cooper KR and Wintermyer M. 2009. A critical review: 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) effects on gonad development in bivalve mollusks. *J. Environ. Sci. Health C Environ. Carcinog. Ecotoxicol. Rev.* 27(4):226-245.
- Croxton AN, Wikfors GH, Schulterbrandt-Gragg RD. 2012. Immunomodulation in eastern oysters, *Crassostrea virginica*, exposed to a PAH-contaminated, microphytobenthic diatom. *Aquat. Toxicol.* 118-119:27-36.
- Custer TW, Hensler GL, Kaiser TE. 1983. Clutch size, reproductive success, and organochlorine contaminants in Atlantic coast black-crowned night-herons. *Auk.* 100:699-710.
- da Silva Rocha AJ, Gomes V, Rocha Passos MJ, Hasue FM, Alves Santos TC, Bicego MC, Taniguchi S, Van Ngan P. 2012. EROD activity and genotoxicity in the seabob shrimp *Xiphopenaeus kroyeri* exposed to benzo(a)pyrene (BaP) concentrations. *Environ. Toxicol. Pharmacol.* 34(3):995-1003.
- Dailianis S. 2009. Production of superoxides and nitric oxide generation in haemocytes of mussel *Mytilus galloprovincialis* (Lmk.) after exposure to cadmium: A possible involvement of Na⁺/H⁺ exchanger in the induction of cadmium toxic effects. *Fish Shellfish Immunol.* 27:446-453.
- Danellakis D, Ntaikou I, Kornaros M, Dilianis S. 2011. Olive oil mill wastewater toxicity in the marine environment: alterations of stress indices in tissues of mussel *Mytilus galloprovincialis*. *Aquat. Toxicol.* 101:358-366.
- Davis HS. Culture and diseases of game fishes. Berkley, California: University of California Press, 1953. pp. 294-295.
- De Swart RL, Ross, PS, Vos, JG, Osterhaus MADE. 1996. Impaired immunity in harbor seals (*Phoca vitulina*) exposed to bioaccumulated environmental contaminants: review of a long-term study. *Environ. Health. Perspect.* 104: 823-828.
- Del Rio D, Steward AJ, Pellegrini N. 2005. A review of recent studies on malondialdehyde as toxic molecule and biological marker of oxidative stress. *Nutr. Metab. Cardiovasc. Dis.* 15:316-328.
- Department of the Environment. 1973b. Preliminary environmental effect assessment superport development Prince Rupert region, summary conclusions and recommendations. Environment Canada. Ottawa, ON.
- DFO. 2001. Fish stocks of the Pacific Coast. Fisheries and Oceans Canada. Ottawa, ON.
- DFO. 2011b. MAPSTER (version 3.0). Fisheries and Oceans Canada, Pacific Region, Oceans, Habitat and Enhancement Branch. Fish and fish habitat related information served from an ESRI ArcIMS Server using the WMS Connector. Available at: http://www-heb.pac.dfo-mpo.gc.ca/maps/maps-data_e.htm. Accessed: November, 2008.
- DFO. 2013k. Pacific Region Integrated Fisheries Management Plan, Crab by Trap, January 1, 2013 to December 31, 2013. F. a. O. Canada.

DiMichele L, Taylor MH. 1978. Histopathological and physiological responses of *Fundulus heteroclitus* to naphthalene exposure. *J. Fish. Res. Bd. Can.* 35: 1060-1066.

Dong W, Teraoka H, Kondo S, Hiraga T. 2001. 2,3,7,8-Tetrachlorodibenzo-p-dioxin induces apoptosis in the dorsal midbrain of zebrafish embryos by activation of arylhydrocarbon receptor. *Neurosci. Lett.* 303(3):169-172.

Dunnivant FM, Elzerman AW. 1988. Aqueous solubility and Henry's Law PCB congeners for evaluation of quantitative structure-property relationships (QSPRs). *Chemosphere.* 17(3): 525-541.

Constant data

Eggleton J, Thomas K. 2004. A review of factors affecting the release and bioavailability of contaminants during sediment disturbance events. *Environ. Int.* 30:973-980.

Elmore LW, Sirica AE. 1991. Phenotypic characterization of metaplastic intestinal glands and ductular hepatocytes in cholangiofibrotic lesions rapidly induced in the caudate liver lobe of rats treated with furan. *Cancer Res.* 51:5752-5759.

EPA. (2000). Bioaccumulation testing and interpretation for the purpose of sediment quality assessment status and needs, EPA-823-R-00-001. Office of Water. 136 pp.

ERDC, Engineer Research and Development Center (ERDC). 2008. Chapter 5- Estimate of Suspended Sediment Loss, "Hamilton Wetland Restoration Project". Received from <http://www.rivermod.elling.com/HamiltonDownloads/TechnicalReport/Chapter_5_ERDC_Final.pdf>

Esterbauer H, Schaur RJ, Zollner H. 1991. Chemistry and biochemistry of 4-hydroxynonenal, malondialdehyde and related aldehydes. *Free Radic. Biol. Med.* 11:81-128.

Faggetter, B. 2013. Chatham Sound Eelgrass Study Final Report. Prepared for World Wildlife Fund by Ocean Ecology. Prince Rupert, B.C.

Faggetter, B.A. 2009b. Flora Bank Eelgrass Survey. Prepared for WWF.

Faggetter, B.A. 2014. Skeena River Estuary Habitat Effects on Juvenile Salmon. Prepared for the Skeena Watershed Conservation Coalition and the Skeena Wild Conservation Trust. In review.

Fairbrother A, Wenstel R, Sappington K, Wood W. 2007. Framework for metals risk assessment. *Ecotoxicol. Environ. Saf.* 68:145-227.

Fallahtafti S, Rantanen T, Brown RS, Snieckus V, Hodson PV. 2012. Toxicity of hydroxylated alkyl-phenanthrenes to early life stages of Japanese medaka (*Oryzias latipes*). *Aquat. Toxicol.* 106-107:56-64.

Farr AJ, Chabot CC, Taylor DH. 1995. Behavioral avoidance of fluoranthene by fathead minnow (*Pimephales promelas*). *Neurotox. Teratol.* 17:265-271.

Fatima M, Ahmad I, Siddiqui R, Raisuddin S. 2001. Paper and pulp mill effluent-induced immunotoxicity in freshwater fish *Channa punctatus* (Bloch). *Arch. Environ. Contam. Toxicol.* 40(2):271-276.

- Feldmannová M, Hilscherová K, Marsálek B, Bláha L. 2006. Effects of N-heterocyclic polyaromatic hydrocarbons on survival, reproduction, and biochemical parameters in *Daphnia magna*. *Environ. Toxicol.* 21(4):425-431.
- Field JA, Sierra-Alvarez R. 2008. Microbial degradation of chlorinated dioxins. *Chemosphere.* 71(6):1005-1018.
- Fisher WS. 1986. Structure and functions of oyster hemocytes. In: Brehelin, M. (Ed.), *Immunity in invertebrates*. Springer-Verlag, Berlin.
- Fleeger JW, Carman KR, Nisbet RM. 2003. Indirect effects of contaminants in aquatic ecosystems. *Sci. Total Environ.* 317:207–233.
- Fletcher N, Hanberg A, Håkansson H. 2001. Hepatic vitamin A depletion is a sensitive marker of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) exposure in four rodent species. *Toxicol. Sci.* 62:166-175.
- Fox GA, Gilbertson M, Gilman AP, Kubiak TJ. 1991. A rationale for the use of colonial fish-eating birds to monitor the presence of developmental toxicants in Great Lakes fish. *J. Great Lakes Res.* 17:151–152.
- Fragoso NM, Hodson PV, Zambon S. 2006. Evaluation of an exposure assay to measure uptake of sediment PAH by fish. *Environ. Monitor. Assess.* 116:481-511.
- Fransson-Steen R, Goldsworthy TL, Kedderis GL, Maronpot RR. 1997. Furan-induced liver cell proliferation and apoptosis in female B6C3F1 mice. *Toxicology.* 118:195-204.
- Frouin H, Pellerin J, Fournier M, Pelletier E, Richard P, Pichaud N, Rouleau C, Garnerot F. 2007. Physiological effects of polycyclic aromatic hydrocarbons on soft-shell clam *Mya arenaria*. *Aquat. Toxicol.* 82(2):120-134.
- Furness RW (1993) Birds as monitors of pollutants. In: Furness RW, Greenwood JJD (eds) *Birds as monitors of environmental change*. Chapman and Hall, New York, pp 86–143.
- Gesto M, Soengas JL, Miguez JM. 2008. Acute and prolonged stress response of brain monoaminergic activity and plasma cortisol levels in rainbow trout are modified by PAHs (naphthalene, β -naphthoflavone and benzo(a)pyrene) treatment. *Aquat. Toxicol.* 86:341-351.
- Gesto M, Tintos A, Rodriguez-Illamola A, Soengas JL, Miguez JM. 2009. Effects of naphthalene, β -naphthoflavone and benzo(a) pyrene on the diurnal and nocturnal indoleamine metabolism and melatonin content in the pineal organ of rainbow trout, *Oncorhynchus mykiss*. *Aquat. Toxicol.* 92:1-8.
- Giannapas M, Karnis L, Dailianis S. 2011. Generation of free radicals in hemocytes of mussels after exposure to low molecular weight PAH components: immune activation, oxidative and genotoxic effects. *Comp. Biochem. Physiol. C Toxicol. Pharmacol.* 155(2):182-189.
- Giesy, JP, Jones, PD, Kannan, K, Newsted, JL, Tillitt, DE, Williams, LL. 2002. Effects of chronic dietary exposure to environmentally relevant concentrations to 2,3,7,8-tetrachlorodibenzo-p-dioxin on survival, growth, reproduction and biochemical responses of female rainbow trout (*Oncorhynchus mykiss*). *Aquat. Toxicol.* 59(1-2):35–53.

- Goncalves R, Scholze M, Ferreira AM, Martins M, Correia AD. 2008. The joint effect of polycyclic aromatic hydrocarbons on fish behaviour. *Environ. Res.* 108:205-213.
- Gottesfeld, A.S., Carr-Harris, C., Proctor, B., Rolston, D. 2008. Sockeye Salmon Juveniles in Chatham Sound 2007. *Pacific Salmon Forum*, July
- Grundy MM, Moore MN, Howell SM, Ratcliffe NA. 1996b. Phagocytic reduction and effects on lysosomal membranes by polycyclic aromatic hydrocarbons in hemocytes of *Mytilus edulis*. *Aquat. Toxicol.* 34(4):273-290.
- Grundy MM, Ratcliffe NA, Moore MN. 1996a. Immune inhibition in marine mussels by polycyclic aromatic hydrocarbons. *Mar. Environ. Res.* 42(1-4):187-190.
- Guillette LJ Jr, Crain DA, Rooney AA, Pickford DB. 1995. Organization versus activation: the role of endocrine-disrupting contaminants (EDCs) during embryonic development in wildlife. *Environ. Health Perspect.* 103 Suppl 7:157-164
- Guinand B, Scribner KT, Page KS, Burnham-Curtis MK. 2003. Genetic variation over space and time: analyses of extinct and remnant lake trout populations in the Upper Great Lakes. *Proc. Biol. Sci.* 270(1513):425-33.
- Guiney PD, Smolowitz RN, Peterson RE, Stegeman JJ. 1997. Correlation of 2,3,7,8-tetrachlorodibenzo-p-dioxin induction of cytochrome P4501A in vascular endothelium with toxicity in early life stages of lake trout. *Toxicol. Appl. Pharmacol.* 143(2):256-273.
- Hack LA, Tremblay LA, Wratten SD, Lister A, Keesing V. 2007. Benthic meiofauna community composition at polluted and non-polluted sites in New Zealand intertidal environments. *Mar. Pollut. Bull.* 54:1801–1812.
- Haddad S, Poulin P, Krishnan K. 2000. Relative lipid content as the sole mechanistic determinant of the adipose tissue: Blood partition coefficients of highly lipophilic organic chemicals. *Chemosphere.* 40:839–843.
- Hall AT, Oris JT. 1991. Anthracene reduces reproductive potential and is maternally transferred during long-term exposure in fathead minnows. *Aquat. Toxicol.* 19:249-264.
- Hamadeh HK, Jayadev S, Gaillard ET, Huang Q. 2004. Integration of clinical and gene expression endpoints to explore furan-mediated hepatotoxicity. *Mutat. Res.* 549:169-183.
- Handley-Goldstone HM, Grow MW, Stegeman JJ. 2005. Cardiovascular gene expression profiles of dioxin exposure in zebrafish embryos. *Toxicol. Sci.* 85(1):683-693.
- Hannah JB, Hose JE, Landolt ML, Miller BS, Felton SP, Iwaoka WT. 1982. Benzo(a)pyrene induced morphologic and developmental abnormalities in rainbow trout. *Arch. Environ. Contam. Toxicol.* 11: 727–734.
- Hannam ML, Bamber SD, Galloway TS, Moody JA, Jones MB. 2010. Effects of the model PAH phenanthrene on immune function and oxidative stress in the hemolymph of the temperate scallop, *Pecten maximus*. *Chemosphere.* 78(7):779-784.

- Hannam ML, Bamber SD, Moody JA, Galloway TS, Jones MB. 2009. Immune function in the Arctic scallop, *Chlamys islandica*, following dispersed oil exposure. *Aquat. Toxicol.* 92:187-194.
- Hatch AC and GA Burton, Jr. 1999. Photoinduced toxicity of PAHs to *Hyalella azteca* and *Chironomus tentans*: effects of mixtures and behavior. *Environ. Pollut.* 106:157-167.
- Hayes, D.F., T.D. Borrowman and P.R. Schroeder. 2007. Process-Based Estimation of Sediment Resuspension Losses during Bucket Dredging. . Proceedings, XVIII World Dredging Congress 2007 Paper presented at the Proceedings, XVIII World Dredging Congress 2007, WEDA, Lake Buena Vista, Florida, USA.
- Heideman W, Antkiewicz DS, Carney SA, Petersen RE. 2005. Zebrafish and cardiac toxicology. *Cardiovasc. Toxicol.* 5(2):203-214.
- Heijden, VD, Jonker MTO. 2009. PAH bioavailability in field sediments: comparing different methods for predicting in situ bioaccumulation. *Environ. Sci. Technol.* 43:3757–3763.
- Henry TR, Spitsbergen JM, Hornung MW, Abnet CC, Peterson RE. 1997. Early life stage toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in zebrafish (*Danio rerio*). *Toxicol. Appl. Pharmacol.* 142(1):56-68.
- Hermes-Lima M, Storey JM, Storey KB. 2001. Antioxidant defenses and animal adaptation to oxygen availability during environmental stress. In: Storey KB, Storey JM (Eds.), *Protein adaptation and signal transduction*. Elsevier Science BV, Amsterdam.
- Hickling KC, Hitchcock JM, Oreffo V, Mally A, Hammond TG, Evans JG, Chipman JK. 2010. Evidence of oxidative stress and associated DNA damage, increased proliferative drive and altered gene expression in rat liver produced by the cholangiocarcinogenic agent furan. *Toxicol. Pathol.* 38:230-243.
- Hites RA, Harless RL (1991) Atmospheric transport and deposition of polychlorinated dibenzo-p-dioxins and dibenzofurans. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development. EPA/600/3-91/002.
- Hoese HD and Moore RH. 1998. *Fishes of the Gulf of Mexico: Texas, Louisiana, and adjacent waters*. Second Edition. Texas A&M University Press. College Station, Texas.
- Hornung MW, Spitsbergen JM, Peterson RE. 1999. 2,3,7,8-Tetrachlorodibenzo-p-dioxin alters cardiovascular and craniofacial development and function in sac fry of rainbow trout (*Oncorhynchus mykiss*). *Toxicol. Sci.* 47(1):40-51.
- Hose JE, Hannah JB, DiJulio D, Landolt ML, Miller BS, Iwaoka WT, Felton SP. 1982. Effects of benzo(a)pyrene on early development of flatfish. *Arch. Environ. Contam. Toxicol.* 11:167-171.
- Hose JE, Hannah JB, Puffer WH, Landoit ML. 1984. Histologic and skeletal abnormalities in benzo(a)pyrene treated rainbow trout alevins. *Arch. Environ. Contam. Toxicol.* 13: 675–684.
- Huovinen PS, Soimasuo MR, Oikari AO. 2001. Photoinduced toxicity of retene to *Daphnia magna* under enhanced UV-B radiation. *Chemosphere.* 45(4-5):683-691.

- Hutz RJ, Carvan MJ, Baldrige MG, Conley LK, King-Heiden TC. 2006. Environmental toxicants and effects on female reproductive function. *Trends Repro. Biol.* 2:1–11.
- Hwang H-M, Wade TL, Sericano JL. 2002. Relationship between lysosomal membrane destabilization and chemical body burden in eastern oysters (*Crassostrea virginica*) from Galveston Bay, Texas USA. *Environ. Toxicol. Chem.* 21:1268–1271.
- Incardona JP, Collier TK, Scholz NL. 2004. Defects in cardiac function precede morphological abnormalities in fish embryos exposed to polycyclic aromatic hydrocarbons. *Toxicol. Appl. Pharmacol.* 196:191-205.
- Inserm (Institut national de la santé et de la recherche médicale) (2000) Dioxins in the Environment. ISBN: 2-85598-784-9
- Johansson E, Reynolds S, Anderson M, Maronpot R. 1997. Frequency of Ha-ras-1 gene mutations inversely correlated with furan dose in mouse liver tumours. *Mol. Carcinogen.* 18:199-205.
- Kaloyianni M, Dailianis S, Chrisikopoulou E, Zannou A, Koutsogiannaki S, Alamdari DH, Koliakos G, Dimitriadis VK. 2009. Oxidative effects of inorganic and organic contaminants on hemolymph of mussels. *Comp. Biochem. Physiol. C.* 149:631-639.
- Keller LF, Waller DM. 2002. Inbreeding effects in wild populations. *Trends Ecol. Evol.* 17:230–240.
- Kelly BC, Ikonomou MG, Blair JD, Morin AE, Gobas FAPC. 2007. Web-specific biomagnification of persistent organic pollutants. *Science.* 317(5835):236-239.
- Kennish MJ. 1992. Ecology of estuaries: anthropogenic effects. Boca Raton, CRC Press
- Kidd KA, Bootsman HA, Heslein RH. 2001. Biomagnification of DDT through the benthic and pelagic food webs of Lake Malawi, East Africa: importance of trophic level and carbon source. *Environ. Sci. Technol.* 35:14-20.
- Kidd KA, Schindler DW, Heslein RH. 1998. Effects of trophic position and lipid on organochlorine concentrations in fishes from subarctic lakes in Yukon Territory. *Can. J. Fish. Aquat. Sci.* 55(4): 869-881.
- King-Heiden TC, Carvan MJ 3rd, Hutz RJ. 2006. Inhibition of follicular development, vitellogenesis, and serum 17 β -estradiol concentrations in zebrafish following chronic, sublethal dietary exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Toxicol. Sci.* 90(2):490-499.
- King-Heiden TC, Hutz RJ, Carvan MJ 3rd. 2005. Accumulation, tissue distribution, and maternal transfer of dietary 2,3,7,8,-tetrachlorodibenzo-p-dioxin: impacts on reproductive success of zebrafish. *Toxicol. Sci.* 87(2):497-507.
- King-Heiden TC, Mehta V, Xiong HM, Lanham KA, Antkiewicz DS, Ganser A, Heideman W, Peterson RE. 2011. Reproductive and developmental toxicity of dioxin in fish. *Mol. Cell. Endocrinol.* 354(1-2):121-138.

- King-Heiden TC, Spitsbergen J, Heideman W, Peterson RE. 2009. Persistent adverse effects on health and reproduction caused by exposure of zebrafish to 2,3,7,8-tetrachlorodibenzo-p-dioxin during early development and gonad differentiation. *Toxicol. Sci.* 109(1):75-87.
- Kirkin MA, Moore MN, Dean RT, Winston GW. 1992. The role of oxyradicals in intracellular proteolysis and toxicity in mussels. *Mar. Environ. Res.* 34:315-320.
- Kogevinas M, Becher H, Benn T. 1997. Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols and dioxins. An expanded and updated international cohort study. *Am. J. Epidemiol.* 145:1061-1075.
- Kogevinas, M., 2001. Human health effects of dioxins: cancer, reproductive and endocrine system effects. *Hum. Reprod. Update.* 7(3):331-339.
- Kretchik, JT. 2002. Persistent organic pollutants. *Chem. Health Saf.* 9:35.
- Lamoureux E, Brownawell B. 1999. Chemical and biological availability of sediment-sorbed hydrophobic organic contaminants. *Env. Toxicol. Chem.* 18:1733-1741.
- Lampi MA, Gurska J, McDonald KI, Xie F, Huang XD, Dixon DG, Greenber BM. 2006. Photoinduced toxicity of polycyclic aromatic hydrocarbons to *Daphnia magna*: ultraviolet-mediated effects and the toxicity of polycyclic aromatic hydrocarbon photoproducts. *Environ. Toxicol. Chem.* 25(4):1079-1087.
- Livingstone DR. 2003. Oxidative stress in aquatic organisms in relation to pollution and aquaculture. *Rev. Med. Vet.* 154:427-430.
- Long ER, MacDonald DD, Smith SL, Calder FD. 1994. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. *Environ. Manage.*
- Longnecker MP, Michalek JE. 2000. Serum dioxin level in relation to diabetes mellitus among Air Force veterans with background levels of exposure. *Epidemiology.* 11(1):44-48.
- Loring DH, Rantala RTT. 1992. Manual for the geochemical analysis of marine sediments and suspended particulate matter. *Earth- Sci. Rev.* 32:235.
- Loring DH. 1991. Normalization of heavy-metal data from estuarine and coastal sediments. *ICES J. Mar. Sci.* 48:101-115.
- Loring DH.. 1990. Lithium: A new approach for the granulometric normalization of trace metal data. *Mar. Chem.* 29:155-168.
- Lyman WJ. 1995. Transport and transformation processes. In: Rand GM. (Ed.), *Fundamentals of aquatic toxicology: effects, environmental fate, and risk assessment.* Taylor & Francis, Washington.
- Lyons BP, Pascoe CK, McFadzen IR. 2002. Phototoxicity of pyrene and benzo(a)pyrene to embryo-larval stages of the Pacific oyster *Crassostrea gigas*. *Mar. Environ. Res.* 54(3-5):627-631.
- Lyytikäinen M, Rantalainen AL, Mikkelsen P, Hämäläinen H, Paasivirt J, Kukkonen JVK. 2003. Similarities in bioaccumulation patterns of polychlorinated dibenzo-p-dioxins and furans and polychlorinated diphenyl ethers in laboratory-exposed oligochaetes and semipermeable

- membrane devices and in field-collected chironomids. *Environ. Toxicol. Chem.* 22(10):2405-2415.
- MacDonald DD. 1993. Development of an approach to the assessment of sediment quality in Florida coastal waters. Prepared for the Florida Department of Environmental Protection. MacDonald Environmental Sciences, Ltd., Ladysmith, BC. Vol. 1, 128 pp.; Vol. 2, 117 pp.
- Mackay D, Shiu WY, Ma KC (1992) Illustrated handbook of physical-chemical properties and environmental fate for organic chemicals: polynuclear aromatic hydrocarbons, polychlorinated dioxins, and dibenzofurans. Chelsea, MI: Lewis Publishers.
- Marple L, Brunck R, Berridge B, Throop L. (1987) Experimental and calculated physical constants for 2,3,7,8-tetrachlorodibenzo-p-dioxin. In: Solving hazardous waste problems. Exner, J.H., Eds. ACS Symposium Series 338. Developed from a symposium sponsored by the Division of Environmental Chemistry at the 191st meeting of the American Chemical Society, New York, New York, April 13-18, 1986. pp. 105-113.
- Martinez-Gomez C, Vethaak AD, Hylland K, Burgeot T, Köhler A, Lyons BP, Thain J, Gubbins MJ, Davies IM. 2010. A guide to toxicity assessment and monitoring effects at lower levels of biological organization following marine oil spills in European waters. *J. Mar. Sci.* 67:1105–1118.
- Martínez-Jerónimo F, Cruz-Cisneros JL, García-Hernández L. 2008. A comparison of the response of *Simocephalus mixtus* (Cladocera) and *Daphnia magna* to contaminated freshwater sediments. *Ecotoxicol. Environ. Saf.* 71:26–31.
- Matsumura F. 2003. On the significance of the role of cellular stress response reactions in the toxic actions of dioxin. *Biochem. Pharmacol.* 66:527-540.
- Mayrand E, St Jean SD, Courtenay SC. 2005. Hemocyte responses of blue mussels (*Mytilus edulis* L.) transferred from a contaminated site to a reference site: can the immune system recuperate? *Aquacult. Res.* 36:962–971.
- Mazurová E, Hilscherová K, Jálová V, Köhler HR, Triebkorn R, Giesy JP, Bláha L. 2008. Endocrine effects of contaminated sediments on the freshwater snail *Potamopyrgus antipodarum* *in vivo* and in the cell bioassays *in vitro*. *Aquat. Toxicol.* 89(3):172-179.
- Meador JP, Sommers FC, Ylitalo GM, Sloan CA. 2006. Altered growth and related physiological responses in juvenile Chinook salmon (*Oncorhynchus tshawytscha*) from dietary exposure to polycyclic aromatic hydrocarbons (PAHs). *Can. J. Fish. Aquat. Sci.* 63:2364-2376.
- Mehler WT, Li H, Pang J, Sun B, Lydy MJ, You J. 2011. Bioavailability of hydrophobic organic contaminants in sediment with different particle-size distributions. *Arch. Environ. Contam. Toxicol.* 61:74–82.
- Mehta V, Peterson RE, Heideman W. 2008. 2,3,7,8-Tetrachlorodibenzo-p-dioxin exposure prevents cardiac valve formation in developing zebrafish. *Toxicol. Sci.* 104(2):303-311.
- Michalek JE, Akhtar FZ, Kiel JL. 1999. Serum dioxin, insulin, fasting glucose and sex-hormone binding globulin in veterans of Operation Ranch Hand. *J. Clin. Endocr. Metab.* 84(5):1540-1543.

- Michel C, Bourgeault A, Gourlay-France C, Palais F, Geffard A, Vincent-Hubert F. 2013. Seasonal and PAH impact on DNA strand-break levels in gills of transplanted zebra mussels. *Ecotoxicol. Environ. Saf.* 92:18-26.
- Michel C, Vincent-Hubert F. 2012. Detection of 8-oxodG in *Dreissena polymorpha* gill cells exposed to model contaminants. *Mutat. Res.* 741(1-2):1-6.
- Miller RA, Norris LA, Hawkes CL. 1973. Toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in aquatic organisms. *Environ. Health Perspect.* 5:177–186.
- Mocarelli P, Gertoux PM, Ferrari E. 2000. Paternal concentrations of dioxin and sex ratio of offspring. *Lancet.* 355:1858-1863.
- Moles A, Bates S, Rice SD, Korn S. 1981. Reduced growth of coho salmon fry exposed to two petroleum components, toluene and naphthalene, in freshwater. *Trans. Am. Fish. Soc.* 110:430-436.
- Monson PD, Ankley GT, Kosian. PA. 1995. Phototoxic response of *Lumbriculus variegatus* to sediments contaminated by polycyclic aromatic hydrocarbons. *Environ. Toxicol. Chem.* 14:891-894.
- Moore JW, Ramamoorthy S. 1984. Aromatic hydrocarbons—polycyclic. In: Organic chemicals in natural waters: Applied monitoring and impact assessment. Springer-Verlag, New York. EPA-600/7-78-074. U.S. Environmental Protection Agency, Environmental Processes Branch, Environmental Research
- Moore MN, Wedderburn RJ, Lowe DM, Depledge MH. 1996. Lysosomal reaction to xenobiotics in mussel hemocytes using BODIPY-FL-Verapamil. *Mar. Environ. Res.* 42:99–105.
- Morán FM, Vandevort CA, Overstreet JW, Lasley BL, Conley AJ. 2003. Molecular target of endocrine disruption in human luteinizing granulosa cells by 2,3,7,8-tetrachlorodibenzo-p-dioxin: inhibition of estradiol secretion due to decreased 17 α -hydroxylase/17,20-lyase cytochrome P450 expression. *Endocrinology.* 144(2):467-473.
- Moro S, Chipman JK, Wegener J, Hamberger C, Dekant W, Mally A. 2012. Furan in heat-treated foods: formation, exposure, toxicity and aspects of risk assessment. *Mol. Nutr. Food Res.* 56:1197-1211.
- Muir DCG, Lawrence S, Holoka M, Fairchild WL, Segstro MD, Webster GRB, Servos MR. 1992. Partitioning of polychlorinated dioxins and furans between water, sediments and biota in lake mesocosms. *Chemosphere.* 25(1-2):199-124.
- Naito W, Jin J, Kang YS, Yamamuro M, Masunaga S, Nakanishi J. 2003. Dynamics of PCDDs/DFs and coplanar-PCBs in an aquatic food chain of Tokyo Bay. *Chemosphere.* 53:347-362.
- Nakajima S, Saijo Y, Kato S, Sasaki S, Uno A, Kanagami N, Hirakawa, H, Hori T, Tobiishi K, Todaka T, Nakamura Y, Yanagiya S, Sengoku Y, Iida T, Sata F, Kishi R. 2006. Effects of prenatal exposure to polychlorinated biphenyls and dioxins on mental and motor development in Japanese children at 6 months of age. *Environ. Health Persp.* 114(5):773-778.
- National Research Council (NRC). (1997). Contaminated sediments in ports and waterways. Washington, DC: National Academy Press.

- National Research Council of Canada. 1983. Polycyclic aromatic hydrocarbons in the aquatic environment: formation, sources, fate and effects on aquatic biota. NRCC No. 18981.
- Neff J.M. 1979. Polycyclic aromatic hydrocarbons in the aquatic environment: sources, fates and biological effects. Applied Sciences Publishers Ltd., London.
- Neff JM, Cox BA, Dixit D, Anderson JW. 1976. Accumulation and release of petroleum-derived aromatic hydrocarbons by four species of marine animals. Mar. Biol. 38:279–289.
- Neff JM. 1979. Polycyclic Aromatic Hydrocarbons in the Aquatic Environment: Sources, Fates and Biological Effects. Applied Science Publishers Ltd., Essex, England. 262 p.
- Nesto N, Romano S, Moschino V, Mauri M, Da Ros L. 2007. Bioaccumulation and biomarker responses of trace metals and micro-organic pollutants in mussels and fish from the Lagoon of Venice, Italy. Mar. Pollut. Bull. 55:469-484.
- Newsted J, Giesy JP. 1987. Predictive models for photoinduced acute toxicity of polycyclic aromatic hydrocarbons to *Daphnia magna*, strauss (Cladocera, Crustacea). 6(6):445-461.
- Nilsson CB, Håkansson H. 2002. The retinoid signalling system – a target in dioxin toxicity. Crit. Rev. Toxicol. 32(2):211-232.
- NRC, 2003. Oil in the sea III: inputs, fates, and effects. The National Academies Press, Washington, D.C.
- Öberg LG, et al (1992) *De novo* formation of hepta- and octachlorodibenzo-p-dioxins from pentachlorophenol in municipal sewage sludge. In: *Dioxin '92: 12th International Symposium on Chlorinated Dioxins and Related Compounds, Tampere, Finland, 24–28 August 1992*. Helsinki, Finnish Institute of Occupational Health, 1992: 351–354 (Organohalogen Compounds, Vol. 9).
- Öberg LG, et al (1993) *De novo* formation of PCDD/Fs in compost and sewage sludge—a status report. In: Fielder, H. et al., ed. *Dioxin '93: 13th International Symposium on Chlorinated Dioxins and Related Compounds, Vienna, 20–24 September 1993*. Vienna, Austrian Federal Environment Agency, 1993: 297–302 (Organohalogen Compounds, Vol. 11).
- Odum EP 1975. Ecology: The link between the natural and the social sciences. Second Edition. Modern Biology Series. Holt, Rinehart, and Winston. New York, New York.
- Oikario A, Niityla J. 1985. Subacute physiological effects of bleached kraft mill effluent (BKME) on the liver of trout, *Salmo gairdneri*. Ecotoxicol. Environm. Saf. 10:159–172.
- Okumura Y, Yamashita Y, Isagawa S. 2003. Sources of polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and coplanar polychlorinated biphenyls (Co-PCBs), and their bioaccumulation through the marine food web in Sendai Bay, Japan. J. Environ. Monitor. 5:610-618.
- Oliver LM, Fisher WS, Volety AK, Malaeb Z. 2003. Greater hemocyte bactericidal activity in oysters (*Crassostrea virginica*) from a relatively contaminated site in Pensacola Bay, Florida. Aquat. Toxicol. 64:363–373.
- Oliver LM, Fisher WS. 1999. Appraisal of prospective bivalve immunomarkers. Biomarkers. 4:510-530.

- Olivieri CE, Cooper KR. 1996. Toxicity of the 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in embryos and larvae of the fathead minnow (*Pimephales promelas*). *Chemosphere*. 34: 1139–1150.
- Palm Jr. R, Powell DB, Skillman A, Godtfredsen K. 2003. Immunocompetence of juvenile chinook salmon against *Listonella anguillarum* following dietary exposure to polycyclic aromatic hydrocarbons. *Environ. Toxicol. Chem.* 22 (12), 2986–2994.
- Pande K, Moran SM, Bradfield CA. 2005. Aspects of dioxin toxicity are mediated by interleukin1-like cytokines. *Mol. Pharmacol.* 67(5):1393-1398.
- Payne JF, Kiceniuk J, Fancey LL, William U. 1988. What is a safe level of polycyclic aromatic hydrocarbons for fish: subchronic toxicity study on winter flounder (*Pseudopleuronectes americanus*). *Can. J. Fish. Aquat. Sci.* 45:1983-1993
- Peachey RBJ. 2005. The synergism between hydrocarbon pollutants and UV radiation: a potential link between coastal pollution and larval mortality. *J. Exp. Mar. Biol. Ecol.* 315:103-114.
- Peakall DB, Boyd H. 1987. Birds as bio-indicators of environmental conditions. *ICBP Tech. Publ.* 6:113–118.
- Pelletier MC, Burgess RM, Cantwell MG, Serbst Jr, Ho KT, Ryba SA. 2000. Importance of maternal transfer of the photoreactive polycyclic aromatic hydrocarbon fluoranthene from benthic adult bivalves to their pelagic larvae. *Environ. Toxicol. Chem.* 19:2691-2698.
- Penning TM, Ohnishi ST, Ohnishi T, Harvey RG. 1996. Generation of reactive oxygen species during enzymatic oxidation of polycyclic aromatic hydrocarbon trans-dihydrodiols catalyzed by dihydrodiol dehydrogenase. *Chem. Res. Toxicol.* 9:84-92.
- Perelo L. 2010. Review: In situ and bioremediation of organic pollutants in aquatic sediments. *J. Haz. Mat.* 177:81-89.
- Peterson LA, Cummings ME, Vu CC, Matter BA. 2005. Glutathione trapping to measure microsomal oxidation of furan to cis-2-butene-1,4-dial. *Drug Metab. Dispos.* 33:1453-1458.
- Peterson LA. 2006. Electrophilic intermediates produced by bioactivation of furan. *Drug Metab. Rev.* 38:615-626.
- Peterson RE, Theobald HM, Kimmel GL. 1993. Developmental and reproductive toxicity of dioxins and related compounds: cross-species comparisons. *Crit. Rev. Toxicol.* 23(3):283-335.
- Poland A, Knutson JC. 1982. 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin and related halogenated aromatic hydrocarbons: Examination of the mechanism of toxicity. *Annu. Rev. Pharmacol. Toxicol.* 22:517–524.
- Pruell RJ, Rubenstein NI, Taplin BK, LiVolsi JA, Bowen RD. 1993. Accumulation of polychlorinated organic contaminants from sediment by three benthic marine species. *Arch. Environ. Contam. Toxicol.* 24:290–300.

- Purdy JE. 1989. The effects of brief exposure to aromatic hydrocarbons on feeding and avoidance behaviour in coho salmon, *Oncorhynchus kisutch*. *J. Fish Biol.* 34:621-629
- Randall DJ, Connell DW, Yang R, Wu SS. 1998. Concentrations of persistent lipophilic compounds in fish are determined by exchange across the gills, not through the food chain. *Chemosphere.* 37(7):1263–1270.
- Rappe C, Bergqvist P, Kjeller L, Swanson, S. 1991. Levels and patterns of PCDD and PCDF contamination in fish, crabs, and lobsters from Newark Bay and the New York Bight. *Chemosphere.* 22:239.
- Rappe C. 1994. Dioxin, patterns and source identification. *Fresenius. J. Anal. Chem.* 348:63–75.
- Regoli F. 1992. Lysosomal responses as sensitive stress index in biomonitoring heavy metal pollution. *Mar. Ecol. Prog. Ser.* 84:63-69.
- Reynaud S, Deschaux P. 2006. The effects of polycyclic aromatic hydrocarbons on the immune system of fish: a review. *Aquat. Toxicol.* 77:229-238.
- Rhodes L, Gardner G, Van Beneden R. 1997. Short-term tissue distribution, depuration and possible gene expression effects of [³H]2,3,7,8-TCDD exposure in soft-shell clams (*Mya arenaria*). *Environ. Toxicol. Chem.* 16:1888–1894.
- Rogers JM, Denison MS. 2002. Analysis of the antiestrogenic activity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in human ovarian carcinoma BG-1 cells. *Mol. Pharmacol.* 61(6): 1393-1403.
- Ross PS, De Swart RL, Reijnders PJH, van Loveren H, Vos JG, Osterhaus ADME. 1995. Contaminant related suppression of delayed-type hypersensitivity and antigen responses in harbor seals fed herring from the Baltic Sea. *Environ. Health Perspect.* 103:162-167.
- Ross PS, De Swart RL, van der Vliet H, Willemsen L, deKlerk A, van Amerongen G., Groen J., Brouwer A, Schipholt I, Morse DC, van Loveren H, Osterhaus ADME, Vos JG. 1997. Impaired cellular immune response in rats exposed perinatally to Baltic Sea herring oil or 2,3,7,8-TCDD. *Arch. Toxicol.* 71:563–574.
- Rubinstein NI, Gilliam WT, Gregory NR. 1984. Dietary accumulation of PCBs from a contaminated sediment source by a demersal fish (*Leiostomus xanthurus*). *Aquat. Toxicol.* 5:331–342.
- Ruus A, Berge JA, Bergstad OA, Knutsen JA, Hylland K. 2006. Disposition of polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) in two Norwegian epibenthic marine food webs. *Chemosphere.* 62:1856-1868.
- SAB (Science Advisory Board for Contaminated Sites in British Columbia). 2004. Screening risk assessment Level 1 (SRAA) guidance. Final Draft. Prepared by Menzie Cura and Associates, Inc. Winchester, Massachusetts. Prepared for: the SAB. University of Victoria. Victoria, British Columbia.
- Safe SH. 1986. Comparative toxicology and mechanism of action of polychlorinated dibenzo-p-dioxins and dibenzofurans. *Annu. Rev. Pharmacol. Toxicol.* 26:371–399.

- Santl H, Brandsch R, Gruber L. 1994. Experimental determination of Henry's Law Constant (HLC) for some lower chlorinated dibenzodioxins. *Chemosphere*. 29(9-11): 2209-2214.
- Saucedo P, Racotta I, Villarreal H, Monteforte M. 2002. Seasonal changes in the histological and biochemical profile of the gonad, digestive gland, and muscle of the calafia mother-of-pearl oyster (*Pinctada mazatlanica*) associated with gametogenesis. *J. Shellfish Res.* 21:127-135.
- Schrap SM, Opperhuizen A. 1990. Relationship between bioavailability and hydrophobicity: reduction of the uptake of organic chemicals by fish due to the sorption on particles. *Environ. Toxicol. Chem.* 9:715-724.
- Schroeder, P. and C.K. Ziegler. (2004). Understanding, predicting and monitoring contaminant releases during dredging., Paper presented at "Addressing Uncertainty and Managing Risk at Contaminated Sediment Sites", USACE/USEPA/SMWG Joint Sediment Conference, US Army Corps of Engineers.
- Schropp SJ, Calder FD, Burney LC, Windom HL. 1989. A practical approach for assessing metals contamination in coastal sediments: An example from Tampa Bay. In: Proc. Sixth Symposium on Coastal and Ocean Management, July 11-14, 1989. American Society of Civil Engineers, Charleston, SC.
- Schropp SJ, and Windom HL. 1988. A guide to interpretation of metal concentrations in estuarine sediments. Coastal Zone Management Section, Florida Department of Environmental Regulation, Tallahassee, FL. 44 pp. + app.
- Schropp, SJ, Lewis FG, Windom HL, Ryan JD, Calder FD, Burney LC. 1990. Interpretation of metal concentrations in estuarine sediments of Florida using a LCluminum as a reference element. *Estuaries* 13(3):227-235.
- Scott JA, Hodson PV. 2008. Evidence for multiple mechanisms of toxicity in larval rainbow trout (*Oncorhynchus mykiss*) co-treated with retene and alpha-naphthoflavone. *Aquat. Toxicol.* 88(3):200-206.
- Segner H. 2006. Lessons from endocrine disruption and their application to other issues concerning trace organics in the aquatic environment. *Environ. Sci. Technol.* 40(3):1084-1085.
- Servos MR, Huestis S, Whittle DM, Munkittrick KR. 1994. Survey of receiving water environmental impacts associated with discharges from pulp mills. III. Levels of dioxins and furans in muscle and liver as measured by GC/MS. *Environ. Toxicol. Chem.* 13:1103-1115.
- Sese BT, Grant A, Reid BJ. 2009. Toxicity of polycyclic aromatic hydrocarbons to the nematode *Caenorhabditis elegans*. *J. Toxicol. Environ. Health A.* 72(19):1168-1180.
- Shiu WY, Doucette W, Gobas FAPC, Andren A, Mackay D. 1988. Physical-chemical properties of chlorinated dibenzo-p-dioxins. *Environ. Sci. Technol.* 22:651-658.
- Siu WH, Cao J, Jack RW, Wu RS, Richardson BJ, Xu L, Lam PK. 2004. Application of the comet and micronucleus assays to the detection of B[a]P genotoxicity in hemocytes of the green-lipped mussel (*Perna viridis*). *Aquat. Toxicol.* 66(4):381-392.

- Spacie A, Hamelink JL. 1995. Appendix D: bioaccumulation. In: Rand, GM. (Ed.), *Fundamentals of aquatic toxicology: effects, environmental fate, and risk assessment*. 2nd ed. Taylor & Francis, Washington, DC.
- Spitsbergen J, Walker MK, Olson JR, Peterson RE. 1991. Pathologic alterations in early life stages of lake trout, *Salvelinus namaycush*, exposed to 2, 3, 7,8-tetrachlorodibenzo-p-dioxin as fertilized eggs. *Aquat. Toxicol.* 19:41–72.
- Stantec Consulting Ltd. 2014. Re-submission fo the application for disposal at sea for the Canpotex potash export terminal, Port of Prince Rupert, British Columbia. File 1231-10264.
- Stehr CM, Brown DW, Hom T, Anulacion BF, Reichert WL, Collier TK. 2000. Exposure of juvenile Chinook and chum salmon to chemical contaminants in the Hylebos Waterway of Commencement Bay, Tacoma, Washington. 7:215-227.
- Suntio LR, Shiu WY, Mackay D. 1988. A review of the nature and properties of chemicals present in pulp mill effluents. *Chemosphere.* 17:1249–1290.
- Sweeney MH, Calvert GM, Egeland GA, Fingerhut MA, Halperin WE, Placitelli LA. 1997. Review and update of the results of the NIOSH medicals tudy of workers exposed to cheicals contaminated with 2,3,7,8-tetrachlorodibenzodioxin. *Teratogen. Carcin. Mut.* 17(4-5):241-247.
- Teraoka H, Kubota A, Dong W, Kawai Y, Yamazaki K, Mori C, Harada Y, Peterson RE, Hiraga T. 2009. Role of the cyclooxygenase 2-thromboxane pathway in 2,3,7,8-tetrachlorodibenzo-p-dioxin-induced decrease in mesencephalic vein blood flow in the zebrafish embryo. *Toxicol. Appl. Pharmacol.* 234(1):33-40.
- Teraoka H, Ogawa A, Kubota A, Stegeman JJ, Peterson RE, Hiraga T. 2010. Malformation of certain brain blood vessels caused by TCDD activation of Ahr2/Arnt1 signaling in developing zebrafish. *Aquat. Toxicol.* 99(2):241-247.
- Thoma H, Mucke W, Kauert G. 1990. Comparison of the polychlorinated dibenzo-p-dioxin and dibenzofuran in human tissue and human liver. *Chemosphere.* 20:433-442.
- Thomae TL, Stevens EA, Bradfield CA. 2005. Transforming growth factor- β 3 restores fusion in palatal shelves exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *J. Biol. Chem.* 280:12742-12746.
- Tillitt DE, Cook PM, Giesy JP, Heideman W, Peterson RE. Reproductive Impairment of Great Lakes Lake Trout by Dioxin-Like Chemicals. In: Di Giulio RT, Hinton DE, editors. *Toxicology of Fishes*. Boca Raton, Florida: CRC Press; 2008. pp. 819–875.
- Toomey BH, Bello S, Han ME, Cantrell S, Wright P, Tillitt DE, Di Giulio RT. 2006. 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) induces apoptotic cell death and cytochrome P4501A expression in developing *Fundulus heteroclitus* embryos. *Aquat. Toxicol.* 53(2):127-138.
- Turyk ME, Anderson HA, Persky VW. 2007. Relationships of thyroid hormones with polychlorinated biphenyls, dioxins, furans and DDE in adults. *Environ. Health Persp.* 115:1197-1203.
- U.S. Environmental Protection Agency. (1990) Background document to the integrated risk assessment for dioxins and furans from chlorine bleaching in pulp and paper mills.

- Washington, D.C.: U.S. Environmental Protection Agency, Office of Toxic Substances. EPA 560/5-90-014.
- USEPA (United States Environmental Protection Agency). 1989. Risk assessment guidance for superfund, volume II, environmental evaluation manual. Interim final. EPA-540/1-89/001. Office of Solid Waste and Emergency Response. Washington, District of Columbia.
- USEPA (United States Environmental Protection Agency). 1992. Framework for ecological risk assessment. EPA/630/R-92-001. Office of Solid Waste and Emergency Response. Washington, District of Columbia.
- USEPA (United States Environmental Protection Agency). 1997. Ecological risk assessment guidance for Superfund: Process for designing and conducting ecological risk assessments. Environmental Response Team. Edison, New Jersey.
- Varanasi U, Casillas E, Arkoosh MR, Hom T, Misitano DA, Brown DW, Chan S-L, Collier TK, McCain BB, Stein JE. 1993. Contaminant exposure and associated biological effects in juvenile Chinook salmon (*Oncorhynchus tshawytscha*) from urban and nonurban estuaries of Puget Sound. NOAA Technical Memorandum NMFS NWFSC-8.
- Varanasi U. 1989. Metabolism of polycyclic aromatic hydrocarbons in the aquatic environment. CRC Press, Inc., Boca Raton.
- Voie OA, Johnsen A, Rosslund HK. 2002. Why biota still accumulate high levels of PCB after removal of PCB contaminated sediments in a Norwegian Fjord. *Chemosphere*, 46:1367–1372.
- Vreugdenhil HJI, Lanting CI, Mulder PGH, Boersma ER, Weisglas-Kuperus N. 2002. Effects of prenatal PCB and dioxin background exposure on cognitive and motor abilities in Dutch children at school age. *J. Pediatr.* 140:48-56.
- Walker MK, Cook PM, Batterman AR, Butterworth BC, Berini C, Libal JJ, Hufnagle LC, Peterson RE. 1994. Translocation of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) from adult female lake trout (*Salvelinus namaycush*) to oocytes: effects on early life stage development and sac fry survival. *Can. J. Fish. Aquat. Sci.* 51:1410–1419.
- Walker MK, Hufnagle LC Jr, Clayton MK, Peterson RE. 1992. An egg injection method for assessing early life stage mortality of polychlorinated dibenzo-p-dioxins, dibenzofurans, and biphenyls in rainbow trout (*Oncorhynchus mykiss*). *Aquat. Toxicol.* 22: 15–38.
- Walker MK, Spitsbergen JM, Olson JR, Peterson RE. 1991. 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) toxicity during early life stage development of lake trout (*Salvelinus namaycush*). *Can. J. Fish. Aquat. Sci.* 48(5):875-883.
- Wan Y, Hu J, Yang M, An L, An W, Jin X, Hattori T, Itoh M. 2005. Characterization of trophic transfer for polychlorinated dibenzo-p-dioxins, dibenzofurans, non- and mono-ortho polychlorinated biphenyls in the marine food web of Bohai Bay, North China. *Environ. Sci. Technol.* 39(8):2417-2425.
- Wan Y, Jones PD, Holem RR, Khim JS, Chang G, Kay DP, Roark SA, Newsted JL, Patterson WP, Giesy JP. 2010. Bioaccumulation of polychlorinated dibenzo-p-dioxins, dibenzofurans, and dioxin-like polychlorinated biphenyls in fishes from the Tittabawassee and Saginaw Rivers, Michigan, USA. *Sci. Total Environ.* 408(11):2394-2401.

- Wannemacher R, Rebstock A, Kulzer E, Schrenk D, Bock KW. 1992. Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin on reproduction and oogenesis in zebrafish (*Brachydanio rerio*). *Chemosphere*. 24:1361–1368.
- Wasserman JC, Barros SR, Lima GBA. 2013. Planning dredging services in contaminated sediments for balanced environmental and investment costs. *Journal of Environmental Management*. 121(30): 48-56.
- Weinstein JE. 2001. Characterization of the acute toxicity of photoactivated fluoranthene to glochidia of the freshwater mussel, *Utterbackia imbecillis*. *Environ. Toxicol. Chem.* 20:412-419.
- Werth M, Russell K, Ziegler K, Stivers C. 2012. Comprehensive model of dredge resuspension and residual effects of remedial alternatives: Portland Harbor feasibility study. PIANC USA.
- Wessel N, Santos R, Menard D, Le Menach K, Buchet V, Lebayon N, Loizeau V, Burgeot T, Budzinski H, Akcha F. 2010. Relationship between PAH biotransformation as measured by biliary metabolites and EROD activity, and genotoxicity in juveniles of sole (*Solea solea*). *Mar. Environ. Res.* 69:S71-73.
- White DH, Hoffman DJ. 1995. Effects of polychlorinated dibenzo-p-dioxins and dibenzofurans on nesting wood ducks (*Aix sponsa*) and Bayou Meto, Arkansas. *Environ. Health Perspect.* 103(Suppl. 4):37-39.
- White DH, Seginak JT. 1994. Dioxins and furans linked to reproductive impairment in wood ducks at Bayou Meto, Arkansas. *J. Wildl. Manage.* 58(1):100-106.
- Wilson DM, Goldsworthy TL, Popp JA, Butterworth BE. 1992. Evaluation of genotoxicity, pathological lesions and cell proliferation in livers of rats and mice treated with furan. *Environ. Mol. Mutagen.* 19:209-222.
- Wintermyer M, Cooper K. 2003. Dioxin/furan and PCB concentrations in eastern oyster (*Crassostrea virginica*) tissues and the effects on egg fertilization and development. *J. Shellfish Res.* 22: 737–746.
- Wintermyer M, Skaidas A, Roy A, Yang Y, Georgapoulos P, Burger J, Cooper K. 2005. The development of a physiologically-based pharmacokinetic (PBPK) model using the distribution of 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) in the tissues of the eastern oyster (*Crassostrea virginica*). *Mar. Environ. Res.* 60(2):133–152.
- Wootton EC, Dyrinda EA, Pipe RK, Ratcliffe NA. 2003. Comparisons of PAH-induced immunomodulation in three bivalve molluscs. *Aquat. Toxicol.* 65(1):13-25.
- World Health Organization (WHO) (2000) Air Quality Guidelines for Europe, Second Edition. WHO Regional Publications, European Series, No. 91.
- Wu WZ, Li W, Xu Y, Wang JW. 2001. Long-term toxic impact of 2,3,7,8-tetrachlorodibenzo-p-dioxin on the reproduction, sexual differentiation, and development of different life stages of *Gobiocypris rarus* and *Daphnia magna*. *Ecotoxicol. Environ. Saf.* 48(3): 293-300.
- Yamauchi M, Kim EY, Iwata H, Shima Y, Tanabe S. 2006. Toxic effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in developing red seabream (*Pagrus major*) embryo: an

association of morphological deformities with AHR1, AHR2 and CYP1A expressions. *Aquat. Toxicol.* 80(2):166-179.

Zhou H, Wu H, Kia C, Diao X, Chen L, Xue Q. 2010. Toxicology mechanism of the persistent organic pollutants (POPs) in fish through the AhR pathway. *Toxicol. Mech. Meth.* 20:279-286.

Zoumis T, Schmidt A, Grigorova L, Calmano W. 2001. Contaminants in sediments: remobilization and demobilization. *Sci. Total Environ.* 266:195–202.