Synthesis of Toxicological Behavior of Oil Sands Process-Affected Water Constituents

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Oil Sands Research and Information Network

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REPORT SUMMARY

Large volumes of oil sands process-affected water (OSPW) are produced by the surface-mining oil sands industry in Alberta. The industry is following a no-release practice for OSPW due to its potential environmental toxicity. Both laboratory and field studies have demonstrated that OSPW is toxic to a variety of organisms including bacteria, invertebrates, fish, amphibians, birds, and mammals.

Naphthenic acids (NAs) are widely considered as the major toxic components of OSPW, exhibiting their toxic effects through multiple modes of action such as narcosis, endocrine disruption, and carcinogenicity. However, other pollutants present in OSPW, including polycyclic aromatic hydrocarbons (PAHs), benzene, toluene, ethylbenzene, and xylenes (BTEX), phenols, dissolved ions, heavy metals, and other unknown constituents may also contribute to or modify the overall OSPW toxicity. Although specific information on the toxicity of the compounds present in OSPW is limited, they have been associated with a wide range of biological dysfunctions in exposed organisms, such as mutagenicity, carcinogenicity, immunotoxicity, and endocrine disruptive effects, caused by the organics (e.g., PAHs, phenols), and ionic imbalances induced by the high levels of total dissolved solids and salts.

This report reviews the adverse effects of individual compounds, or mixtures of compounds, that are present in OSPW and/or other oil-related sources. Data on the additive, synergistic, and/or antagonistic effects caused by different constituents present in OSPW are not available, at present.

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1 INTRODUCTION

1.1 Background

The oil sands in Alberta, Canada, are large deposits of bitumen (a semi-solid form of crude oil), representing the third largest reservoir of crude oil in the world, after Venezuela and Saudi Arabia (Bari et al. 2014). Alberta's oil sands deposits contain approximately 1.8 trillion barrels of bitumen, although only around 170 billion barrels are considered as economically recoverable (Swart and Weaver 2012). These deposits are concentrated in three distinct regions: Athabasca, Cold Lake, and Peace River, covering an area of more than 142,000 km^2 (Cho et al. 2014). About 80% of crude bitumen reserves are considered recoverable by *in-situ* methods and 20% by surface mining (Bari et al. 2014). However, the bitumen recovery factors using current *in situ* methods are relatively low, with 20% to 25% for Cyclic Steam Stimulation (CSS), and up to 60% via Steam Assisted Gravity Drainage (SAGD), compared with surface mining that can achieve approximately 90% of bitumen recovery (Macadam 2013, Shah et al. 2010). The bitumen production by the Alberta's oil sands industry has reached 1.5 million barrels/day in 2010, and is estimated to grow to 3.7 million barrels/day by 2025 (Bari et al. 2014).

Surface mining is an efficient method to recover bitumen in shallow deposits lying within 75 m of the surface using a modified Clark hot water extraction process (Allen 2008a, Clemente and Fedorak 2005). In this process, the oil sands slurry is digested with hot water (79 to 93°C) with sodium hydroxide used as a process aid (Giesy et al. 2010). The elevated pH increases the solubility of asphaltic acids and naphthenic acids (NAs) promoting their release from the bitumen. These asphaltic acids and NAs act as surfactants, significantly reducing the oil-water interfacial tension, and resulting in the recovery of the bitumen (Allen 2008a, Gao et al. 2010). Consequently, the slurry separates into three distinct layers of raw bitumen (also called bitumen froth), water, and sands (Bauer 2013, Suncor Energy Inc. 2009). The bitumen is skimmed off the top to be further refined and upgraded to synthetic crude oil (Bauer 2013). Both the bitumen extraction and froth treatment processes produce tailings with approximately 4 m^3 of fluid tailings produced for each m^3 oil sands processed (Peters et al. 2007).

Generally, tailings contain approximately 70% to 80 wt% water, 20% to 30 wt% solids, and 1% to 3 wt% bitumen (Allen 2008a). The coarse fraction of solids (sands and coarse silt) quickly falls to the bottom, while the fine particles (silts and clays) settle very slowly (Farkish and Fall 2013). These fine tailings require several years to reach 30% to 40 wt% solids when the suspension is termed mature fine tailings (MFT) (Farkish and Fall 2013). Treatment methods are under investigation to accelerate the dewatering of the fine tailings, but few methods have been applied at a commercial scale (e.g., composite tailings, paste technology) (Allen 2008a). The upper layer is the clarified water that is commonly referred to as oil sands process-affected water (OSPW). About 80% to 88% of OSPW is recycled back into the extraction operations (Syncrude Canada Limited 2012); however, there are limits to recycling as water reuse causes the deterioration of water chemistry, leading to the corrosion and scaling of extraction facilities, as well as to the reduction of bitumen recovery (Allen 2008a).

OSPW is a complex mixture of unrecovered bitumen, dissolved inorganics (e.g., salts and metals) and organic compounds (e.g., NAs, polycyclic aromatic hydrocarbons (PAHs), phenols, and benzene, toluene, ethylbenzene, and xylenes (BTEX)), as reviewed by Allen (2008a). In the present review, data for OSPW chemistry were collected from previous references and unpublished work recently done by Dr. Gamal El-Din's research group; they are illustrated in Tables 1 to 3 and explained in detail in the following sections. It should be noted that the inorganic ions and metals can be analyzed quite easily with ion chromatography and inductively coupled plasma mass spectrometry (ICP-MS), respectively, while the organic fraction is difficult to fully characterize due to its highly complex mixtures of aromatic and saturated hydrocarbons, organic acids (NAs), phenols, PAHs, and heteroatom (S/N) substituted species found in OSPW[1].

Following a no-release practice, more than 10^9 m^3 of OSPW is currently stored in on-site tailings ponds, with the volumes of OSPW continuously increasing due to the ongoing expansion of oil sands operations (Energy Resources Conservation Board 2010, 2012, He et al. 2011, 2012a). These immense volumes of OSPW create multiple contamination routes including groundwater and surface water contamination and accidental or deliberate OSPW release, which may negatively affect wildlife and human health in the Athabasca region (Headley and McMartin 2004, Melvin et al. 2013). An environmental impact assessment conducted for the Frontier Oil Sands Mine Project (Silver Birch Energy Corp.) revealed that, in the local study area approximately 110 km north of Fort McMurray, the levels of several inorganic and organic contaminants in natural undisturbed water bodies were elevated due to natural background contamination (TECK 2011). For example, aluminium, iron, and chromium concentrations (1.2, 1.8, and 0.0026 mg/L, respectively) in the Athabasca Lake runoff exceeded both acute and chronic toxicity guidelines. The project assessed the predicted concentration of various inorganic and organic contaminants in the proposed end-pit lakes for the years 2068 and 2157. For example, the predicted concentration of aluminium will decrease from 1.7 mg/L to 0.52 mg/L, cadmium from 1.5 µg/L to 0.83 µg/L, and chromium from 5.2 µg/L to 2.6 µg/L by 2157. Similarly, the NA concentrations are predicted to decrease from 3.7 mg/L to 2.4 mg/L by 2157. However, these values are still above the chronic and acute toxicity guidelines, thus the OSPW would still not be released into the environment without further treatment.

To achieve the sustainability of the oil sands industry, OSPW needs to be treated to reduce its toxicity and to facilitate its reclamation for safe discharge into the receiving environment and to minimize the uptake of fresh water through recycling (Giesy et al. 2010). Currently, there are several options being considered for the treatment of OSPW, including flocculation, ozonation, advanced oxidation, membrane treatment, petroleum-coke adsorption, and biological treatment, among others, as well as the combinations of two or more treatment processes (Afzal et al. 2012,

[1] See, for example
Zhao, B., R. Currie and H. Mian, 2012. Catalogue of Analytical Methods for Naphthenic Acids Related to Oil Sands Operations. OSRIN Report No. TR-21. 65 pp. http://hdl.handle.net/10402/era.26792
Pereira, A.S. and J.W. Martin, 2014. On-Line Solid Phase Extraction – HPLC – Orbitrap Mass Spectrometry for Screening and Quantifying Targeted and Non-Targeted Analytes in Oil Sands Process-Affected Water and Natural Waters in the Athabasca Oil Sands Region. OSRIN Report No. TR-45. 33 pp.
http://hdl.handle.net/10402/era.37793

Alpatova et al. 2014, Anderson et al. 2012b, Drzewicz et a. 2012, Gamal El-Din et al. 2011, Garcia-Garcia et al. 2011a, Islam et al. 2014, Kim et al. 2012, 2013, Liang et al. 2011, Pourrezaei et al. 2011). Some of these treatments have been shown to remove some of the contaminants present in OSPW and reduce/remove OSPW toxicity, but no full-scale treatment process has been implemented for the remediation of OSPW to allow its release into the natural environment. As a prerequisite to the evaluation of OSPW treatment options, water quality and toxicological properties of OSPW need to be investigated that promote the identification of target pollutants and establishment of water treatment priorities (Allen 2008a).

Multiple studies have revealed the toxicity of OSPW with NAs widely considered as the major toxicants in OSPW. However, information for other pollutants present in OSPW, including PAHs, BTEX, phenols, dissolved ions, heavy metals, and other unknown constituents, is limited. In this review, some toxicity data for industrial wastewaters from the mining and oil industries are presented, as the wastes share similar groups of compounds that are present in OSPW. Petroleum refinery wastes originate primarily from industries refining crude oils and industries related to the manufacturing of petrochemicals, grease and lubricants, as well as fuels. These wastes are a major source of hydrocarbons, PAHs, phenols, metals and other toxic compounds that are persistent in the environment (De Jonge et al. 2013, Mrayyan and Battikhi 2005, Wake 2005). In addition, data on the assessment of the toxic effects of naturally occurring compounds as well as anthropogenic oil spills may be good indicators of the possible effects of oil and petroleum products on a given ecosystem (Hong et al. 2014, Incardona et al. 2013, Macias-Zamora et al. 2014, Peterson 2001, Turcotte et al. 2011).

In general, the exposure of organisms to petroleum-affected wastewaters (including OSPW) leads to a wide range of biological dysfunctions including neurological effects, developmental delays and deformities, impaired reproduction, suppression of immune responses, inflammation, and increased susceptibility to infectious diseases (Collier et al. 2014, Garcia-Garcia et al. 2011a,b, 2012, Leclair et al. 2013). These toxicological endpoints can be used to examine the adverse effects of xenobiotics, including both anthropogenic and natural toxic compounds. The use of model organisms greatly helps to understand and determine the possible toxic effects of contaminants on humans and animals, as many aspects of the physiology of animals such as fish, rats and mice are shared with humans due to evolutionary links. However, care must be taken when extrapolating toxicological endpoints across taxa.

1.2 Objectives

This is a review of the literature covering the toxicity of whole OSPW as well as select OSPW constituents. Literature on oil spills and wastes from oil related industry greatly helps to elucidate the toxicity of OSPW, as these sources have many compounds in common with OSPW. Groups of compounds are discussed in detail such as NAs, PAHs, BTEX, phenols, dissolved ions, and metals.

2 COMPOSITION AND TOXICITY OF THE ORGANIC FRACTION OF OIL SANDS PROCESS-AFFECTED WATER

Organic compounds detected in OSPW include unrecovered bitumen, NAs, asphaltenes, phenols, cresols, humic and fulvic acids, phthalates, BTEX, and PAHs (Allen 2008a). As shown in Table 1, OSPW contains elevated concentrations of organic compounds in terms of dissolved organic carbon (DOC), chemical oxygen demand (COD), and biochemical oxygen demand (BOD), compared to the regional freshwaters. Of significance are a group of carboxylic acids, known as NAs, that have become a great concern due to their reported toxicity to various target organisms (Garcia-Garcia et al. 2011a,b, 2012, Scarlett et al. 2013). The concentrations of NAs in OSPW range widely from 12 to 120 mg/L (Table 1) (Clemente and Fedorak 2005). Aged OSPW has relatively low concentrations of NAs, ranging from 14 to 43 mg/L (Table 1). The ambient levels of NAs in surface waters in the Athabasca oil sands region are typically less than 1 mg/L (Table 1). Recent research has provided evidence of OSPW migrations beyond containment systems and entering into the Athabasca River system (Frank et al. 2014).

Compared with NAs, there are limited data for the concentrations of other organics present in OSPW that may also contribute to OSPW toxicity. As reviewed by Allen (2008a), PAH levels in Syncrude's Mildred Lake Settling Basin (MLSB; 0.01 mg/L) have exceeded the Canadian Council of Ministers of the Environment (CCME) guidelines for the protection of aquatic ecosystems (0.01 to 0.06 μg/L for individual compounds). Fresh OSPW collected from MLSB and Suncor tailings ponds exhibited greater concentrations of phenols (0.008 to 1.8 mg/L) than aged OSPW (Syncrude demonstration ponds; 0.001 to 0.003 mg/L), with the data exceeding CCME phenol guidelines (0.004 mg/L) (Allen 2008a, CCME 1999e). Regarding BTEX, historical concentrations of benzene (<0.01 to 6.3 mg/L), toluene (<0.01 to 3 mg/L), and ethylbenzene (<0.01 to 0.14 mg/L) in OSPW have also exceeded the CCME guidelines (0.37, 0.002, and 0.09 mg/L, respectively) (Allen 2008a, CCME 1996a,b, 1999a, Suncor Energy Inc. 2009).

Multiple studies have demonstrated that OSPW is toxic to a variety of organisms, including microorganisms (Jones et al. 2011, Wang et al. 2013), aquatic invertebrates (Anderson et al. 2012a,b), fish (Heuvel et al. 2012, Kavanagh et al. 2011, 2012, 2013, Leclair et al. 2013, MacDonald et al. 2013, McNeill et al. 2012, Nero et al. 2006a,b, Peters et al. 2007, Sansom et al. 2013, Van den Heuvel et al. 2014), amphibians (Hersikorn et al. 2010, Melvin and Trudeau 2012a,b, Melvin et al. 2013, Pollet and Bendell-Young 2000, Smits et al. 2012), birds (Gentes et al. 2006, 2007a,b, Gurney et al. 2005, Harms et al. 2010) and mammals (Garcia-Garcia et al. 2011a,b, 2012, Rogers et al. 2002). NAs are believed to be the primary source of OSPW toxicity, and their chemical structures, physiochemical and toxicological properties have been fully reviewed (Clemente and Fedorak 2005, Headley et al. 2004, Kannel and Gan 2012). However, recent findings have shown that NAs account for less than 50% of the organic fraction in OSPW, which suggests that other organic compounds present in OSPW, such as PAHs and BTEX may also contribute to the toxicity (Collier et al. 2014, Grewer et al. 2010, Headley et al. 2009, Nero et al. 2006a,b, Reynaud and Deschaux 2006, Upham et al. 1994, West et al. 2014). Furthermore, elevated levels of inorganic ions in OSPW were reported to modify the toxic

properties of both organic compounds and metals, suggesting antagonistic or synergistic reactions among different compounds (Kavanagh et al. 2012, Kennedy 2012, Wurts and Perschbacher 1994).

Table 1. Organic water chemistry of oil sands process-affected waters, natural surface waters, and the Athabasca River.

Parameters [mg/L]	OSPW (active tailings ponds)	Aged OSPW (reclamation ponds)	Natural surface waters	Athabasca River water[c]
DOC	29 to 207[a,b,c]	26 to 58[d]	14 to 27[e]	10
BOD	4.6 to 13.6[a]			<2[e]
COD	173 to 670[a,b]			40
NAs	12 to 120[a,f]	14 to 43[f]	<0.5 to 0.9[g]	<1[e]

Notes:
OSPW: oil sands process-affected water; DOC: dissolved organic carbon; BOD: biochemical oxygen demand; COD: chemical oxygen demand; MFT: mature fine tailings; WIP: West In Pit; AURTP: Aurora tailings pond; SEP: South East Pond; MLSB: Mildred Lake Settling Basin; DP: Demonstration Pond.
Ranges indicate mean values for multiple sites; NAs are determined by FT-IR method.
[a] Gamal El-Din, unpublished; Sampling period: September 2010 to August 2013.
[b] Suncor Energy Inc. (2009); Suncor Energy Inc. Tailings reduction operations application. OSPW from active Suncor mine sites.
[c] Zubot et al. (2012); Syncrude WIP, and Athabasca River; Sampling period: 2006 to 2008.
[d] Siwik et al. (2000); Syncrude DP; Sampling period: 1996-1997.
[e] Allen (2008a), Golder Associates Limited (2002).
[f] Han et al. (2009); Syncrude active settling ponds (MLSB, WIP, AURTP, SEP), and experimental reclamation ponds (FE5, Pond 9, DP, CT-POND); Sampling period: December 2006.
[g] Kavanagh et al. (2009); Reference sites (Gregoire Lake, Beaver Creek and Popar Creek), Syncrude ponds (Pond 1, Pond 3, Pond 9, DP), and Suncor's MFT South pond.

2.1 Naphthenic Acids

2.1.1 *Composition and Chemical Structure of NAs*

Carboxylic acids (including NAs) are naturally occurring in petroleum and oil sands deposits, which constitute approximately 2% (by weight) of raw bitumen in the Athabasca oil sands (Kannel and Gan 2012). Classical NAs are conventionally defined as acyclic and polycyclic monocarboxylic acids with a general formula of $C_nH_{2n+z}O_2$, where n is the number of carbon atoms and z is zero or a negative even integer representing the hydrogen deficiency of the NAs resulted from the ring formation or double bonds (Headley and McMartin 2004, Rowland et al. 2011b). In addition to these classical NAs, recent reports have demonstrated the presence of oxy-NAs ($C_nH_{2n+Z}O_x$, x = 3 to 5), and heteroatoms containing NAs ($C_nH_{2n+z}SO_3$; $C_nH_{2n+z}NO_x$,

x = 1 to 3) in the Athabasca oil sands (Barrow et al. 2010, Grewer et al. 2010, Han et al. 2009, Kannel and Gan 2012). As well, some classical NAs have been characterized to include tri-, tetra- and pentacyclic diamondoid acids, diamantine methyl and dimethyl diamantine acids, aromatic carboxylic acids, estrogen-like steroidal acids, and acids with ethanoic acid side chains (Headley et al. 2009, Rowland et al. 2011a,b,c,d,e).

Different sources of oil worldwide exhibit varying compositions of NA species. For example, 60% of the acidic components of California crude oil samples from the San Joaquin Valley contain two or more oxygens. Compounds containing only oxygen heteroatoms accounted for less than 10% of the acidic compounds identified. Approximately one-half of the species contained nitrogen and about one-fourth contained sulfur (Tomczyk and Winans 2001). A family of C_{80} isoprenoid carboxylic acids were identified in samples from oilfield deposits in Great Britain, the Norwegian continental shelf, UK, China and West Africa (Kannel and Gan 2012). Also of interest are some NAs that were shown to have more than one -COOH, such as the $C_{80}H_{138}O_8$ with four -COOH functional groups (Kannel and Gan 2012).

NAs recovered from petroleum have been refined to produce commercial NAs (C-NAs) of varying purities and compositions, depending on the petroleum sources and the refining techniques (Clemente and Fedorak 2005). In general, C-NAs have smaller carbon number (n) ranges of $7 \leq n \leq 17$, as compared to those in petroleum and oil sands that have broader n ranges, extending in some cases from 7 to 40 and even to greater than 80 (Scott et al. 2005, Tollefsen et al. 2012). NAs have surfactant-like structures containing both hydrophobic alkyl groups and hydrophilic carboxylic moieties, suggesting their water solubilities are pH-dependent (Kannel and Gan 2012). During the Clark extraction process, NAs present in alkaline OSPW exist primarily in ionized forms that are thought to be primarily responsible for the water toxicity (Kannel and Gan 2012, Kavanagh et al. 2011).

2.1.2 *Toxicity of NAs*

Most toxicological data for NAs have been generated for aquatic species, although some microbial, avian, and mammalian toxic effects have also been documented. The interpretation of specific toxicological results should be made with caution, since NA composition and toxicity are known to be source-specific (Garcia-Garcia et al. 2012, Grewer et al. 2010, Kindzierski et al. 2012, Tollefsen et al. 2012). For example, C-NAs were found to be more toxic than oil sands-derived NAs (Armstrong et al. 2008, 2010, Scott et al. 2005), which was likely due to the lower molecular weight components present in C-NAs that are presumably more interactive with biological systems (Clemente and Fedorak 2005).

The surfactant-like structures of NAs enable them to act as necrosis agents by disrupting membranes via insertion into the lipid bilayer, causing increased membrane fluidity and cell death (Frank et al. 2008, Klopman et al. 1999, Konemann 1981). Recent studies also suggest that both C-NA and petroleum or oil sands NAs may affect the endocrine systems by modulating sex steroid production and acting as weak estrogen receptor (ER) agonists and androgen receptor (AR) antagonists (Rowland et al. 2011b, Scarlett et al. 2012, Thomas et al. 2009). As well, the carcinogenic potential of NAs, particularly those containing one aromatic ring, has been

predicted using the ADMET™ predictor (Scarlett et al. 2012). To determine how NAs interact with organisms on the cellular level, Zhang et al. (2011a) assessed the genomic response of *Escherichia coli* to NAs. The authors reported the up-regulation of genes involved with NADP or NADPH, and down-regulation of genes involved in ATP binding.

Although the specific mechanism(s) of NA toxicity have yet to be fully elucidated, it is becoming increasingly clear that NAs toxicity may have multiple modes of action. The following is a synopsis of toxicity of NA mixtures and NA-contaminated wastewater samples, including OSPW, on different organisms reported to date.

2.1.2.1 Bacteria (*Vibrio fischeri*)

The Microtox® bioassay using the marine bacterium *V. fischeri* has been used as a quick assessment tool for the determination of the acute toxicity of NAs. Toxicity is often expressed as effective concentration causing a 50% suppression of bioluminescence (EC_{50}) in the bacterium. Using Microtox®, Frank et al. (2008) demonstrated an inverse relationship between OSPW-derived NA fractions toxicity and NA molecular weights, as evidenced by EC_{50} values for NAs having $\geq$ 22 carbons of ~ 64.9 mg/L and smaller NA molecules having < 22 carbons of 41.9 to 58.1 mg/L. These results are in agreement with the findings that C_{22+} NAs are the least toxic fractions of OSPW NA mixtures (Holowenko et al. 2002). Presumably, the increased -COOH content in higher molecular weight NAs resulted in decreased hydrophobicity and, consequently, reduced the narcotic potency of NAs (Frank et al. 2009). Structure-specific toxicity of NAs was also reported by Jones et al. (2011), where the EC_{50} values of pure individual NAs were found to vary with NA structures. The NAs with greater alkyl chain length displayed enhanced toxicity.

2.1.2.2 Aquatic Invertebrates

The majority of studies that examined NA toxicity in aquatic invertebrates have been conducted using OSPW. *Daphnia magna* (cladoceran freshwater water flea) exposed to OSPW displayed a 96 h-EC_{50} value of 10% v/v (MacKinnon and Boerger 1986), and *Chironomus dilutus* (nonbiting midges, formerly known as *C. tentans*) exposed to OSPW had a 14-d LC_{50} value of 71% v/v (Whelly 1999); however, the concentrations and composition of NAs were not specified in these studies. With an assumed NA concentration of 80 mg/L in OSPW, and a *D. magna* LC_{50} of 76% to 98% OSPW, the equivalent LC_{50} for NAs would range from 61 to 78 mg/L (Armstrong et al. 2008, 2010, CEATAG 1998). Similarly, the LC_{50} of NAs to *C. dilutus* was estimated to be 52 to 65 mg/L (Whelly 1999).

Midges of the genus *Chironomus* comprise a large proportion of the biomass of aquatic ecosystems in the Athabasca oil sands region (Bendell-Young et al. 2000), indicating they can be potentially important targets of toxic effects of OSPW. Studies have shown that exposure to OSPW impaired the growth and development (reduced larvae mass, less pupation, and decreased emergence rate) of *C. dilutus* larvae (Anderson et al. 2012a,b, Wiseman et al. 2013). The suppressed larval growth might be due to oxidative stress and disruption of endocrine processes, demonstrated by the alterations in expression of genes involved in related pathways (Wiseman et al. 2013). Oxidative stress could initiate caspase-independent apoptosis (programmed cell

death), leading to the release of apoptosis-inducing factors (AIFs) from the inner mitochondrial membrane and translocation to the nucleus where AIFs mediate chromatin condensation and degradation (Cande et al. 2002, Delavallee et al. 2011). Endocrine disruption was indicated by the alterations in abundances of transcripts of steroid hormone receptors (i.e., ultraspiricle protein, ecysteroid receptor and estrogen related receptor) that are involved in mediating hormone actions (Wiseman et al. 2013). While NAs were correlated strongly with toxic endpoints (e.g., lesser mass, pupation, and emergence), some metals (e.g., Ni, Mn, and U) present in OSPW may also contribute to these effects (Anderson et al. 2012a).

Lower toxicity was observed in reclaimed OSPW compared to fresh OSPW, which was correlated with lower concentrations of NAs found in reclaimed OSPW (Anderson et al. 2012a). This reduced toxicity can be attributed to the biodegradation of NAs, effectively reducing their concentration to non-toxic levels, and/or metabolized parent NAs, resulting in the generation of less toxic by-products (Wiseman et al. 2013). However, the detoxification of OSPW through *in situ* biodegradation is a slow and incomplete process (Anderson et al. 2012a, Han et al. 2008, 2009, Leung et al. 2001), possibly due to the presence of recalcitrant NAs. For example, Han et al. (2009) reported that while the saturated groups of NAs were most easily degraded, the more cyclic/unsaturated forms were quite difficult to biodegrade.

The toxicological characteristics of wetlands constructed in mined-out pits as a method for *in situ* reclamation of OSPW have been examined. These OSPW-impacted wetlands exhibited decreased invertebrate community diversity (Bendell-Young et al. 2000, Whelly 1999), an increase in densities of opportunistic invertebrate *Chironomus spp*. (Bendell-Young et al. 2000), and inhibition of growth of *C. riparius* larvae (Kennedy 2012).

It should be noted that the NAs are not the only contaminants that contribute to the toxicity of OSPW. When reared in water mimicking combinations of salts and NAs, *C. riparius* survival was significantly negatively correlated with salt and NA concentrations, and there was an antagonistic interaction between these two toxicants (Kennedy 2012). Importantly, species-specific toxic effects have also been reported (MacKinnon and Boerger 1986, Toor et al. 2013). For example, MacKinnon and Boerger (1986) found that OSPW was more toxic to *D. magna*, than fish (trout), and least toxic to *V. fischeri*, suggesting that the toxic effects of OSPW or NAs may be species-specific and that the effects observed in prokaryotic organisms may not be applicable to eukaryotic organisms, including higher vertebrates. This finding also suggests that it is important to test multiple species and trophic levels to determine the OSPW toxicity.

2.1.2.3 Fish

A number of studies have demonstrated the toxic effects of NAs towards a variety of fish species. In several studies, C-NAs have been used for toxicity assessments as representatives of oil sands or petroleum-derived NAs. The exposure of freshwater fish to relatively high concentrations of C-NAs caused mortality in fish with 96-h LC_{50} values ranging from 25 to 75 mg/L, depending on fish species and age (Dokholyan and Magomedov 1983). Recently, it has been reported that exposure of early life stages of fish (yellow perch *Perca flavescens* and Japanese medaka *Oryzias latipes*) to C-NAs caused increased embryo deformities and reduced

length at hatch (Peters et al. 2007). Exposure of the same species to OSPW caused lower toxicity than would have been predicted, based on the concentrations of NAs, when compared to that caused by the C-NAs (Peters et al. 2007). In contrast, another study reported higher toxicity of an OSPW-derived NA fraction versus C-NAs, where 100% mortality of yellow perch fingerlings was observed at concentrations of 3.6 mg/L and 6.8 mg/L, respectively (Nero et al. 2006a). It should be noted that the OSPW-derived NA fraction also included other potentially toxic organic compounds. A recent study examined the effects of aromatic NAs extracted from OSPW showing higher toxicity for zebrafish (*Danio rerio*) from aromatic versus alicyclic NAs (Scarlett et al. 2013). These results suggest that the toxicological effects induced by NAs are both OSPW source-specific and NA composition and structure-specific.

The toxic effects of NAs on fish immune systems have been demonstrated by alterations in gene expression of pro-inflammatory cytokines and ability to control parasites of goldfish (*Carassius auratus*) exposed to C-NAs (Hagen et al. 2012), and reduced blood leukocytes and spleen thrombocytes in rainbow trout (*Oncorhynchus mykiss*) injected intraperitoneally with OSPW derived NAs (MacDonald et al. 2013). As well, OSPW has been shown to affect fish host immune responses, as evidenced by the increased prevalence of opportunistic diseases including lymphocystis and severe fin erosion in yellow perch (Palmer et al. 2012, Van den Heuvel et al. 2000), decreased total leukocyte numbers, and reduced antibody production against the pathogenic bacterium *Aeromonas salmonicida* in rainbow trout (McNeill et al. 2012). While the exact causative agent responsible for the alteration of fish immunity after OSPW exposure is unknown, there appears to be a correlation between the immune system dysfunction and the presence of NAs (McNeill et al. 2012). Leclair et al. (2013) found that immune system disruption caused by OSPW-derived NAs was lower than that seen in fish exposed to the whole OSPW, suggesting that in addition to NAs, other compounds in OSPW contributed to the observed immunotoxicity.

A number of studies have examined the toxic effects of whole OSPW exposure in fish. Exposure of early life stages of fathead minnow (*Pimephales promelas*), yellow perch and Japanese medaka to OSPW increased deformities (e.g., hemorrhage, pericardial edema, malformation of the spine), mortality, premature hatching, and abundances of transcripts associated with oxidative stress (He et al. 2012a, Peters et al. 2007, Siwik et al. 2000). Oxidative stress can result in damage to mitochondria and promote activation of caspase enzymes and apoptotic cell death (He et al. 2012a). Larval malformations (e.g., edemas, hemorrhages and skeletal, craniofacial and eye defects) and mortality were also reported in fish exposed to sediments from the oil sands region and wastewater ponds. The observed mortality and eye pathology were related to CYP1A which is a protein that can be induced by PAHs (Colavecchia et al. 2004, 2006, 2007). Due to the chemical complexity of OSPW sediments, other compounds besides PAHs (such as hydrocarbon degradation products, NAs, and metals) are present in the sediments and may also contribute to overall toxicity.

Consistent with results obtained from the Microtox® assay, acute toxicity of fresh OSPW declined over time under natural conditions likely due to a decrease in the proportion of lower molecular weight NAs (Holowenko et al. 2002, MacKinnon and Boerger 1986). However, aged

OSPW still induced toxic effects manifested by altered hepatic mixed-function oxygenase (MFO) activity and blood cell composition. In addition, histological changes in organs (liver, gill and fin), viral tumors, and impairment of the immune responses in different fish species, have been reported (Farrell et al. 2004, Heuvel et al. 2012, McNeill et al. 2012, Peters et al. 2007).

C-NAs and NAs in waters from offshore oil platforms have been reported to be weak ER agonists and potent AR antagonists (Thomas et al. 2009). The presence of OSPW-derived NAs with structural similarities to estradiol has been reported (Rowland et al. 2011b), and other research predicted that some polycyclic NAs with a single aromatic ring may induce estrogenic and androgenic activity in humans (Scarlett et al. 2012). These observations suggest that some OSPW NAs may act as endocrine disrupting compounds (EDCs) that are capable of disrupting synthesis, secretion, transport, binding, and/or elimination of steroids in organisms. The hormones and steroids that may be affected, such as estradiol (E2) or testosterone (T) and/or their receptors, play roles in maintenance of homeostasis, reproduction development, and behavior of organisms (Government of Canada 1999).

OSPW has exhibited endocrine disruptive effects on fish, evidenced by reduced plasma levels of testosterone, estradiol, or cortisol (Lister et al. 2008), and decreased *in vitro* production of sex hormones by fish gonadal tissues (Lister et al. 2008, Tetreault et al. 2003). These disruptions have been attributed to alterations to both reproductive and glucocorticoid hormone biosynthesis (Lister et al. 2008). In contrast, He et al. (2011) observed no effects of OSPW on the glucocorticoid receptor signaling pathway. Interestingly, while decreased E2 levels were observed in fish, elevated E2 production was observed in H295R cells exposed to OSPW that was associated with increased aromatase activity and decreased E2 metabolism (He et al. 2010). Other studies showed no effects on either aromatase gene expression and activity or E2 metabolism in fish exposed to OSPW (Lister et al. 2008, Van den Heuvel et al. 1999). The contradictory findings on EDC effects of OSPW may be due to different chemistry of test waters samples (e.g., different NA profiles), and the difference of specificity and sensitivity of endocrine disruptive properties between fish and human cell lines. Another study has shown that OSPW exposure caused altered abundances of transcripts of regulatory genes in all tissues of the brain-gonad-liver (BGL) in fathead minnows (He et al. 2012b), which may be correlated to the impaired reproductive capacity of fathead minnows (e.g., less fecundity and second sex characteristics, and altered sex steroids production) (Kavanagh et al. 2011, 2012). The toxicity of NAs could be modified by the high concentration of salts found in wastewaters such as OSPW, which is discussed in the section on salts toxicity.

2.1.2.4 Amphibians

Amphibian larvae are extremely vulnerable and sensitive to contaminants in aquatic environments, thus increased efforts have been made to investigate the impact of OSPW and NAs on the growth, development, metabolism, and physiological processes in tadpoles (Melvin et al. 2013). C-NAs are acutely toxic to tadpoles (96-h LC_{50} = 3.04 to 4.76 mg/L for northern leopard frogs (*Lithobates pipiens*)), and cause significant reduction in growth and development,

as well as increased deformities at relatively low C-NA concentrations (2 to 4 mg/L for *Lithobates pipiens* and tropical clawed frogs (*Silurana tropicalis*) after 9- and 3-d exposure, respectively) (Melvin and Trudeau 2012a,b). *L. pipiens* tadpoles exposed to C-NAs (1 to 2 mg/L) during their development experienced delayed growth, as well as disruption in normal physiological functions (Melvin et al. 2013). Interestingly, the exposure of adult frogs (*L. pipiens*) to higher concentrations of C-NAs (20 and 40 mg/L) under saline conditions caused minor toxicological effects (Smits et al. 2012). It appears that the differences in sensitivity in response to a xenobiotic stress may be related to the developmental stage of the amphibian. It should be noted that the concentrations of C-NAs which produced toxic effects in tadpoles were well below typical NAs concentrations found in whole OSPW and are similar to levels identified in groundwater samples taken in the oil sands region. Although C-NAs are not necessarily an adequate surrogate for the OSPW-derived NAs (Garcia-Garcia et al. 2012), these results suggest that exposure to NAs at environmentally relevant concentrations may adversely impact tadpole populations.

The toxicity of water from OSPW-impacted wetlands towards amphibians has also been examined. Higher mortality, stunted growth and development, as well as alterations in hormone production were observed in tadpoles of western toad (*Bufo boreas)* and wood frog (*Lithobates sylvaticus*), where water from recently constructed wetlands induced higher toxic effects compared to that from aged wetlands (Hersikorn and Smits 2011, Hersikorn et al. 2010, Pollet and Bendell-Young 2000). These findings are in agreement with those obtained when aquatic invertebrates and fish were exposed to water from aged constructed wetlands, suggesting that partial detoxification of OSPW may be possible over time due to *in situ* microbial degradation and photolysis of contaminants such as NAs and PAHs (Hersikorn and Smits 2011, Lai et al. 1996).

2.1.2.5 Birds

The tree swallow (*Tachycineta bicolor*) is an indicator species for the evaluation of environmental health of wetlands. Reduced reproductive performance of the tree swallow and increased mortality of nestlings in OSPW-impacted wetlands were observed during harsh weather conditions (Gentes et al. 2006). When the weather was less challenging, the mortality rates were lower; however, lower weight and higher hepatic ethoxyresorufin O-deethylase (EROD) activity of nestlings were still observed (Gentes et al. 2006). Similar increase in EROD activity was previously documented in nestlings exposed to impacted wetlands (Smits et al. 2000). EROD activity is normally measured to indicate the presence of chemicals that could induce hepatic detoxification enzymes in wastewaters. Custer et al. (2001) and Bishop et al. (1999) demonstrated an EROD induction in tree swallows exposed to PAHs, and Rogers et al. (2002) suggested that the increased liver weights of rats exposed to OSPW derived NAs might be associated with the induction of hepatic detoxification enzymes. Gentes et al. (2007a) found higher production of thyroid hormones in tree swallow nestlings living in reclaimed wetlands, although this was not directly attributable to NAs. Altered thyroid function may negatively affect the metabolism, behavior, feather development, and molting, which compromises post fledging survival (Gentes et al. 2007a). Gentes et al. (2007b) found that tree swallow nestlings

could tolerate short-term exposures to environmentally relevant concentrations of NAs. However, the effects of chronic exposures to NAs have yet to be evaluated.

Toxic effects of OSPW towards other avian species have also been examined. Zebra finches (*Taeniopygia guttata*) orally exposed to OSPW showed no decrease in T-lymphocyte numbers but exhibited enlarged Bursa of Fabricius which is a primary lymphoid organ for the differentiation and amplification of antibody-producing B lymphocytes (Smits and Williams 1999). Lower body mass and skeletal size were also observed in mallard (*Anas platyrhynchos*) ducklings living in OSPW-impacted wetlands, influencing the survival of juvenile waterfowl amongst other possible adverse effects (Gurney et al. 2005).

2.1.2.6 Mammals

As compared to aquatic organisms, information about the toxicity of NAs in mammals is limited. The oral LD_{50} values of C-NAs for rats ranged 3 to 5.2 g/kg bw (body weight), with death caused by gastrointestinal disturbances (Lewis 2000) with similar results at 3.55 g/kg bw in mice (CEATAG 1998). When intraperitoneally exposed to NAs, the LD_{50} value was 0.86 g/kg bw in mice, with animals exhibiting depressed central nervous system, convulsions, and respiratory arrest (CEATAG 1998).

Female rats acutely exposed (14 d) to high-dose (300 mg/kg bw) OSPW NA extracts experienced increased ovarian and spleen mass with more prevalent liver and heart damage, while males displayed increased mass of testes and hearts as well as brain hemorrhage (Rogers et al. 2002). However, since the high dose used in these studies was approximately 50 times likely a "worst-case single-day acute exposure scenario for wild animals", this concentration would not be found in the natural environment. In a sub chronic (90 d) toxicity assessment, rats fed 60 mg/kg bw/d showed higher liver mass and increased liver glycogen storage, indicating that the liver was a target organ for NA toxicity (Rogers et al. 2003). It was also demonstrated that exposure to OSPW-derived NAs induced impaired embryonic implantation, which was likely associated with the changes in cholesterol availability and a resultant decrease in progesterone synthesis (Rogers 2003). Recently, research using mice found that the OSPW organic fraction (OSPW-OF) had immunotoxic properties both *in vitro* and *in vivo*, affecting various macrophage microbicidal functions and immune gene expression in different organs (Garcia-Garcia et al. 2011a,b). Additionally, different gene expression profiles observed in mice exposed to C-NAs compared to those exposed to OSPW-OF has further supported the viewpoint that C-NAs are not good surrogates for NAs and other organic species in OSPW (Garcia-Garcia et al. 2012).

2.2 Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs are a large group of organic compounds with two to eight conjugated ring systems. They can have a range of substituents such as alkyl, nitro, and amino groups in their structure (Fieser and Fieser 1956). Nitrogen, sulfur, and oxygen atoms can also be incorporated into their ring system (Fieser and Fieser 1956, McElroy et al. 1985). The precursors for PAHs found in crude oil are natural products, such as steroids, that have been chemically converted to aromatic hydrocarbons over time (Feng et al. 2009). These compounds are ranked ninth on the list of

hazardous substances by the U.S. Agency for Toxic Substances and Disease Registry (ATSDR 2011). They are released to the environment through both natural and anthropogenic pathways (Kelly et al. 2009, Collier et al. 2014), and are divided into petrogenic and pyrogenic PAHs. Petrogenic PAHs are present in oil and some oil products, and are introduced into aquatic systems from natural oil seepages, the petroleum industry, and oil sands processing. Wang et al. (2003) analyzed the composition and properties of spilled oils, fuels and petroleum products, demonstrating that the petrogenic PAHs consist of 85% or more alkyl congeners. Pyrogenic PAHs are formed by incomplete combustion of organic material and are composed of larger ring systems than the petrogenic PAHs. Sources for pyrogenic PAHs are forest fires, incomplete combustion of fossil fuels and tobacco smoke. PAHs are present in natural oil sands, in oil sands tailings pond sediments, and in surface waters from tributaries of the Athabasca River (Collier et al. 2014, Headley and Akre 2001, Tetreault et al. 2003). Although the specific toxicity of PAHs derived from oil sands has not received significant attention, research on the toxic effects on fish of individual PAHs, PAH mixtures, crude oils, PAH-contaminated sediments, and waters has shown that PAHs possess mutagenic, carcinogenic, immunotoxic, and endocrine disrupting properties, as reviewed by Collier et al. (2014). PAH exposure also elicits deleterious effects on mammals, as demonstrated in studies using different model compounds such as naphthalene, benzo[a]pyrene (BaP), 3-methylcholanthrene (3-MC) and 7,12-dimethylbenz[a]anthracene (DMBA). However, model PAHs are inadequate surrogates for PAHs in the environment, where they are found in complex mixtures containing many PAHs with different chemical, physical and toxic characteristics.

2.2.1 *Toxicity of PAHs in Fish*

2.2.1.1 Neoplasms and Lesions

Liver tumors or neoplasms, toxicopathic hepatic lesions, and skin tumors have been observed in wild fish collected from habitats characterized by elevated concentrations of PAHs (Baumann and Harshbarger 1998, Leadly et al. 1999, Logan 2007, Myers et al. 1998, Pinkney and Harshbarger 2006, Schiewe et al. 1991, Solt et al. 1977, Vogelbein and Unger 2006, Vogelbein et al. 2008). Many studies have demonstrated that the prevalence and intensity of liver tumors or lesions was related to PAH concentrations in sediments (Baumann and Harshbarger 1998, Leadly et al. 1999, Pinkney and Harshbarger 2006, Vogelbein et al. 2008), and it has been suggested that the prevalence of liver tumors could be used as an indicator of PAH exposure (Logan 2007).

Other biomarkers used to indicate the degree of fish exposure to PAHs include the activity of enzymes such as CYP1A and glutathione S-transferase (GST), the presence of PAH metabolites in bile, and PAH-DNA adducts in tissues (Colavecchia et al. 2007, Peltonen and Dipple 1995, Pinkney et al. 2004). CYP1A is involved in metabolizing PAHs (Colavecchia et al. 2007), and GST plays a role in eliminating hydroxylated PAH intermediates in bile (Pinkney et al. 2004). The transformation process of PAHs mediated by the CYP1 family generates reactive intermediates that bind to DNA to form PAH-DNA adducts (Myers et al. 1998, Solt et al. 1997). As reviewed by Collier et al. (2014), field studies have shown an overall positive relationship

between the occurrence of liver tumors or lesions and PAHs exposure as measured by these biomarkers.

2.2.1.2 PAHs and Compromised Immunity

The immunotoxic effects of fish exposures to PAHs have been previously reviewed by Reynaud and Deschaux (2006). A compromised immune system can result in an increase in susceptibility to different diseases (Jacobson et al. 2003), and consequently threaten the survival of exposed organisms (Loge et al. 2005, Spromberg and Meador 2005). PAH exposures can increase fish susceptibility to pathogenic bacteria (e.g., *Aeromonas salmonicida, Yersinia ruckeri,* and *Listonella anguillarum*) (Arkoosh et al. 2001, Bravo et al. 2011, Carlson et al. 2002) or parasites (e.g., *Cryptocotyle lingua*) (Khan 2003). However, increased resistance to bacteria *A. salmonicida*, or the digestive tract parasite *Steringophorus furciger* in fish exposed to PAHs were also reported (Hogan et al. 2010, Khan 2003). It is interesting that when zebrafish were simultaneously exposed to phenanthrene and *Mycobacterium marinum*, the survival was greater than that of fish exposed to phenanthrene alone (Prosser et al. 2011). This may be explained by the fact that the pro-inflammatory cytokines induced by bacteria inhibited the CYP1A biotransformation of PAHs to more toxic chemicals (Morgan 2001, Prosser et al. 2011).

Studies have shown that fish exposure to PAHs affects their innate and adaptive immune responses. Changes in both lysozyme concentrations and haemolytic alternative complement activity have been observed in *in vitro* studies of European sea bass (*Dicentrarchus labrax*) exposed to various PAHs (Bado-Nilles et al. 2009), while in Dab (*Limanda limanda*) exposed to sediments spiked with PAHs, lysozyme concentrations were similar to non-exposed fish (Hutchinson et al. 2003). Studies have demonstrated that exposure of fish to individual or mixtures of PAHs, or PAH-contaminated sediments caused enhanced apoptosis of lymphocytes and phagocytes, and inhibition of phagocytosis (i.e., reduction in phagocytic capacity, chemotaxis, and respiratory burst activity) (Carlson et al. 2002, Hutchinson et al. 2003, Reynaud and Deschaux 2005, Rice and Schlenk 1995, Weeks and Warinner 1986, Weeks et al. 1986). In contrast, other reports found that exposure of fish to PAHs actually enhanced macrophage oxidative functions (Kelly-Reay and Weeks-Perkins 1994, Reynaud 2002, Reynaud et al. 2001). The apparent differences in immune responses of fish exposed to PAHs may be due to the different fish species used, exposure routes, doses, and compositions of PAHs tested. Phagocytes are involved in both innate and adaptive immune responses. The pattern recognition proteins (PRPs) on microbes, or "non-self" host tissues, can be recognized by pattern recognition receptors (PRRs) on macrophages, which trigger phagocytosis and lead to activation of adaptive or specific immunity (Collier et al. 2014, Janeway et al. 2005, Magnadottir 2010).

The impacts of PAH exposure on fish adaptive immunity have been studied by examining the ability of lymphocytes to proliferate primarily using mitogen-based assays. Both increased or suppressed proliferation of T and B cells and reduced total white blood cell numbers have been observed in several fish species exposed to PAHs (individual or PAH mixtures) (Carlson et al. 2002, 2004a,b, Reynaud and Deschaux 2005, Reynaud et al. 2001, 2003). In addition to the studies using individual or mixtures of model PAHs, immunotoxic effects on fish were also

reported after exposure of fish to petroleum products, oil-production wastewaters, and water-soluble oil fractions, with the effects being fish species- and PAH composition-specific (Danion et al. 2011a,b, Karrow et al. 2001, Perez-Casanova et al. 2010, 2012, Song et al. 2012a,b).

The mechanisms underlying the immunotoxicity of PAHs in fish have not been fully elucidated. It has been suggested that PAHs may directly affect the primary immune organs and cells of the immune system (Reynaud and Deschaux 2006), or interact with the aryl-hydrocarbon receptor (AhR) in fish (e.g., rainbow trout) leukocytes (Nakayama et al. 2008). Other studies have shown that PAHs can directly modulate intracellular Ca^{2+} homeostasis causing the inhibition of calcium-dependent immune responses (Reynaud and Deschaux 2005, Reynaud et al. 2003).

2.2.1.3 Endocrine Disrupting Effects

PAHs have been shown to disrupt fish endocrine systems. Exposure to individual PAHs, mixtures of PAHs, and PAH-contaminated sediments caused reduction in plasma sex steroid levels in female fish (Monteiro et al. 2000a, Pollino et al. 2009, Sol et al. 2000, Thomas 1988, Tintos et al. 2006, 2007) and steroids in ovarian tissues (Monteiro et al. 2000b, Tetreault et al. 2003, Thomas and Budiantara 1995). Alterations in reproductive hormones were associated with suppressed vitellogenin (VTG) production, inhibited gonadal and oocyte development, and reduced gonadosomatic index (GSI) (Khan 2013, Logan 2007, Nicolas 1999, Pait and Nelson 2009). Interestingly, slimy sculpin (*Cottus cognatus*) collected in Steepbank River within the oil sands deposit, where PAHs were present in sediments, experienced decreased *in vitro* synthesis of steroid hormones by the ovarian tissue, but the alterations in gonadal development were not consistently observed (Tetreault et al. 2003).

Previous studies on the endocrine disrupting effects of PAHs in male fish have been inconclusive. Decreased plasma T and 11-KT levels, increased T metabolism, reduced GSI and impaired testicular development were reported in male fish exposed to PAHs of varying sources including crude oils and PAH-contaminated sediments/waters (Idler et al. 1995, Khan 2013, Martin-Skilton et al. 2006, 2008, Pait and Nelson 2009, Truscott et al. 1992). As well, a significant negative correlation between fish GSI and PAH concentrations was documented (Pait and Nelson 2009). However, in a different fish species (English sole, *Parophrys vetulus*) exposed to PAH-contaminated sediments there was no correlation between PAH exposure and either 11-KT concentrations or gonad weight (Sol et al. 2008). In contrast to the decreased steroid production in response to PAH exposure in many studies, increases in both plasma testosterone (T) concentrations and *in vitro* T production were observed in several fish species exposed to model PAHs (e.g., naphthalene, BaP and retene) as reviewed by Collier et al. (2014). These conflicting observations may be due to the differences in the duration of exposure, composition and concentrations of PAHs, fish species and life stages used, as well as the presence of other contaminants that can also impact fish health.

PAHs may act as EDCs through several potential mechanisms. For example, PAHs were found to inhibit the enzymatic activities involved in the synthesis of androgens (CYP17, CYP11β) and estrogens (CYP19) in male and female carp *Cyprinus carpio* (Fernandes and Porte 2013). PAH binding to AhR may stimulate the activity of CYP1A, in some cases together with CYP1C,

which is primarily responsible for E2 metabolism, causing alterations in blood E2 levels (Scornaienchi et al. 2010, Spink et al. 1990). PAHs could also exert estrogenic effects by interacting with the estrogen receptor α (ERα), which may affect ERα-mediated processes such as vitellogenesis (Ohtake et al. 2007, Ota et al. 2000, Swedenborg et al. 2012). Other reports showed that PAHs may affect the endocrine process by altering the levels of brain monoaminergic neurotransmitters such as serotonin that play a role in the modulation of reproduction in fish (Gesto et al. 2006, 2008, 2009, Rahman et al. 2011).

2.2.1.4 PAH Effects on Reproduction and Development

Fish at early life stages are highly sensitive to PAH exposure. Fish exposed to PAHs (natural oil sands sediments, tailings ponds sediments, crude oil, or individual PAHs) have had increased embryo and larval mortality (Carls et al. 1999, Colavecchia et al. 2004, 2007, Incardona et al. 2012, Marty et al. 1997a,b), and exhibited reduced larval growth and premature hatching (Carls et al. 1999, Colavecchia et al. 2006, Leung and Bulkley 1979, Parrott et al. 2011, Rhodes et al. 2005). However, the PAH effects on both premature and delayed hatching in fish are dependent on the oil source and dose (Colavecchia et al. 2004, Linden 1978). Other embryo-larval toxic effects observed in fish exposed to oil sands or crude oils include reduced growth and larval malformations such as edemas (pericardial, yolk sac, and subepidermal), hemorrhages, and spinal, craniofacial, and eye defects (Carls et al. 2008, Colavecchia et al. 2004, 2006, 2007, Couillard 2002, Incardona et al. 2005, McIntosh et al. 2010, Pollino and Holdway 2002). Some signs of these toxic effects resemble those observed in fish exposed to toxic AhR-binding compounds, such as 2,3,7,8 tetrachlorodibenzo-*p*-dioxin (TCDD) (Elonen et al. 1998, Guiney et al. 1997), retene (Billard et al. 1999, Brinkworth et al. 2003), and alkyl-PAH mixtures in crude oil (Carls et al. 1999, Couillard 2002, Marty et al. 1997a,b). In addition, in some cases these effects were paralleled by an increased CYP1A induction (Billard et al. 1999, Brinkworth et al. 2003, Colavecchia et al. 2007). The mechanism(s) of actions underlying the toxicity of AhR-binding chemicals may involve CYP1A induction, oxidative stress, and endothelial cell damage (Bauder et al. 2005, Colavecchia et al. 2007).

Tricyclic PAHs (i.e., fluorine, dibenzothiophene, and phenanthrene) were found to be the major contributors to the toxicity of petrogenic PAHs (Incardona et al. 2004, 2005, 2006, 2011, Scott et al. 2011). Dibenzothiophene and phenanthrene have been shown to exert direct effects on cardiac function in zebrafish that may lead to the secondary consequences for late stages of cardiac morphogenesis, kidney development, neural tube structure, and formation of the craniofacial skeleton (Billard et al. 1999, Incardona et al. 2004, 2005). The fractions of these tricyclic PAHs increase in crude oil from weathering, particularly the alkylated derivatives that would become dominant compounds (Carls et al. 1999, Heintz et al. 1999, Marty et al. 1997b). High proportions of alkylated tricyclic PAHs have also been detected in sediments from oil sands and tailings ponds (Colavecchia et al. 2006).

It is worth noting that some 3 to 5 ring PAHs have photo-enhanced toxicity to aquatic organisms (Oris and Giesy 1985). The photoactivated PAHs, induced by UV light, can transfer energy through to biomolecules resulting in damage to the structure and function of proteins and lipids

(Giesy et al. 2010). As reported by Giesy et al. (2010), some PAHs can become as much as 50,000-fold more toxic in the presence of ambient solar light. Therefore, although OSPW contains small concentration of PAHs, their photodynamic potential must be considered in the overall evaluation of their toxicity.

Multigenerational effects have been observed in fish exposed to PAHs, as evidenced by reduced hatching frequency of eggs produced by F1 female fathead minnows, and decreased F2 larvae survival, caused by F0 exposure to BaP (White et al. 1999). Reports also suggest that fish may adapt to PAH exposure, developing resistance to the teratogenicity and lethality caused by PAHs, and that this resistance may be partially inheritable (Meyer and Giulio 2002, 2003, Wills et al. 2010, Wirgin and Waldman 2004).

2.2.2 *Effects of PAH Exposure in Mammals*

The deleterious effects of PAHs on mammalian health have been well documented. The lethal effects of some model PAHs (naphthalene, phenanthrene, anthracene, fluoranthene, pyrene, and BaP) have been determined (Bamforth and Singleton 2005). Compounds with more benzene rings appeared to be more toxic than those with lower number of rings, as indicated by LD_{50} values that generally decreased with the increasing number of aromatic rings (Bamforth and Singleton 2005). However, data should be examined giving consideration to the exposure routes. For example, 4-ring pyrene and 5-ring BaP have LD_{50} values of 514 and 232 mg/kg bw in mice, respectively, through intraperitoneal administration, while the intravenous exposure of 4-ring fluoranthene indicated an LD_{50} of 100 mg/kg (Bamforth and Singleton 2005).

PAHs have been shown to produce carcinogenic, mutagenic, neurotoxic, immunotoxic, and endocrine disruptive effects on mammals (Bamforth and Singleton 2005, Cheng et al. 2013, Juhasz and Naidu 2000, Kummer et al. 2008, Niu et al. 2010, Thurmond et al. 1988, Xia et al. 2011). As one of the most potent carcinogens (IARC 2013), BaP has been shown to induce tumours and carcinomas (e.g., stomach tumours, mammary tumours, hepatomas and lung adenomas, mammary and uterine carcinomas) in mammals (e.g., mice, rats, rabbits) through different exposure routes (Collins et al. 1991, Juhasz and Naidu 2000). Other PAHs including DMBA, 3-MC, dibenzo[def,p]chrysene (DBC), benzo[b]fluoranthene (BbF), and dibenz[a,h]anthracene (DB[a,h]A) have been shown to be carcinogenic in mice, causing tumors in the liver, lung, breast, ovaries and hematopoietic tissue (Kim et al. 2011a, Malik et al. 2013, Siddens et al. 2012). It has been shown that PAHs can exert toxicity, including carcinogenesis, by altering the expression of AhR-regulated genes such as CYP1A1 and CYP1B1 (Andrysik et al. 2011, Siddens et al. 2012), and induction of oxidative stress (Kumar et al. 2012). It should be noted that PAH toxicity is structure-specific, evidenced by the greater carcinogenic potency of DB[a,h]A than BaP (Okona-Mensah et al. 2005), which is most likely due to the higher affinity of DB[a,h]A for AhR (Machala et al. 2001).

PAH exposure has been shown to alter the nervous system function in mice and rats, which leads to a range of developmental disorders or behavioural deficits such as decreased motor activity, poor learning and memory, and physiological and autonomic abnormalities (Bouayed et al. 2009a,b, Cheng et al. 2013, Grova et al. 2007, Qiu et al. 2011, Sram et al. 1999). The

neurotoxicity was associated with PAH-induced imbalances in neurotransmitters that play key roles in the differentiation and survival of neurons, behavior, cognition, memory and learning (Angoa-Perez et al. 2012, Bouayed et al. 2009a,b, Cheng et al. 2013, Cunha et al. 2009, 2010, Luo et al. 2011, Myhrer 2003, Toyo-oka et al. 2003, Xia et al. 2011).

Immunotoxicity of PAHs in mammals has been reported, evidenced by the alterations in antibody response, suppressed T cell proliferation, and generation of cytotoxic T cells (Davila et al. 1995, 1996, Smialowicz et al. 1997, Thurmond et al. 1988). Exposure to PAHs has also negatively affected reproductive and endocrine systems in mice or rats, manifested by the infertility and disruption in estrogenic responses (Kummer et al. 2008, MacKenzie and Angevine 1981, Mattison et al. 1982).

The toxicity of industrial and municipal wastewaters containing PAHs has been examined in several studies. Male mice chronically exposed to municipal wastewater effluent experienced liver and kidney pathologies, disruptions in multiple metabolic pathways, and alterations in signaling pathways leading to cellular responses including inflammation and immune deficiency syndromes (Zhang et al. 2013). PAHs, phthalic acid esters (PAEs), and organochlorine pesticides (OCPs), detected in these effluents have been shown to induce changes in immune responses and/or modulate endocrine system, all of which may contribute to the combined toxicity of wastewater (Ansar 2000, Kojima et al. 2003, Nilsson 2000, Van Grevenynghe et al. 2003). Mice gavaged with an organic fraction containing PAHs and other organics extracted from wastewater-irrigated soil experienced DNA damage in white blood cells (Gao et al. 2013). This toxicity may be partially attributed to the PAH exposure, based on the findings in other reports that DNA damage such as DNA breakage and DNA protein cross-links were caused by PAH exposure (Gao et al. 2013, Popp et al. 1997).

Mouse bone marrow cells exposed to coking wastewater displayed significantly increased micronucleum frequencies in a concentration-dependent manner, which was suggested to be primarily due to PAHs and phenolic species (Zhu et al. 2013). High-boiling petroleum substances that contain a wide variety of PAHs have induced toxic effects in mice or rats including death, decreased body weight, altered serum chemistry, and histopathological changes in organs (Roth et al. 2013). Furthermore, research using mammals has demonstrated that PAH exposure can cause epigenetic modifications to somatic- and germ-cell lines, which may affect their offspring (Collier et al. 2014).

To date, the specific toxicity of OSPW-derived PAHs to mammals has not been examined. However, considering the potential for photoenhanced toxicity (Giesy et al. 2010), the previously documented toxic effects of PAHs on fish and mammals (Collier et al. 2014, Logan 2007), and the potential for biomagnification in the food chain (due to their high lipid solubility that enables PAHs to be rapidly absorbed into gastrointestinal tract) (Bamforth and Singleton 2005), further research on mammalian toxicity of PAHs derived from OSPW or oil sands-related sediments is required. Llobet et al. (2006) investigated PAHs uptake through a specific seafood diet finding that the intake was age and sex dependent between 3.3 and 5.3 ng/kg/day. This intake would be associated with a $0.27/10^6$ increase in the risk of development of cancer over a 70-year life span which would be considered as acceptable being below $1/10^6$. Also, Wang et al. (2010)

investigated the bioaccessability of PAHs through fish consumption. Low molecular weight PAHs had a higher bioaccessability than high molecular weight PAHs and relative bioaccumulation factors for 2 and 3 ring PAHs were > 1, indicating relative accumulation during digestion.

2.3 BTEX and Phenols

2.3.1 *Toxicity of BTEX*

BTEX is an acronym for benzene, toluene, ethylbenzene, and xylenes. They are aromatic hydrocarbons containing one unsubstituted or methyl-substituted benzene ring. BTEX have been recently considered as one of the most common and serious threats to groundwater reservoirs because of their relatively high water solubility and known toxicity. These compounds frequently co-occur at numerous hazardous waste sites, including areas used for petroleum operations and refineries (including oil sands tailings ponds). The bitumen separated from the oil sands is quite viscous (200 to 1200 Pa.s), so naphtha (major component is BTEX) is often used as a diluent solvent for further upgrading bitumen to synthetic crude oil (Siddique et al. 2007). Most of the diluent is recovered, but a small fraction (<1%) of naphtha is lost to the tailings waste (Siddique et al. 2007).

There is little literature data available regarding the toxicity of "whole" mixtures of BTEX. As with PAHs, the mixtures of these aromatic hydrocarbons may differ from their individual toxicity through antagonistic, additive, and synergistic effects. The gene expression of stress related proteins and reactive oxygen species (ROS) showed that the combined effect of benzene, toluene and xylene was less toxic to *Drosophila melanogaster* than the effect of benzene alone, while toluene and xylene in the absence of benzene showed increased toxicity (Singh et al. 2010). Therefore, toluene and xylene may be antagonistic to benzene, but synergistic in combination. Xylene in combination with toluene produced more toxic effects in *Chironomus plumosus* larvae than ethylbenzene alone (Li et al. 2013).

In fish, 15.2 ppm of BTEX produced 100% mortality in rainbow trout (*Oncorhynchus mykiss*) after one hour of exposure, as a result of liver damage, adverse effects on blood clotting enzymes and red blood cell depletion (Zbanyszek and Smith 1984). Reduced hematopoietic tissues have also been observed in juvenile striped bass (*Morone saxatilis*) as a result of exposure to benzene for 30 days (Taberski 1982). Sub-lethal concentrations of zinc and benzene led to decreased lymphocytes in striped bass after nematode infection and eventually altered immunoglobulin production (Sakanari et al. 1984). Exposure to BTEX led to cellular necrosis, potentially due to the modification of fatty acids in the lipid bilayer (Di Marzio et al. 2001).

Apart from the immunotoxic effects of BTEX, these compounds also exhibit endocrine disrupting effects. After exposure to xylene, fish cell CYP1A gene expression and EROD activity was shown to decrease (Della Torre et al. 2011). BTEX concentrations in OSPW are low such that no reported significant direct impacts on toxicity to aquatic organisms have been reported. In addition, the aerobic and anaerobic degradation of BTEX generally leads to complete mineralization, thus there are normally no additional problems caused by toxic

metabolites. However, synergistic effects of BTEX compounds both within the group and with other organic/inorganic compounds may occur. As a result, further study on this group is still needed.

2.3.2 *Toxicity of Phenols*

Phenolic compounds, including phenol and its derivatives, are aromatic molecules containing hydroxyl, methyl, amide or sulfonic groups attached to a benzenoid ring structure (Huang et al. 2007). These compounds are commonly present in wastewaters from industries such as resin manufacturing, petrochemical, oil refineries, coking, and oil sands mining (Allen 2008a, Kavitha and Palanivelu 2004). In wastewater from the petroleum/oil industry, concentrations of phenolic compounds range widely from 2.5 to 80 mg/L (Campos et al. 2002, Hansen and Davies 1994, Jou and Huang 2003, Knight et al. 1999), while their levels in OSPW are much lower, ranging from 1 μg/L to 1.8 mg/L (Allen 2008a). Phenols can exert toxicity at low concentrations with many of them classified as hazardous pollutants because of their potential harm to human health (Huang et al. 2007, Pardeshi and Patil 2008). The concentrations of phenols in OSPW, in many cases, exceed the CCME guidelines for the protection of aquatic life (0.004 mg/L) and/or the maximum daily discharge limit of 1 mg/L under Alberta's *Environmental Protection and Enhancement Act* (EPEA) (Allen 2008a). Thus, OSPW cannot be discharged unless treated to meet these regulatory standards. As well, its seepage into the environment may pose a significant threat to aquatic biota with potentially negative impacts of phenols on higher organisms (including humans) due to their potential for bioaccumulation.

Alkylated phenols or alkylphenols (APs) are natural components of crude oil and are present in the aqueous phase after water/oil separation due to their high water solubility (Meier et al. 2007a). These compounds have been detected in produced water (PW; by-product of offshore oil production) (Meier et al. 2007a,b) and OSPW (Hargesheimer et al. 1984). APs have been identified as EDCs, possibly expressing their activity either by binding to nuclear receptors such as ER and AR, or by interacting with plasma transport proteins for sex steroids (Fang et al. 2003, Tollefsen 2007, Tollefsen and Nilsen 2008). Some studies have shown high sensitivity of fish e.g., Atlantic cod to APs exposures. Oral administration of low doses (≤ 20 μg APs/kg bw) of a mixture of four APs (with differing chain lengths of C4 to C7) resulted in induced VTG production in males, decreased levels of sex steroids in the blood of both males (11-KT) and females (E2), and delayed ovary and testes development (Meier 2007, Meier et al. 2007b, Sundt et al. 2009). A follow-up study conducted by Meier et al. (2011) demonstrated that juvenile females exposed to AP mixtures reached puberty and maturation more rapidly, while gonad development was delayed in both maturing females and males at low concentrations of APs (4 μg APs/kg bw). Similarly, other reports demonstrated the reduction of GSI in fish exposed to xenoestrogens such as high molecular weight APs, and industrial and municipal effluents, which were associated with inhibited spermatogenesis (Hemming et al. 2001, Jobling et al. 1996, Sepulveda et al. 2003). In contrast, Tollefsen et al. (2011) observed no negative effects on GSI in Atlantic cod exposed to AP mixtures or AP-containing oils. Differences in the results are likely due to variable exposure routes, composition and concentrations of APs tested, as well as the life stages of fish used.

Phenolic compounds have been shown to impact CYP enzymes. Fish exposed to APs and PAHs, experienced an up-regulation of CYP1A (Knag and Taugbol 2013, Meier et al. 2010). This may be due to the presence of PAHs that can bind to AhR and translocate the AhR to the nucleus where the transcription of responsive genes may be initiated (Knag and Taugbol 2013). In contrast, other reports demonstrated down-regulation of CYP1A and/or CYP3A in various fish species exposed to APs (Arukwe et al. 1997, Hasselberg et al. 2004b, Navas and Segner 2001), which may be associated with the anti AhR/CYP1A effects caused by APs (Sturve et al. 2006). Both CYP1A and CYP3A are involved in the metabolism of various xenobiotics (Sturve et al. 2006). Down-regulated expression of CYP genes or inhibition of CYP enzymes activity may impair the cells' ability to detoxify foreign harmful compounds (Sturve et al. 2006).

Phenolic compounds may impair the immune systems of fish, as evidenced by decreased phagocytic activity and phagocytic index in Nile tilapia (*Oreochromis niloticus*) (Soliman et al. 2002) and grass carp (*Ctenopharyngodon idella*) (Soliman 1997) exposed to sub-lethal doses of phenol, which may lead to reduced tolerance to invading pathogens. APs have also been shown to induce oxidative stress in fish, demonstrated by increased hepatic total glutathione and elevated activity of glutathione reductase in Atlantic cod exposed to AP mixtures (Hasselberg et al. 2004a). Another report demonstrated depletion of glutathione levels in cod, caused by nonylphenol exposure (Sturve et al. 2006). Glutathione is an important antioxidant and a decreased concentration of this compound may make cells more vulnerable to reactive oxygen species, potentially causing damages such as DNA fragmentation and induced apoptosis (Halliwell and Gutteridge 2007, Higuchi 2004).

Given that APs have been shown to accumulate in the tissue of fish (Wenzel et al. 2004) and molluscs (Ozaki and Baba 2003), they may impose toxic effects on higher organisms, including mammals, through bioaccumulation. Exposure of mice to phenol has been shown to cause morphological and structural changes in placental tissue (Monfared et al. 2013) and fetal resorption (Jones-Price et al. 1983). Other reports also demonstrated reproductive system disruptions of both female (e.g., accelerated vaginal opening and disrupted estrous cycle) and male (e.g., decreased testicular and epididymal masses, and reduced epididymal sperm counts) rats exposed to nonylphenol (Chitra et al. 2002, Hossaini et al. 2001, Lee 1998). The impaired male reproductive system was due to the induction of oxidative stress (Chitra et al. 2002). Furthermore, multiple studies have shown that many phenolic compounds trigger mutagenic and carcinogenic activities in mammals, resulting in skin tumours, skin cancer and epithelium cancer, papillomas in stomach, and lung cancer (as reviewed by Michalowicz and Duda 2007).

To date, information on the assessment of the toxicity of OSPW-derived phenolic compounds is limited. APs comprise a minor portion of some petroleum derived NA extracts and the toxicity attributed previously to NAs may, in part, be due to these compounds (West et al. 2011). Although known to be present in OSPW (Hargesheimer et al. 1984), APs were not initially detected by GC × GC-ToF-MS analysis, possibly because they were not extracted into the NA fraction using the extraction procedures (Frank et al. 2006, Scarlett et al. 2012). Recently, the effects of six APs and 54 NAs previously identified in OSPW were modeled for a range of environmental and human toxicity related endpoints using ADMET^{TM} predictor software. APs

were predicted to have a slightly greater tendency to bioaccumulate as compared to NAs, and were predicted to disrupt diagnostic liver enzymes including lactate dehydrogenase (by iso-propylphenols) and gamma glutamyl transpeptidase (by all APs) (Scarlett et al. 2012). Some APs were also predicted to bind to ER and AR, disrupt reproductive development, and be substrates for CYPs that are involved in the detoxification of many xenobiotics (Scarlett et al. 2012). These results indicate that, although phenols and APs (both known and unknown) are found at much lower concentrations than NAs (considered as the primary OSPW toxicants), they may still contribute to the toxicity of various wastewaters including OSPW.

3 COMPOSITION AND TOXICITY OF THE INORGANIC FRACTION OF OIL SANDS PROCESS-AFFECTED WATER

3.1 Metals

Metals information in Table 2 corresponds to that used in previous environmental impact assessments to describe tailings water quality at active Suncor mine sites (Suncor Energy Inc. 2009). OSPW contains a variety of metals including heavy metals that have shown toxicity to various organisms. Heavy metals are conservative in nature; as a result, they bioaccumulate and remain in the environment. Bioaccumulation occurs within a trophic level with metal concentrations increasing in certain tissues of the organism due to absorption from food and the environment. Most of the heavy metals are soluble in water in one form or another, thus, are readily absorbed by fish and other aquatic organisms. Some plants and animals are able to regulate their metal content up to a certain extent; however, metals that cannot be excreted from the body keep accumulating in an organism over its lifetime. Biomagnification occurs when the concentration of a substance in an organism exceeds the background concentration of the substance in its diet. This increase can occur as a result of persistence, food chain energetics and low or non-existent rate of internal degradation or excretion of the substance (Wright and Welbourn 2002). Given this biomagnification, organisms at the highest trophic levels generally have the highest concentrations of heavy metals (Atchison et al. 1987).

Heavy metals enter the environment through various natural and anthropogenic sources. Natural sources include rocks, soils, groundwaters, and surface waters. For example, the deep groundwater of Bangladesh and West Bengal is contaminated with arsenic (As), which is a serious health hazard leading to acute poisoning (Harvey et al. 2005) when untreated water is consumed. There are elevated As and uranium levels in the Beaver River Watershed (Alberta Health and Wellness 2000, Lemay et al. 2005, Moncur et al. 2011) and As levels of up to 145 µg/L in the Cold Lake and Beaver River aquifers in Alberta (Alberta Environment 2006).

Table 2. Characteristics and toxicity risk of metals in oil sands process-affected waters.

Metal	Atomic number	Weight (u)	MP [°C]	Density [g cm^{-3}]	CCME ST/LT [µg L-1]	Concentration in OSPW[a] Total Metals [µg L^{-1}]	Dissolved Metals [µg L^{-1}]
Aluminium, Al	13	26.98	660	2.7	5 to 100 (HD)	70 to 99,000	<1 to 3,860
Arsenic, As	33	74.92	615*	5.73	5	<1 to 33	1 to 28
Barium, Ba	56	137.99	727	3.5	NRG	7 to 250	20 to 550
Cadmium, Cd	48	112.41	321	8.65	1/0.09	<0.5 to 200	<0.2 to 10
Cobalt, Co	27	58.93	1,495	8.9	2.5	<1 to 570	<0.2 to 23
Chromium, Cr	24	51.99	1,857	7.19	NRG; (Cr(VI) - 1 Cr(III) - 8.9)	<2 to 400	<1 to 46
Copper, Cu	29	63.55	1,083	8.96	2 to 4 (HD)	2 to 840	<0.2 to 13
Iron, Fe	26	55.85	1,538	7.9	300	100 to 34,600	<10 to 830
Mercury, Hg	80	200.59	-39	13.55	0.026	<0.02 to 0.17	
Magnesium, Mg	12	24.31	650	1.7	NRG	2,000 to 33,000	
Manganese, Mn	25	54.94	1,244	7.21	NRG/50 (CWQG)	10 to 480	<4 to 3,610
Molybdenium, Mo	42	59.94	2,617	10.28	73	2 to 1,120	58 to 2,120
Nickel, Ni	28	58.69	1,453	8.91	25 to 150 (HD)	14 to 270	<0.5 to 66
Lead, Pb	82	207.2	328	11.34	1 to 7 (HD)	1 to 226	<0.3 to 40
Antimony, Sb	51	121.76	630	6.7	NRG	1.2 to 12	<0.2 to 7
Selenium, Se	34	78.96	217	4.3 to 4.8	1	<1 to 40	<0.2 to <70
Strontium, Sr	38	87.62	777	2.6	NRG[#]	200 to 500	60 to 1,560
Uranium, U	92	238.03	1,132	19.1	33/15		<0.2 to <500
Vanadium, V	23	50.94	1,910	6	NRG	1 to 800	1 to 1,720
Zinc, Zn	30	65.39	420	7.14	30	1 to 1,240	<0.6 to 130

MP – Melting Point
ST/LT – Short-Term/Long-Term
NRG – No recommended guideline
CWQG – Canadian Water Quality Guidelines
HD – Hardness dependant
[a] Suncor Energy Inc. (2009); Suncor Energy Inc. Tailings reduction operations application. OSPW from active Suncor mine sites.
*Sublimation Temperature
US EPA drinking water upper limit of 4 mg/L (Qu et al. 2012)
"-" = not applicable

Anthropogenic sources from agricultural and industrial activities include fertilizers, pesticides, sewage effluents, biosolids, mining tailings, coal and petroleum combustion, and solid waste disposal. The history of anthropogenic sources of heavy metals in the environment can be seen within glacial core samples in Greenland and can be traced back as far as the Bronze Age and Iron Age (Hong et al. 1996). Anthropogenic activities add significantly more heavy metals to the environment compared to natural processes (Arctic Monitoring and Assessment Programme 2005), with industrial development contributing to heavy metal accumulation in local waters (Atchison et al. 1987), especially in the developing countries due to improper treatment of heavy metal emissions sources. Anthropogenic emissions in Asia are several orders of magnitude higher than those of Europe, North America and Australia (Shahidul and Tanaka 2004). In addition, aquatic ecosystems are adversely affected by the elevated levels of metals that can influence nutrient cycles, energy flow, primary and secondary production, and decomposition of organic matter (Batty and Halberg 2010, Stoertz et al. 2002, Wolkersdorfer 2004). The specific mechanism(s) of how metals affect all these processes is a huge topic and is beyond the scope of this review.

3.1.1 *Toxicity of Metals*

Heavy metals may induce neurotoxic effects, immune system suppression, and fetal abnormalities for mammals. However, not all heavy metals are toxic as some are only toxic in certain oxidation states (e.g., Cr (III) is less toxic than Cr (VI)), while others exhibit toxicity only at high concentrations (Anderson 1981). Heavy metals like mercury and lead are highly toxic, even at low concentrations (Kelly et al. 2010). Heavy metal toxicity is dependent on various factors including pH, temperature, ionic strength, and source of contamination. For example, a higher rate of mercury accumulation has been observed in fish during summer than in winter as a result of their higher metabolic rates at higher temperatures (Wright and Welbourn 2002). Heavy metals may affect the reproduction and population dynamics of fish. For example, the tolerance of fathead minnow embryos towards other toxins decreases in the presence of high concentrations of copper (Kelly et al. 2010).

3.1.1.1 Aluminium

Aluminium (Al) has the atomic number 13, an atomic weight of 26.98, a melting point of 660.3 ^{o}C and a density of 2.7 g cm^{-3}. Al has a strong affinity for oxygen and readily forms oxides or silicates, so is never found in its elemental state. It is a major component in a number of minerals such as feldspars, micas, and clays. Its various oxidation states are I, II, and III. Al has been used in the production of automobiles, cans, foil, windows, doors, cooking utensils, walking poles, and laptop heat sinks and casings.

Investigation of Al toxicity is generally lacking for aquatic environments; however, it has been determined that fish are more sensitive than aquatic invertebrates to Al (Sparling et al. 1997). Al causes toxicity in adult fish by causing ion regulatory and respiratory effects in the gills (Gensemer and Playle 1999). Al has been found to have severe toxic effects on the central nervous systems of humans and laboratory rats (Dave et al. 2002, Sharma and Mishra 2006,

Zatta et al. 1999). Additionally, for humans, it has been related to various neurological disorders such as dialysis syndrome, Parkinson's disease, amyotrophic lateral sclerosis and Alzheimer's disease (Miu et al. 2003, Platt et al. 2007, Sharma and Mishra 2006).

The source of Al in OSPW includes alum used for the coagulation/flocculation process for the remediation of OSPW (Pourrezaei et al. 2011). Al concentrations in Suncor OSPW range from <1 µg/L to 99,000 µg/L (Table 2), and similar concentrations have been observed in Albian and Syncrude tailing ponds (Gibson et al. 2011), with the values in many cases exceeding CCME guidelines (5 µg/L to 100 µg/L; pH dependant). Consequently, the presence of Al in OSPW may contribute to the overall OSPW toxicity.

3.1.1.2 Arsenic

Arsenic (As) has the atomic number 33, an atomic weight of 74.92, a density of 5.727 g cm^{-3} and a sublimation temperature of 615 ^{o}C (Smedley et al. 2002). Elemental As is obtained from the reduction of As oxides. Arsenic forms stable compounds by covalently bonding with most non-metals and metals and exists in the environment as arsenious acids (H_3AsO_3, $H_3AsO_3^{2-}$), arsenic acids (H_3AsO_4, $H_3AsO_4^{2-}$), arsenates, arsenites, and methylarsenic acids. Anthropogenic sources of As include pesticides, mining, smelting and coal combustion. Arsenic ingested via drinking water leads to skin, bladder, lung, and kidney cancers, changes in the skin pigmentation and thickness (hyperkeratosis), and neurological disorders (Jain and Ali 2000, Mandal and Suzuki 2002). The presence of As in natural waters is a global problem with 21 countries known to have As groundwater contamination. Of these countries, Bangladesh and West Bengal in India are considered to have the largest risk of As exposure to humans (Das et al. 1995, Jain and Ali 2000, Rahman et al. 2002).

Trivalent arsenic (III) is predominant in reducing conditions and is very mobile in groundwater and sediments; therefore it is considered to be the most toxic form compared with its pentavalent form As (V) (Harrington et al. 1998). As (III) and As (V) have high affinities for lipids, proteins, and other cellular components so it readily accumulates in tissues (Ferguson and Gavis 1972). Arsenic uptake in aquatic invertebrates including mussels, crustaceans and molluscs is directly proportional to arsenic concentrations in sediment (Ferguson and Gavis 1972, Maeda et al. 1990, Tamaki and Frankenberger 1992), while fish apparently do not bioaccumulate arsenic (Tanner and Clayton 1990).

The concentrations of As in OSPW range from <1 µg/L to 33 µg/L (Table 2) indicating higher levels than recommended by CCME guidelines (5 µg/L) in some water samples. Therefore, the presence of As may contribute towards the overall OSPW toxicity.

3.1.1.3 Barium

Barium (Ba) has the atomic number 56, an atomic weight of 137.33, a melting point of 727 ^{o}C and a density of 3.5 g cm^{-3}. It is a soft, silvery white metal and is very reactive, so it is never found as a free element in nature. Various sources of Ba includes vacuum tubes, steel and cast iron manufacturing, oil well drilling, electroceramics, rodenticides, textile and paper industries (Purdey 2004), and precipitation of barium sulfate in boilers (Wang et al. 2006).

Soluble Ba salts are harmful to a variety of cellular and developmental processes in animals and plants; for example, the inhibition of ciliary function in trochophores and protozoans (Jacobs et al. 2002, Llugany et al. 2000, Purdey 2004). These effects have been suggested to result from Ba interaction with cellular calcium homeostasis, where membrane polarity and other calcium dependent processes get perturbed as barium moves more easily than calcium through the voltage dependent calcium channels (Spangenberg and Cherr 1996). Low concentrations of Ba ions in blood have been shown to act as a muscle stimulant; however, high concentrations affect the nervous system causing weakness, anxiety and cardiac irregularities. Ingestion of soluble acid salts of Ba ($BaCO_3$ or $BaCl_2$) usually causes intoxication (Dallas and Williams 2011, Dietz et al. 1992, Johnson and VanTassell 1991, Kuperman et al. 2006). The kidneys seem to be the most sensitive endpoint of chronic soluble barium ingestion (Dallas and Williams 2011).

The data for Ba toxicity to non-humans and related water quality guidelines are limited. However, it has been indicated that exposure to Ba may pose risks to aquatic organisms (daphnids), while fish appear to be at a lesser risk (Wishart et al. 2006). Ba concentrations in OSPW range widely from 7 µg/L to 550 µg/L (Table 2), and further work is needed to investigate the potential role of Ba in OSPW toxicity.

3.1.1.4 Cadmium

Cadmium (Cd) has the atomic number 48, an atomic weight of 112.4, a melting point of 321 ^{o}C and a density of 8.65 g cm^{-3}. Cd is mostly divalent in stable compounds and forms complex ions and hydroxides with ammonia and cyanide. It is present in soil and sediments, but the amount of Cd recovered as organic Cd compounds is insignificant (Hickey and Kittrick 1984).

Anthropogenic activities such as mining and smelting contribute the most to bioavailable Cd soil contamination (Asami et al. 1995, Ramos et al. 1994, Xian 1989), mainly present as free Cd^{2+} and $CdHCO_3^{+}$ (Hirsch and Banin 1990). Additional anthropogenic sources of Cd include refining of Cd and Zn ores, phosphate fertilizers (Cd containing ores are used for production), and land application of municipal sewage sludge. Cd is used in alloys, electroplating, batteries, as a stabilizer for polyvinyl plastics, and for protecting iron and steel from corrosion (Gaur et al. 2014). The use of Cd has been restricted worldwide due to its extensive environmental impacts. Fresh water normally contains 10 to 500 ng/L of dissolved Cd; however, industrialized regions can have >17 mg/L of Cd (Jones 2001, Murphy et al. 1978).

The divalent Cd^{2+} ion is the most bioavailable and the most toxic Cd form with exposures inducing the synthesis of metallothionein that can bind with Cd and reduce its toxicity. This reaction takes place in the liver of fish and humans. However, if the concentration of Cd is too high, there is over production of metallothionein that may cause systemic damage to organisms (Bradl 2005, Montaser et al. 2010, Wright and Welbourn 2002). Cd poisoning leads to skeletal deformities and kidney malfunction in fish and humans. Skeletal deformities further impair the ability of fish to search for food and avoid predators. Chloride ions help reduce Cd toxicity through combining with Cd to form insoluble $CdCl_2$ (Bradl 2005, Wright and Welbourn 2002). Cd toxicity is also dependent on the type of organism, e.g., fathead minnows are more sensitive

to metals than goldfish, potentially due to the fact that goldfish can excrete Cd via their gills (Landis and Yu 2003). Marine crustaceans that have a hepatopancreas (combined liver and pancreas) show cellular damage upon prolonged Cd exposure. Also, sublethal exposure to Cd from the surrounding water during embryonic and larval development of marine isopods reduced survival rates (Bradl 2005, Wright and Welbourn 2002). Cd leads to developmental abnormalities, slow growth, and premature hatching in teleosts (e.g., carps (*Cyprinus carpio*), eels (*Anguilla anguilla*), cod (*Gadus morhua*), and perches (*Perca flavescens*)). Cd has also been demonstrated to have endocrine disrupting abilities (e.g., low level of plasma T4 and impaired secretion of cortisol) (Hontela et al. 1996, Jones 2001).

One of the most severe Cd poisoning incidents happened near Fuchu, Japan where untreated effluents from mines containing Cd were used to water rice fields (Nordberg 2009). The rice plants in the paddies absorbed the Cd where it was bioconcentrated to toxic levels. The local population was using this rice as part of their daily diet leading to a daily intake of Cd between 600 and 1,000 µg, while the recommended intake should not exceed 300 µg. The high doses of Cd led to Itai-Itai disease with symptoms including skeletal deformities, kidney malfunction, and severe pain in the joints and spine. Cd blocks vitamin D synthesis which is necessary for calcium deposition in the bones leading to brittle and soft bones (osteoporosis and osteomalacia diseases). Cd inhibits kidney enzymes that are required for reabsorption of blood cells, glucose and proteins back into the blood stream. It inhibits estrogen receptors, disrupts the secretion of growth hormones (Jones 2001), and alters iodine metabolism leading to inhibition of thyroid hormones. Cd has been classified as a Category I (human) carcinogen by the International Agency for Research on Cancer (Bradl 2005, Wright and Welbourn 2002). Cd is a strong mutagen, which can cause multi-locus deletions (Gaur et al. 2014).

The 96-h LC_{50} values for rainbow trout (*Oncorhynchus mykiss*) and grass carp (*Ctenopharyngodon idellus*) have been found to be 0.47 µg/L and 0.1 µg/L, respectively (CCME 2014). There has been evidence that linked the presence of Cd to fish egg swelling due to excessive water influx (Jezierska et al. 2009). Cd is also known to disrupt estrogen receptors (Le Guevel et al. 2000), inhibit growth hormone (Jones 2001), and thyroid hormone synthesis in fish by modifying iodine metabolism (Chaurasia et al. 1996). Cd concentrations in OSPW range from <0.2 µg/L to 200 µg/L (Table 2), with values in many cases exceeding the recommended CCME guidelines (1 µg/L). As well, it has been shown that it would take 60 to 90 years to eliminate 87.5% of a given Cd concentration from the human body (Landis and Yu 2003). Consequently, the discharge of OSPW to the environment may pose a health hazard for both aquatic organisms and humans due to the potential exposure to Cd.

3.1.1.5 Cobalt

Cobalt (Co) has the atomic number 27, an atomic weight of 58.93, a melting point of 1,495 ^{o}C and a density of 8.9 g cm^{-3}. It has been used in the manufacture of steels, magnetic alloys, and superalloys. Other uses include as a glass decolourizer and as a pigment in paints and varnishes. (Younis 2011). Cobalt compounds were used to stabilize beer foam in Canada in the 1960s that

resulted in a peculiar form of toxin-induced drinker's cardiomyopathy (Barceloux 1999, Morin et al. 1969).

Co is a vital constituent of cobalamine (vitamin B_{12}) which is a required vitamin for animals (Okamoto and Eltis 2011). It has been reported that deficiency of Co in animals caused reduced growth, loss of body weight, and anemia (Tvermoes et al. 2014). However, long-term exposure of Co in the aquatic environment have been shown to negatively impact invertebrates (*Hyalella azteca* (28-d IC_{25}-growth-2.9 µg/L)) and fish (zebrafish *Danio rerio* (16-d IC_{25}-growth-340 µg/L)) (Government of Canada 2013). Acute Co exposures (5 to 20 mg/l for ≤96 h) have been shown to reduce growth of the cyanobacterium *Anabena variabilis* and the green algae *Chlorella vulgaris* (Ahluwalia and Kaur 1988, Rachlin and Grosso 1993). Freshwater invertebrate toxicity results indicated an acute LC_{50} (24 to 96-h) range of 1.1 mg/L (*Daphnia magna*) to 239 mg/L (*Tubifex tubifex*) (Kim et al. 2006), and lobster larvae (*Homarus vulgaris*) were found to have a 96-h LC_{50} range of 4.5 to 22.7 mg/L (Amiard 1976). Co is also acutely toxic to mammals with rats exhibiting an LD_{50} of 6,170 mg/kg. Co levels in OSPW range from <0.2 µg/L to 570 µg/L (Table 2), which in many cases exceed the acceptable concentration for fresh waters (2.5 µg/L) (Environment Canada 2013). Consequently, the presence of Co may be a contributor to OSPW toxicity.

3.1.1.6 Chromium

Chromium (Cr) is a transition metal with the atomic number 24, an atomic weight of 51.99, a melting point of 1,857 ^{o}C and a density of 7.19 g cm^{-3}. The most common uses of Cr are in stainless steel manufacturing, the production of mortars and castables, paper manufacturing, leather tanning, fertilizers, and other chemical industries. Cr is present in the wastewaters of these industries as well as wastewaters from metal processing, electroplating, and glue manufacturing. Several hundreds to thousands ppm of Cr can be present in fertilizers and sewage sludge (Sommers 1977). Other anthropogenic sources of Cr include atmospheric deposition from stack emissions for electric furnaces, coal-fired power plants and steel production (Stoeppler 1992).

Cr is a crucial element for animal and human nutrition since it is required for carbohydrate metabolism with a function closely related to that of insulin. Sufficient amounts of Cr in diet lead to a reduced requirement for insulin (Anderson 1981), while its deficiency leads to impaired glucose tolerance, and elevated serum insulin, cholesterol, and total triglycerides (Cohen et al. 1993). Adverse effects due to Cr exposure have been reported for aquatic organisms including reduced fecundity and survival, growth inhibition, and abnormal movement patterns (US EPA 1980). Cr exposure to fish has been shown to reduce growth and disease resistance, create morphological changes, and result in chromosomal aberrations. Cr does not biomagnify in aquatic food chains; however it has been shown to bioaccumulate in algae, aquatic vegetation, and invertebrates (ATSDR 1993). Compared to Cr (III), Cr (VI) is toxic while both are potent carcinogens and mutagens. Cr (VI) is readily converted to Cr (III) in animals, which protects the organisms from the adverse effects of Cr (VI) (Eisler 1986). Cr (VI) mainly targets the respiratory tract, creating symptoms including perforations and ulcerations of the septum, and

pneumonia. Cr can cause skin allergies, which lead to contact dermatitis (Shigematsu et al. 2014); these tests were done on mice, but can be generalized to affect humans as well.

The recommended Cr (VI) concentration in fresh water is 1 µg/L and for Cr (IV) is 8.9 µg/L (CCME 1999b). Cr concentrations in OSPW range from <1 µg/L to 400 µg/L (Table 2), which in many cases are much higher than recommended levels (CCME 1999b). Consequently, Cr may contribute towards the overall acute and/or chronic OSPW toxicity.

3.1.1.7 Copper

Copper (Cu) has the atomic number 29, an atomic weight of 63.55, a melting point of 1,083 ^{o}C and a density of 8.96 g cm^{-3}. It is widely used in the production of wires and in the electrical industry. It is also used in kitchenware, fertilizers, fungicides, bactericides, food additives, and as a disease control agent in poultry and livestock production. The main sources of Cu in the environment are acid mine drainage, chalcopyrite, fertilizers, fungicides, bactericides, metallurgical processes, coal combustion, and industrial emissions (Dudka and Adriano 1997, Johnson and Hallberg 2005, Kaneshiro et al. 2010).

Cu is a required micronutrient in organism nutrition. It is an important constituent of plant enzymes involved in various physiological functions like photosynthesis, cell wall metabolisms, respiration, and production of seeds. The deficiency of Cu in humans may lead to anemia, cardiovascular diseases, skeletal disorders, and the deterioration of the nervous system (Danks 1988).

The Cu^{2+} form is the most toxic in aquatic systems (Allen and Hansen 1996, Ma et al. 1999). It can bioconcentrate in fish and molluscs and has been shown to effectively interfere with branchial ion transport, plasma ion concentration, hematologic parameters, and enzyme activities (Stouthart et al. 1996). Also, it is linked to fish egg swelling due to excessive water influx (Jezierska et al. 2009), as well as decreased inflammatory responses and chronic toxicity in mammals and fish (Montaser et al. 2010, Sokolik et al. 2006). Cu concentration in OSPW ranges from <0.2 µg/L to 840 µg/L (Table 2), which exceeds the recommended CCME guidelines (2 to 4 µg/L; hardness dependant) in some cases. As a result, Cu may contribute to the overall OSPW toxicity.

3.1.1.8 Iron

Iron (Fe) has the atomic number 26, an atomic weight of 55.85, a melting point of 1,538 ^{o}C and a density of 7.9 g cm^{-3}. Iron has been used in conjunction with Al powder in welding and purifying ores, in the manufacture of steel, in engineering applications such as construction machine tools, and automobiles due to its high strength.

Fe has a crucial role in organisms, as it forms complexes with molecular oxygen in hemoglobin and myoglobin and is an important component of enzymes for various physiological functions (Jomova and Valko 2011, Pietrangelo 2011). However, excessive levels of Fe in the blood exceed the capability of transferrin to bind with Fe and leads to the production of free radicals which can damage DNA, proteins, and lipids (Jomova and Valko 2011). Excess Fe in human

blood has been shown to lead to adverse effects like coma, liver failure, adult respiratory distress syndrome, long-term organ failure, and shock (Chang and Rangan 2011, Pietrangelo 2011). The chief source of Fe in aquatic animals is water (source of dissolved Fe (II)) and food (source of Fe (III)). Once inside the animal body, Fe is actively transported across membranes by endocytosis (Luoma 1983, Simkiss and Taylor 1989).

Little information is available on the toxicity of Fe to aquatic animals; however, the Fe (II) species has been shown to be toxic, especially under acidic conditions (Gerhardt 1992). Fe toxicity to aquatic animals has been suggested to be due to the motion inhibiting or smothering effects of Fe hydroxide or Fe-humic precipitates on gills, eggs or other surfaces. As a result of these toxic effects, the organism access to crucial resources, including oxygen or food becomes limited. As well, Fe toxicity may also occur through DNA and membrane damage; however, the mechanism of this damage is currently unknown (Kari-Matti 1995). Fe concentrations in OSPW range from <10 µg/L to 34,600 µg/L (Table 2). Since the concentration of Fe often exceeds the recommended guidelines (CCME; 300 µg/L), it is assumed that Fe may contribute to the overall OSPW toxicity.

3.1.1.9 Mercury

Mercury (Hg) is a transition metal with the atomic number 80, an atomic weight of 200.6, a density of 13.55 g cm^{-3} and a melting point of -38.8 ^{o}C, being the only metal in liquid state at standard temperature and pressure. Hg is a good conductor of electricity, but a poor conductor of heat. It is available in the atmosphere as elemental Hg vapour; however, in soil, water and sediments, it exists as inorganic salts and organic Hg compounds and complexes. Various mercury sources include cinnabar (ore) and fossil fuels (coal/petroleum). It has both industrial (paints, fossil fuel, batteries, etc.) and clinical (thermometer, barometers, etc.) applications. Mining, smelting and industrial discharges lead to contamination of the environment with inorganic Hg compounds. Hg vapor from air is deposited into the aquatic environment, where elemental Hg is methylated to organic methylmercury by bacteria present in aquatic sediments. Hebert et al. (2013) found increasing Hg levels in colonial waterbird eggs downstream of the oil sands industrial region. Kirk et al. (2014) found that levels of Hg in snow increased with proximity to major developments. Radmanovich (2013) found that the levels of total Hg increased three fold in the Athabasca River at development sites and two fold downstream in comparison to upstream levels of 3.3 ng/L. As well, they found higher concentrations in summer compared to those in winter.

The most toxic forms of Hg are organic Hg compounds (Gaur et al. 2014) that can easily cross biological membranes. They accumulate in fatty tissue due to their non-polar properties and undergo biomagnification (Wright and Welbourn 2002). In aquatic environments, Hg bioaccumulates in fish, reaching toxic levels which can produce adverse side effects (Wang et al. 2005). For example, in the 1950s large quantities of inorganic mercury catalysts were discharged into Minamata Bay, Japan. The inorganic Hg compound was methylated which resulted in increased uptake of Hg throughout the food chain. As a result of this discharge, 41 deaths and about 30 cases of profound brain injuries in newborns from mothers consuming

contaminated fish during pregnancy were reported (Harada 1995). Recently, it was determined that about half of the worldwide emissions of Hg were from Asia (India, China, North and South Korea) (Pacyna et al. 2006).

The concentration of Hg in aquatic organisms is dependent on the concentration of Hg in the surrounding waters as well as Hg from the food supply. Various biomonitoring studies of Hg have been conducted on a wide range of organisms. For example, Hg concentrations in molluscs have been determined at various locations worldwide (Phillips and Muttarasin 1985, Solaun et al. 2013).

The highest Hg concentrations can be found in the Gulf of California, probably due to low mixing with the Pacific Ocean and the warm temperatures that favor the conversion of inorganic Hg compounds to organic compounds that bioaccumulate. Similarly, Zorita et al. (2007) reported an Hg level of 0.23 ± 0.15 mg/g in the northwest Mediterranean Sea since this area is influenced by a large range of industrial activities along the Mediterranean shoreline. Generally, the distribution and presence of particulate organic matter in waters may influence the biological availability of mercury (Kehrig et al. 2006).

Hg damages the nervous system and causes enzyme inhibition, which leads to abnormal neurotransmission and cardiac problems (Clarkson et al. 2003, Grandjean et al. 2004, Guallar et al. 2002, Murata et al. 2004). It affects cell division, DNA content and can cause cancer and birth defects. As well, Hg is known to cause behavioral changes in individuals that used Hg to treat felt for making hats (Neal et al. 1937, Waldron 1983). Hg poisoning can also lead to Korsakoff's syndrome and visual field constriction in human adults, and in human fetuses it can lead to seizures, hypertonia, deafness and blindness (Clarkson et al. 2003).

Methylated organic Hg is more toxic and bioaccumulative than inorganic Hg (Bradl 2005). The methylation of Hg is higher at low pH (Wright and Welbourn 2002). Hg was found to be less toxic when present with cadmium, as cadmium acts to protect aquatic organisms from Hg uptake and toxicity (Bradl 2005, Wright and Welbourn 2002). Hg toxicity was reduced by hard water (Ca^{2+} ions) inhibiting mercury uptake across cell membranes (Landis and Yu 2003). Temperature, organic compounds, and microbial inhibitors impact the methylation rate of Hg. For example, the 96-h LC_{50} of Hg^{2+} for the freshwater crayfish *Procambarus clarkia* (Girard) was found to be temperature dependent, with values of 0.79 mg/L (20 °C), 0.35 mg/L (24 °C) and 0.14 mg/L (28 °C) (Del Ramo et al. 1987). Hg concentrations in OSPW range from <0.02 µg/L to 0.17 µg/L which are generally higher than CCME guidelines (0.026 µg/L) (Table 2). Therefore, the potential for toxic effects of Hg present in OSPW would be expected.

3.1.1.10 Magnesium

Magnesium (Mg) has an atomic number of 12, an atomic weight of 24.3, a melting point of 650 ^{o}C and a density of 1.7 g cm^{-3}. Mg is found in the natural environment in deposits of magnesite and dolomite. Mg has been used in alloys with aluminum, in the production of iron and steel, for automotive components (e.g., wheels), in various electronics components (e.g., laptops, mobile phones, and cameras), and in the manufacture of laxatives and antacids.

Magnesium ions are essential for metabolism in human body cells primarily as MgATP. Many enzymatic reactions require Mg (Cowan 2002). Mg deficiency has been shown to cause cardiac arrhythmias, hypertension, hypocalcemia, myocardial infarction, osteoporosis, and hypokalemia (Rude 1998). The recommended daily intake of Mg for human males and females is 420 and 320 mg/day, respectively (Rude and Gruber 2004). Cases of toxicity due to Mg exposure have been extremely rare and there are no reports indicating that Mg causes systemic poisoning; however, there have been reported mild toxic effects including lethargy, nausea, double vision, confusion, and muscle weakness (Saris et al. 2000).

Mg ion concentrations in OSPW range from 2 mg/L to 33 mg/L (Table 2). There are no set environmental regulations for Mg due to the lack of evidence for any adverse health effects from exposures (Health Canada 2012). Therefore, Mg may not be considered as a contributor for the overall OSPW toxicity.

3.1.1.11 Manganese

Manganese (Mn) has the atomic number 25, an atomic weight of 54.94, a melting point of 1,244 ^{o}C and a density of 7.21 g cm^{-3}. Metamorphic, sedimentary, and igneous rocks are the main environmental sources of Mn. The main anthropogenic sources of Mn include industrial activities including metal smelting and refining, atmospheric deposition from fossil fuel combustion, and agricultural practices. For the metallurgical industry, it is a vital ingredient of steel for increased strength and hardness. Mn is used in the manufacturing of alloys of aluminum, copper, and steel. In addition, Mn is used in the production of batteries, ceramics, welding rods, alkaline batteries, glass, paints, and electrical coils.

Manganese is an essential micronutrient and is involved in many metabolic processes by activating enzymes required for the metabolism of organic acids, P, N and during the Hill reaction in plant chloroplasts (Adriano 2001). Mn is an important constituent of the animal diet where it acts as a cofactor for enzymes including kinases, decarboxylases, and hydrolases (Adriano 2001).

Mn toxicity is very rare in mammals and is only a result of chronic inhalation of airborne Mn, where it can be a neurotoxin leading to manganism disease in the brain (Erikson et al. 2007, Quintanar 2008). High doses of Mn are also known to cause alteration of brain chemicals, gastric irritation, low birth weight, delayed testicular development, muscular weakness, and behavioral changes in mammals. Mn can be toxic to mouse embryos and fetuses (Colomina et al. 1996, Gerber et al. 2002, Torrente et al. 2002) and can cause malformations and developmental delays in invertebrates such as sea urchin (*Anthocidaris crassispina*) (Kobayashi and Okamura 2005). High levels of Mn in birds are known to cause anemia, reduced growth, and decreased hemoglobin (ATSDR 2012). Due to large-scale production of Mn-containing compounds in the past, it is a newly emergent contaminant of concern in the aquatic environment. Mn chloride has been reported as the most toxic species of Mn (Pinsino et al. 2010). The concentrations of Mn in OSPW range from <4 µg/L to 3,610 µg/L (Table 2). Canadian Water Quality Guidelines show the maximum acceptable concentration of Mn in drinking water is 50 µg/L, thus indicating that Mn may contribute to the overall OSPW toxicity.

3.1.1.12 Molybdenum

Molybdenum (Mo) has the atomic number 42, an atomic weight of 95.94, a melting point of 2,617 ^{o}C and a density of 10.28 g cm^{-3}. Natural sources of Mo include sandstone, limestone, coal, ash, groundwater, igneous rocks, and seawater. As well, it is present in many minerals such as ferrimolybdenite, powellite, molybdenite, wulfenite, and jordisite. Mo is used in the production of various alloy steels and stainless steels where it has been widely accepted as a substitute for Cr and other metals. Other uses of Mo include catalysts, dyes, corrosion inhibitors, rubber parts, high pressure grease, corrosion inhibitor, industrial gear oils, and fertilizers. Mo fertilizers, sewage sludge, coal combustion and mining, and smelting are the chief anthropogenic sources of Mo to the environment (Eisler 1988).

Mo is a crucial nutrient for plants and animals (Rajagopalan 1988). It is an important component of enzymes associated with various metabolic activities including N metabolism (nitrogenase) where plants deficient in Mo exhibit N deficiency (Parker and Harris 1977). As well, Mo acts as a catalyst for various enzymes in animals and humans (Iobbi-Nivol and Leimkuhler 2013, Marelja et al. 2013). Low Mo concentrations in water lead to restricted nitrogen fixation in algae which limits photosynthesis (ter Steeg et al. 1986).

The physical and chemical state of the Mo, route of exposure, and compounding factors such as dietary copper and sulfur levels may all affect toxicity. Mo toxicity in humans has rarely been reported (Rajagopalan 1988) with toxic effects including increased blood xanthine oxidase, elevated uric acid in urine and blood, and gout (Johnson 1999). The chief sources of Mo exposure to humans are baked products, legume seeds, cereal grains, milk, and milk products (Vahčić et al. 2010). In animals, acutely toxic oral doses of Mo result in severe gastrointestinal irritation with diarrhea, coma, and death from cardiac failure. Oral LD_{50} values of 125 and 370 mg Mo/kg for molybdenum trioxide and ammonium molybdate, respectively, have been reported in laboratory rats (Venugopal and Luckey, 1978).

Aquatic organisms are relatively resistant to Mo with toxic effects on growth only occurring at concentrations > 50 mg/L. Freshwater and marine fish were found to be extremely resistant to Mo with observed 96-h LC_{50} values between 70 mg/L and <3,000 mg/L. Acute toxicity for fathead minnows (*P. promelas*) has been found to be in the range of 70 mg/L to 370 mg/L (96-h). The 7-d LC_{50} for goldfish embryos (*Carassius auratus*) was 60 mg/L (CCME 1999d), the chronic LC_{50} value for *O. mykiss* eggs has been found to be 0.79 mg/L (28 d), and newly fertilized rainbow trout eggs having a lower LC_{50} value of 0.79 mg/L (28 d) (Eisler 1988). Mo is essential for aquatic plant growth; however the required water concentration is not known with certainty (Henry and Tundisi 1982, Schroeder et al. 1970).

Mo concentrations in OSPW range from 2 µg/L to 2,120 µg/L (Table 2). The values from some water samples have far exceeded the recommended CCME guidelines (73 µg/L), indicating that Mo may contribute to OSPW toxicity.

3.1.1.13 Nickel

Nickel (Ni) is a transition metal with the atomic number 28, an atomic weight 58.69, a melting point of 1,453 ^{o}C and a density of 8.91 g cm^{-3}. Ni can readily complex with organic ligands. Various natural sources of Ni include sandstone, limestone, coal, ash, igneous rocks, soils, seawater, and fresh water. Ni is mostly used for Ni-Cd batteries, electronic components, electroplating, alloy production (Ni-Fe, Ni-Cr, Ni-Cu and Ni-Ag), and during the hydrogenation of fats. One of the broadest applications of Ni is in the production of stainless steel where it is found in products including coins, kitchen appliances, sinks, surgical implements, and automobiles. Anthropogenic sources of Ni into the environment include sewage sludge, mining, smelting, and fuel and coal combustion (Fuge 2013).

Although Ni is toxic to organisms at elevated levels, it is required in trace amounts for various biological processes including as a constituent in urease, methyl coenzyme M reductase, carbon monoxide dehydrogenase, and hydrogenase (Ahmad and Ashraf 2011). In the aquatic environment, Ni may cause tissue damage, growth reduction, and genotoxicity. Low levels of Ni may result in reduced skeletal calcification and asphyxiation in fish. In freshwater organisms (e.g., rainbow trout) Ni concentrations in the range of 24 to 10,000 µg/L have produced acute or chronic toxicity (Environment Canada and Health Canada 1994). Crustaceans and molluscs are more sensitive to Ni than other organisms (Environment Canada and Health Canada 1994). Ni poisoning in humans can be the result of inhalation of Ni components (e.g., Ni_3S_2) which produces diseases including pneumonitis, hepatic degeneration, asthma, and cancer of respiratory eosinophilia. As well, contact dermatitis may be the result of long-term Ni skin contact in humans (Zoroddu et al. 2014).

Leaching from metals in contact with water (pipes and fittings) are the primary source of Ni, however, it may also be present in groundwater as a result of dissolution form nickel ore-bearing rocks (WHO 2005). Ni concentrations in OSPW range from <0.5 µg/L to 270 µg/L (Table 2). The toxicity of Ni is dependent on various factors, including the hardness (Craig et al. 2009) of the medium and the age and diet of the organism (Anderson et al 1979, Puttaswamy and Liber 2012). Depending upon the hardness, the recommended levels of Ni under CCME guidelines range from 25 µg/L to 150 µg/L. The level of Ni in OSPW sometimes exceeds the acceptable concentrations, thus contributing towards overall OSPW toxicity.

3.1.1.14 Lead

Lead (Pb) has an atomic number of 82 and an atomic weight of 207.2, a melting point of 328 ^{o}C and a density of 11.34 g cm^{-3}. Pb has been in use by humans for several thousand years. It is used in industries such as mining, smelting, manufacturing, and waste disposal which add substantial amounts of Pb into the environment.

Pb is an important metal used in automobiles, antiknock agents, paints, and in many alloys (Gaur et al. 2014). The use of tetraethyl lead in gasoline as an antiknock agent has been banned in the US, Canada, and Europe since the 1980s. However, it is still in use in various countries including China, Russia, and India (Bradl 2005, Soto-Jimenez et al. 2006). Significant quantities of Pb have been found in canned milk products in the past, which is a serious problem for

childhood development due to Pb poisoning (Shea 1973). The presence of Pb in canned milk was attributed to the solder and flux used to seal the cans; the practice of using lead has been discontinued and replaced by welded side seams (Brody 1997). Another source of Pb is solder used in pipes and water coolers that leaches out as it can be mobilized by soft (acidic) water. Even though the use of Pb has been reduced or completely discontinued, the legacy of Pb contamination still remains, especially in soils (Wixon and Davies 1993, Wright and Welbourn 2002). Bitumen production and development of the oil sands (mining, processing, and tailing pond leakage) are the prime source of Pb in oil sands (Kelly et al. 2010).

Most Pb salts settle being insoluble in water and are therefore commonly found in sediments that act as a Pb reservoir that can be mobilized by bacteria allowing Pb to enter the food chain (Wright and Welbourn 2002). Lead acts as a nonspecific toxin that can inhibit many enzymatic activities within livestock, fish, and wildlife. Typically, it affects hematological functions, the central nervous system, and the reproductive system (Gurer and Ercal 2000, Jackim et al. 1970, Sandhir et al. 1994). Both organic and inorganic Pb enters the body via inhalation (air and dust) and ingestion (food and water), while organic lead can also enter through skin contact (Adriano 2001).

Inorganic Pb does not undergo biological transformation; however organic Pb is metabolized in the liver (Adriano 2001). Metabolized Pb gets distributed in blood, soft tissue (kidney, liver, brain) and mineralizing tissue (bones and teeth) of wildlife. Pb bioconcentrates in the skin, bones, liver, and kidneys of fish rather than muscle, and does not biomagnify in the food chain. However, consuming entire exposed fish and/or wildlife can definitely expose one to high concentrations of Pb (Wright and Welbourn 2002). Pb toxicity is species- and concentration-dependent; for example, goldfish are more resistant to Pb than other fish species because they have abundant gill secretion. In birds, Pb paralyzes the gizzard that leads to starvation and ultimately death (Landis and Yu 2003). It is not possible to get rid of Pb from the human body as the half-life of Pb is 20 years and the release of Pb from the body is a very slow process. Bones and teeth are not harmed by lead; however they function as reservoirs and release Pb into the bloodstream, where it targets organs such as the brain. It can cross the placenta barrier, leading to miscarriages, stillbirths, and neurological damage. In addition, Pb inhibits neurotransmission as it mimics calcium (Bradl 2005, Landis and Yu 2003, Wright and Welbourn 2002).

Pb concentrations in OSPW range from <0.3 µg/L to 226 µg/L (Table 2). It has been found that the oil sands industry substantially increases the concentration of toxic pollutants including Pb into the Athabasca River (Kelly et al. 2010). Like Cd, Pb has also been known to cause fish egg swelling (Jezierska et al. 2009). The LC_{50} value for lead for freshwater catfish (*Heteropneustes fossilis*) after a 24 h exposure has been found to be 885 mg/L (Srivastav et al. 2013). Also, depending upon the hardness of the water, the recommended levels of Pb range from 1 µg/L to 7 µg/L. Consequently, it is highly likely that the Pb would contribute towards overall OSPW toxicity. Furthermore, bioaccumulation of Pb has been observed in muscle and liver of fish species exposed to lead contaminated river and marine in Khouzestan (Askary Sary and Mohammadi 2012).

3.1.1.15 Antimony

Antimony (Sb) has the atomic number 51, an atomic weight of 121.7, a melting point of 630.6 ^{o}C and a density of 6.7 g cm^{-3}. Sb has been used in alloys, fire retardants, antiprotozoan drugs (Harder 2002), and a variety of cosmetics (e.g., lipsticks and eye pencils) (Bocca et al. 2014).

Sb and its compounds have been found to produce toxic effects similar to As poisoning (headaches, confusion, severe diarrhea, and drowsiness), however the toxicity produced is far less than that of As at similar exposure levels (Ferreira et al. 2014). Prolonged skin contact with Sb has been shown to result in dermatitis, liver and kidney damage, and frequent vomiting which could lead to death (Sundar and Ckakravarty 2010).

Sb does not bioaccumulate or bioconcentrate in fish and other aquatic organisms. Nevertheless, high concentrations of Sb have been measured in the tissues of upper trophic level macroinvertebrates in aquatic ecosystems exposed to mining activity (Fu et al. 2010, Telford et al. 2009).

The effect of Sb on Japanese medaka, planktonic crustacean and green algae were studied and it was observed that Sb was less toxic to larval medaka (24-h LC_{50}, 261 mg/L; 48 h-LC_{50}, 238 mg/L), while it was 20 to 50 times more toxic to planktonic crustacean (24-h LC_{50}, 12.83 mg/L and 24-h LC_{50}, 4.92 mg/L). Growth inhibition was also observed in green algae in the presence of Sb (72-h EC_{50}, 206 mg/L) (Nam et al. 2009). Sb concentrations in OSPW range from <0.2 µg/L to 12 µg/L (Table 2). As the reported LC_{50} and EC_{50} values are generally much higher than the concentrations of Sb in OSPW, it is anticipated that Sb may not significantly contribute to the overall OSPW toxicity.

3.1.1.16 Selenium

Selenium (Se), a non-metal, has the atomic number 34, an atomic weight of 78.96, a melting point of 217 ^{o}C and a density of 4.3 to 4.8 g cm^{-3}. One of the primary sources of Se in nature is volcanic eruptions. Se has been used in photoelectric cell devices (e.g., photographic exposure meters) and xerography, rubber manufacture, glass production, shampoos as conditioner, insecticides, fungicides, and lubricants. Se can be found as a by-product of the electrolytic refining of Cu. Se contamination has been reported due to Se-rich soil and drainage from coal fly ash ponds (Frankenberger and Engberg 1998, Government of Alberta 2010).

Selenium is a required nutrient that has been shown to have a complementary role to vitamin E where it helps prevent dietary hepatic necrosis and exudative diathesis in rats and chicks (Mayland 1994). It is believed that Se exerts its effects via the antioxidant action of the Se-dependent enzyme GSHPx (Frankenberger and Engberg 1998). It has been shown to be an essential part of the glutathione peroxidase enzyme (major cellular antioxidant) converting free radicals to peroxides and then to water and oxygen. Se deficiency in humans and animals has been shown to reduce growth and appetite, reduce reproductive fertility, cause weakness, and lead to anemia (Combs and Combs 1984). Se exists in inorganic and organic (more toxic and bioaccumulative) forms in surface waters (Besser et al. 1993). An organisms' exposure to selenium may occur through water or food. It has been shown to be required in the diet of fish at

0.1 to 0.5 µg/g dry weight (Frankenberger and Engberg 1998); however, if the concentration increases to >3 µg/g it is potentially toxic.

Se toxicity has been reported in fish, waterfowl, and other animals over the past several years (Frankenberger and Engberg 1998). For aquatic organisms, Se is known to cause loss of equilibrium, neurological disorders, reproductive failure, liver damage, reduced movement rate, reduced hemoglobin, chromosomal aberrations, increased white blood cell count, and necrosis of ovaries (Eisler 1985).

Se concentrations in OSPW range from <0.2 µg/L to <70 µg/L (Table 2), which exceeds the maximum acceptable concentration for Se in drinking water (1 µg/L) as per CCME guidelines (CCME 1999b). Therefore, it is believed that presence of Se may contribute to the overall OSPW toxicity.

3.1.1.17 Strontium

Strontium (Sr) has the atomic number 38, an atomic weight of 87.62, a melting point of 777 ^{o}C and a density of 2.6 g cm^{-3}. In natural environments, Sr has been mainly found in the form of its sulphate mineral celestite ($SrSO_4$) and carbonate strontianite ($SrCO_3$). Sr has been used in glass for color television cathode ray tubes (blocks X-rays), as a salt in fireworks (produces a deep red color), to help improve the mechanical properties of Mg alloys (Fan et al. 2007), and to enhance the corrosion resistance of Mg by modifying its surface (Suganthi et al. 2011, Zheng et al. 2006). In addition, Sr was recently established to have medical applications (Gu et al. 2012). Sr plays a crucial role in human biological processes, especially those of the heart, muscles, and bones (Nielsen 2004). It has been shown to enhance the growth of osteoblasts that improve bone formation (Boanini et al. 2010, Kung et al. 2010, Pramatarova et al. 2005), and inhibit bone resorption (Bigi et al. 2007, Dahl et al. 2001, Marie 2005, Zhang et al. 2011b). Inhalation of Sr can lead to anaphylactic reaction, extreme tachycardia and difficulty in respiration. It can cause rickets in children by inhibiting normal calcium absorption. The United States Environmental Protection Agency (EPA) has set an upper limit of 4 mg/L for Sr^{2+} in drinking water (Qu et al. 2012).

Low acute toxicity of Sr has been observed in freshwater organisms, with an LC_{50} of 0.2 mg/L reported in embryo larval tests for rainbow trout (28 days) and eastern narrowmouthed toad (7 days) (Watts and Howe 2010). Sr led to reduced calcification in freshwater green alga *Gloeotaenium* at a concentration of 150 mg/L (Prasad 1984). During the early life stages of rainbow trout, it has been observed that the 10% effects level based on teratogenic responses and mortality was 49 µg/L. Sr also leads to reduced egg hatchability in fathead minnow (*Pimephales promelas*) (Chowdhury and Blust 2011). The concentrations of Sr in OSPW range between 60 µg/L and 1,560 µg/L (Table 2). These results suggest that Sr may contribute to the overall OSPW toxicity.

3.1.1.18 Uranium

Uranium (U) has the atomic number 92, an atomic weight of 238.03, a melting point of 1,132 ^{o}C and a density of 19.1 g cm^{-3}. It is used as a constituent of atomic bombs, nuclear power plants,

and photographic chemicals and radioisotopes used for the diagnosis of disease and research. U is highly soluble at alkaline pH, making it mobile and available in groundwater and soil (Nilgiriwala et al. 2008).

U exposure can be as a result of dust inhalation or via contaminated water or food. Soluble U compounds quickly pass through the body; however, insoluble U compounds have been shown to pose a serious health hazard as they tend to bioaccumulate and remain in bone tissues (Barillet et al. 2011, Kraemer and Evans 2012). There have been a number of aquatic toxicity reports for U; however, there is a lack of information available regarding potential mechanism(s) of U toxicity. It has been observed that U can inhibit ATPase and cause oxidative stress in animal tissue (Barillet et al. 2007, Nechay et al. 1980, Periyakaruppan et al. 2007, Ribera et al. 1996) and can disrupt gill, muscle, hepatopancreas ,and gonadal tissues of aquatic organisms (Al Kaddissi et al. 2011, Barillet et al. 2010). Initiation of reactive oxygen species (ROS) in the lungs of rats (Periyakaruppan et al. 2007) and in fish (Barillet et al. 2007, 2010, Buet et al. 2005) has been associated with the failure of cellular antioxidant mechanisms which are used to suppress the increased oxidative species (Trenfield et al. 2012).

U concentrations in OSPW range from <0.2 µg/L to <500 µg/L (Table 2). The reported short-term toxic concentrations of uranium range from 1,670 to 59,000 µg/L and 60 to 74,340 µg/L, for fish and invertebrates, respectively (24 to 96-h LC_{50}) (CCME 2011b). CCME regulates long-term U concentrations at 15 µg/L and short-term concentrations at 33 µg/L. It is worth noting that these guidelines are only for chemical toxicity and it excludes radiation toxicity that is expected to be minimal due to its low penetrating power. As the concentration of U in OSPW exceeds the CCME guidelines, the overall contribution of U towards OSPW toxicity may be expected.

3.1.1.19 Vanadium

Vanadium (V) has the atomic number 23, an atomic weight of 50.94, a melting point of 1,910 ^{o}C and a density of 6 g cm^{-3}. It is found in the natural environment in fossil fuel deposits and in many different minerals. V is used in steel alloys including high speed tool steel, bicycle frames, crankshafts, axles, and gears. It forms strong nitrides and carbides which significantly increase the strength of steel (Reardon 2011). It has also been used in V redox flow batteries and catalysts (Kear et al. 2012, Xia et al. 2012, Zhang 2014). Burning of petroleum, coal, and oil are large sources of V discharge into the environment (Venkataraman and Sudha 2005).

V has been shown to have a limited role in biological processes and is more relevant in marine environments than terrestrial ones (Sigel and Sigel 1995). It has been observed that V is associated with marine algae enzymes (Butler and Carter-Franklin 2004, Gribble 1999). V is a micronutrient in mammals; however, its exact function is not clear. V deficiency has been shown to result in reduced growth and impaired reproduction in chickens and rats (Schwarz and Milne 1971). Conversely, V compounds have been found to cause adverse effects on the respiratory system (Ress et al. 2003, Sax and Bruce 1975, Wörle-Knirsch et al. 2007) and lipid metabolism (Imura et al. 2013).

V toxicity in aquatic environments has not been fully characterized. It has been observed that V can be toxic toward fish, however, specific information regarding marine and estuarine fish species is limited (Gravenmier et al. 2005). Fish (4 to 6-d, LC_{50}-0.5 to 22 mg/L) have been shown to be generally more sensitive to V than invertebrates (9-d, LC_{50}-10 to 65 mg/L) (Venkataraman and Sudha 2005). The 96-h LC_{50} of vanadium to adult American flagfish (*Jordanella floridae*) was 11.2 mg/L in very hard water. Larvae showed 28-d LC_{50} of 1.13 and 1.88 mg/L of vanadium with larger larvae being more resistant (Douglas and Sprague 1979). V was moderately toxic to juvenile rainbow trout and whitefish (96-h LC_{50}, 6.4 and 17.4 mg/L, respectively) and the toxicity increased slightly with decreasing pH (Giles et al. 1979). Vanadium concentrations of 2.4 to 5.6 mg/L were lethal in 7 days to rainbow trout of wet weight 1.2 to 6.2 g (Sprague et al. 1978). V concentrations in OSPW range widely from 1 µg/L to up to 1,720 µg/L (Table 2). Therefore, it is expected that V may contribute to the overall toxicity of OSPW.

3.1.1.20 Zinc

Zinc (Zn) has the atomic number 30, an atomic weight of 65.38, a melting point of 420 ^{o}C and a density of 7.14 g cm^{-3}. Zn has been used in the automobile industry for the coating of steel and iron, dry cell batteries, cosmetics, paints, rubber, and antiseptics. As well, it has been used as a micronutrient fertilizer, insecticide, and wood preservative. The chief anthropogenic sources of Zn to the environment include fertilizers, mining and smelting, and sewage sludge (Adriano 2001, Zheng et al. 2010).

Zn is important for plants and animals. For plants, it is required for various metalloenzymes, and for the stability of the root cell plasma membrane. Zn toxicity is very rare in humans; however, Zn is crucial for various biological processes, including more than 300 enzymes and proteins. Human Zn deficiency has been shown to result in anaemia, dermatitis, neuropsychological dysfunction, and poor wound healing (Prasad 2012).

Phytotoxic symptoms like chlorosis can occur if the Zn concentration is elevated (>100 ppm) (Beyer et al. 2013). In the aquatic environment, Zn tends to partition into sediments and is less frequently dissolved as hydrated Zn ions and organic and inorganic complexes (MacDonald 1994). Toxic effects such as retarded fertilization and poor embryo development have been seen in the embryo development of the European clam (*Ruditapes decussatus*) as a result of exposure to Zn concentrations between 128 and 256 g/L (Fathallah et al. 2010). The LC_{50} value for Zn is not available for freshwater invertebrates; however, an LC_{50} of 80 mg/kg dry soil has been reported for the earthworm *Eisenia foetida* (CCME 1999f).

Zn can cause acute and chronic toxicity. The acute effects include nausea, loss of appetite, diarrhea, and vomiting. In one report, vomiting and nausea was reported within 30 minutes of ingesting zinc gluconate (4 g) (Lewis and Kokan 1998). The chronic effects daily intake of zinc (150 to 450 mg) includes reduced immune function and low levels of high density lipoproteins (Hooper et al. 1980). Zn concentrations in OSPW range from <0.6 µg/L to 1,240 µg/L (Table 2), which in some cases, are much higher than the CCME guidelines (30 µg/L). Therefore, the presence of Zn in OSPW may be expected to contribute to the overall OSPW toxicity.

3.2 Salts

The elevated levels of salinity found in OSPW are the result of the addition of sodium hydroxide (NaOH) during bitumen extraction and calcium sulphate ($CaSO_4$) used for consolidated tailings (CT) creation, as well as the leaching of ions and dissolved solids from the ore (Allen 2008a,b). Other contributors include the oxidation on the beaches, the use of groundwater with high salinity, the fly ash dumped into ponds, and microbial activities. Typically, OSPW is considered to be moderately hard (15 to 25 mg/L Ca^{2+} and 5 to 10 mg/L Mg^{2+}) with elevated concentrations of total dissolved solids (TDS) (2,000 to 2,500 mg/L) (Allen 2008a). The composition of OSPW varies with the ore quality, variability in the extraction process (e.g., quantity of added NaOH), quantity of water recycling, input of wastewaters from utility/upgrade processes, tailings treatment process (e.g., gypsum-based composite tailings), and *in situ* processes (Zubot et al. 2012).

A summary of recent reports on the inorganic chemistry of OSPW from active tailings settling basins and surface waters potentially impacted by OSPW is shown in Table 3. The TDS ranges for non-OSPW and OSPW sources are 80 to 190 mg/L and 200 to 2,600 mg/L, respectively. As well, the conductivities (Λ) for non-OSPW and OSPW sources are 256 to 663 μS/cm and 237 to 4,730 μS/cm, respectively.

The major ions typically responsible for high conductivity in OSPW include Na^+ (37 to 1,080 mg/L), Cl^- (4.4 to 866 mg/L), HCO_3^- (539 to 1,019 mg/L), and SO_4^{2-} (29 to 1,590 mg/L) (Table 3). Generally, for surface waters these major ion concentrations are either at or below the low end of the ranges of concentrations in OSPW (Table 3). The chloride ion (Cl^-) that is regulated by CCME (2011a) is considered to be a hydrologically and chemically inert substance, and the concentrations normally remain high in OSPW. Cl^- levels from many OSPW sites have exceeded the reported long-term exposure Canadian Water Quality Guidelines (120 mg/L) for the protection of freshwater life and, in some cases, the exceedance of the short-term exposure guideline (640 mg/L) has been observed (CCME 2011a).

Increasing concentrations of ammonia is another concern associated with OSPW. The ammonia concentration in MLSB increased from 4 mg/L in 1980 to 14 mg/L in 2001 (Allen 2008a). Based on more recent data, the levels of ammonia from various OSPW sites are quite variable from insignificant to 65 mg/L (Table 3), with values from most sites having exceeded the 2010 CCME guidelines for the protection of aquatic life (0.053 to 2.33 mg/L NH_3 for pH 8 to 9) (CCME 2010).

Table 3. Inorganic water chemistry of oil sands process-affected waters, natural surface waters, and the Athabasca River.

Parameters [mg/L] unless noted otherwise	OSPW (active tailings ponds)[a,1]	Aged OSPW (reclamation ponds)[a,2]	Natural surface waters[a,3]	Athabasca River water[d]
pH [-]	7.2 to 8.9 [a,1, c,e,f]	7.2 to 9.2	7.4 to 9.3	8.2
TDS	200 to 2,600[b,c]	-	80 to 190[d]	170
Λ (µS/cm)	237 to 4,290 [a,1, c]	342 to 4,730	256 to 663	280[h]
Sodium	159 to 844 [a,1,b,c]	37 to 1,080	18 to 94	21[h]
Calcium	2 to 103 [a,1,b,c]	5.7 to 200	23 to 39.7	30
Magnesium	2 to 34.6 [a,1, b,c]	3 to 118	8.7 to 22.1	8.5
Chloride	42 to 866 [a,1, c,e,f]	4.4 to 690	4.7 to 56	15[h]
Bicarbonate	610 to 1,019[f,g,h]	539 to 781[f]	81[f]	130[h]
Sulphate	22 to 470 [a,1, c,e,f]	29 to 1,590	5 to 38	22
Ammonia	<0.01 to 65 [a,1,b]	<0.01 to 2.1	<0.01 to 0.16	0.06

Notes:
TDS: total dissolved solids; Λ: conductivity; WIP: West In Pit; AURTP: Aurora tailings pond; MLSB: Mildred Lake Settling Basin; DP: Demonstration Pond; OSPM: oil sands-processed materials.
Ranges indicate mean values for multiple sites.
[a,1] Sansom et al. (2013); Syncrude OSPW sites including active settling basins and waters from dyke seepage and seepage control system; Sampling period: July 2007 and August 2008.
[a,2] Sansom et al. (2013); Syncrude reclamation sites containing aged OSPW and OSPM; Sampling period: July 2007 and August 2008.
[a,3] Sansom et al. (2013); Natural non-OSPW surface waters in the area of Syncrude's Mildred Lake surface operation; Sampling period: July 2007 and August 2008.
[b] Suncor Energy Inc. (2009); Suncor Energy Inc. Tailings reduction operations application. OSPW from active Suncor mine sites.
[c] Gamal El-Din, unpublished; Sampling period: September 2010 to August 2013.
[d] Allen (2008a), Golder Associates Limited (2002).
[e] Holden et al. (2011); Suncor South tailings pond; Sampling period: August 2008.
[f] Kavanagh et al. (2011); Gregoire Lake, Syncrude ponds (FE5, Pond 9, DP) and Suncor's North and South tailings ponds.
[g] Toor et al. (2013); Syncrude MLSB, and Suncor Pond 2/3; Sampling period: 2006 to 2007.
[h] Zubot et al. (2012); Syncrude WIP, and Athabasca River; Sampling period: 2006 to 2008.

3.2.1 *Toxicity of Salts*

TDS and conductivity are indicators of the ionic strength of water (Koshy et al. 2008) with highly ionic waters having exhibited toxicity towards various aquatic species (Chapman et al. 2000, Kennedy et al. 2005). Waters that exhibit elevated TDS and conductivity, as found in OSPW, may induce ionic imbalances in exposed organisms leading to osmotic stress and potentially mortality. While the sensitivity is species-specific, waters with TDS greater than 1,340 mg/L and conductivity above 2,000 mS/cm have been generally considered as sufficient to cause toxicity in freshwater species (Goodfellow et al. 2000, Koshy et al. 2008).

The toxicity of some specific ions in waters has raised concerns. For example, freshwater organisms (invertebrates, amphibians, and fish) exposed to elevated levels of Cl^- have exhibited increased mortality, inhibited growth, reduced embryo viability, and impaired reproduction (CCME 2011a). However, it should be noted that these results commonly relied on exposures in reconstituted water using Cl^- salts such as NaCl and $CaCl_2$, thus they may not necessarily reflect organism responses in actual environmental conditions. Hardness and other water chemistry variables may have ameliorating effects on Cl^- toxicity (Elphick et al. 2011, Gillis 2011).

Ammonia is another chemical of concern. 96-h LC_{50} of ammonia to fish ranged from 0.56 to 2.37 mg/L, and 48-h LC_{50} values for freshwater invertebrates ranged from 1.10 to 22.8 mg/L (Environment Canada 1999). The toxicity of ammonia is associated with the speciation, with un-ionized ammonia exhibiting more toxic than the ammonium ion, due to its greater capacity of disusing across biological membranes (CCME 2010). Therefore, the impacts of factors such as pH and temperature on speciation of ammonia should be taken into account when evaluating the water toxicity.

The elevated concentration of salts in OSPW may contribute to its overall toxicity. A multi-fish cell line bioassay conducted by Sansom et al. (2013) found a high correlation between reduced cell viability and increased OSPW Na^+ and HCO_3^- concentrations, which implies that they are contributing to OSPW toxicity. Kennedy (2012) exposed *C. riparius* to water mimicking combinations of salts and NAs, finding that their survival was negatively correlated with both salt and NAs concentrations. In fact, early studies have shown that *C. plumosus* is intolerant of salinities greater than their internal salinity due to lack of osmoregulation (Lauer 1969), while the emergence of *C. riparius* was shown to be negatively impacted by salt stress (Bervoets et al. 1996). It should be noted that although most *Chironomus* species appear to be salt-intolerant (Cannings and Scudder 1978), their sensitivity to salts is quite variable. For instance, *Tanytarsus*, *Derotanypus*, and *Cricotopus* were found to be dominant in salty, OSPW-affected wetlands, but only *Derotanypus* was completely absent in low-conductivity reference wetlands, making it a good indicator of saline conditions (Whelly 1999).

The adverse effects of salts on fish have also been reported. Increased alterations of gill proliferative changes were observed in fish exposed to NAs with the addition of Na_2SO_4 (Nero et al. 2006a). These gill pathological changes were suggested to be associated with a reduced gill surface area, which might cause a reduction in both NAs transport within fish and the exchange of vital respiratory gases. As well, a reduced spawning and secondary sexual characteristics

have been shown in fathead minnows when exposed to >25 mg/L NA along with high salt concentration (Kavanagh, et al. 2011). Interestingly, while both NAs and salts exerted toxicity to *C. riparius*, there was an antagonistic interaction between these two toxicants (Kennedy 2012) with similar ameliorating effects of salts on NAs also reported in fish. The mortality of yellow perch (*Perca flavescens*) exposed to C-NAs and OSPW-derived NAs was reduced following the addition of Na_2SO_4 (Nero et al. 2006a). Other research showed decreased inhibitory effects of NA extracts on the number of reproductive tubercles and plasma T levels in male fathead minnows after addition of $NaHCO_3$, possibly through reducing the uptake of NAs by fish (Kavanagh et al. 2012). $NaHCO_3$ also reduced the toxic effects of NAs extracts on fathead minnow embryo and larvae mortality; however, no reduction was observed for the salts NaCl or Na_2SO_4 (Kavanagh et al. 2012). In addition, the presence of ions such as HCO_3^- is also known to reduce the uptake and toxicity of various metals, usually through the formation of less toxic metal-base complexes (Wurts and Perschbacher 1994).

4 REMEDIATION

OSPW needs to be remediated using appropriate treatment processes that eliminate their toxicity. Treated OSPW could then be reclaimed either via viable aquatic habitats or released to the environment. Currently, a number of approaches have been proposed with focus on NA degradation, given their predominant role in the OSPW toxicity, and research is underway to optimize some of the promising treatment methods such as biodegradation, ozonation, and reclamation using wet or dry landscapes.

Biodegradation, also known as OSPW aging, allows the microbial populations indigenous to the tailings ponds to degrade NAs and other constituents over many years. This method is the most cost-effective way to decrease NA concentrations and mitigate their toxicity with selective removal of lower molecular mass NAs (Kannel and Gan 2012). However, observations of the degradation in tailings ponds have indicated that NAs are poorly or very slowly degraded on-site (Scott et al. 2008) and that there is persistent toxicity (e.g., impaired reproductive capacity of fathead minnows) of aged OSPW (~20 years) (Kavanagh et al. 2011). The slow and incomplete removal of NAs and the residual toxicity of OSPW might be due to the presence of recalcitrant and toxic NAs and/or other toxic constituents in OSPW. Due to the complexity of NAs and the other pollutants present in OSPW, coupled with the poorly understood mechanisms by which microbial communities are capable of breaking down these compounds, it is suggested that biodegradation alone is not an effective remediation strategy for the extensive volumes of OSPW (Kannel and Gan 2012, Martin et al. 2010, Whitby 2010). In addition, the biodegradation produces methane that is a potent greenhouse gas.

Ozonation is a promising remediation technique for OSPW[2] given it is capable of reducing NAs either partially (He et al. 2011) or totally (Scott et al. 2008), with subsequent reduction in overall OSPW toxicity. Various studies have shown that ozonation reduced the toxicity of OSPW for

[2] See Liang, J., F. Tumpa, L.P. Estrada, M. Gamal El-Din and Y. Liu, 2014. Ozone-Assisted Settling of Diluted Oil Sands Mature Fine Tailings: A Mechanistic Study. OSRIN Report No. TR-46. 43 pp. http://hdl.handle.net/10402/era.38226 for additional discussion of ozonation.

nearly all endpoints examined, including acute toxicity to *V. fischeri* (Scott et al. 2008, Wang et al. 2013), endocrine disruptive effects in cell lines and fish (He et al. 2010, 2011), adverse impacts on the growth and development of invertebrates and fish (Anderson et al. 2012b, He et al. 2012a), transcriptional responses of the brain-gonad-liver axis of fish (He et al. 2012b), and immunotoxicity in mice (Garcia-Garcia et al. 2011a). However, He et al. (2011) found ozonation did not reduce the estrogenic effect of OSPW in a human cell line. This may be due to ozonation preferentially degrading bioresistant NAs with higher carbon and Z numbers (Barrow et al. 2010, Gamal El-Din et al. 2011, Martin et al. 2010) and leaving less cyclic and branched NAs that could be ER agonists (He et al. 2011). Interestingly, the selective degradation of NA fractions by ozonation (preferentially $n \geq 22$) and microorganisms (preferentially ≤ 21) leads to the potential for a combination of these two processes. Recent research has shown that ozone pre-treatment of OSPW resulted in the acceleration of microbial degradation of the residual NAs (Hwang et al. 2013, Kannel and Gan 2012, Martin et al. 2010). In addition to ozonation, some other advanced oxidation processes have shown the potential to remove NAs in OSPW (Afzal et al. 2012, Drzewicz et al. 2012, Liang et al. 2011).

Other technologies have also shown potential for OSPW remediation, including coagulation/flocculation/sedimentation (CFS), adsorption, and membrane filtration. CFS was shown to be capable of reducing NA concentrations, especially those with more rings and carbons that have lower solubility (Marr et al. 1996, Pourrezaei et al. 2011). Adsorption using activated carbon alone, or combined with other adsorbents, has been shown to be effective in the removal of organic compounds (e.g., NAs and BTEX) from wastewaters including OSPW (Adhoum and Monser 2004, Allen 2008b, Petrova et al. 2010). Recently, the performance of synthetic copolymers and petroleum coke (PC) in reducing organics via adsorption has also been studied. Copolymer adsorbents containing β-cyclodextrin have displayed favorable sequestration of OSPW-derived NAs, although their adsorption capacity was found to be lower than that of granular activated carbon (Mohamed et al. 2011).

Petroleum coke (PC) is a waste by-product generated during bitumen processing that contains an intrinsic adsorptive capacity. PC has been shown to remove phenol (Zamora et al. 2000) and PAHs from wastewaters and/or aqueous solutions (Yuan et al. 2010). For OSPW treatment, PC alone or combined with zero valent iron has been shown to effectively remove dissolved organic carbon, the acid-extractable fraction (AEF), and NAs, with the resulting effluent being without acute toxicity to *V. fischeri* and rainbow trout (Pourrezaei et al. 2011, Zubot et al. 2012).

Membrane filtration methods (e.g., micellar-enhanced ultrafiltration, nanofiltration, and ceramic membrane filtration) have also shown potential to remove organic contaminants including NAs from synthetic waters mimicking OSPW, actual OSPW, and oilfield produced waters (Alpatova et al. 2014, Deriszadeh et al. 2009, Peng et al. 2004). However, the toxicity of the resulting effluents has not examined.

The high TDS and salinity concentrations of OSPW hamper the reuse of OSPW in process operations and impact the safe discharge of OSPW to the environment. Therefore, there is a

necessity to consider the development of desalination methods for OSPW[3]. Nanofiltration (NF) or reverse osmosis (RO) membranes have been shown to be the most effective methods to remove TDS (Greenlee et al. 2009); however, their applications for OSPW treatment are hindered by membrane fouling caused by high suspended solids (Kim et al. 2011b, 2012). Kim et al. (2011b) recently demonstrated that filtration (both NF and RO) after CFS pre-treatment of OSPW is an effective desalination technology with the addition of the coagulants used in the CFS process, leading to the reduction of membrane fouling and resultant enhancement of membrane performance. A subsequent study revealed that NF and RO filtration of OSPW following a coagulation-flocculation (CF) process (without sedimentation procedure) showed similar benefits; thus the combined processes represent a potential cost-effective and energy-efficient OSPW desalination method (Kim et al. 2012).

Treatment of heavy metals found in OSPW is of concern due to their recalcitrance and persistence in the environment. Current methods that have been shown to be useful for removing heavy metals from wastewaters have been reviewed by Fu and Wang (2011). These technologies include chemical precipitation (hydroxide, and sulphide precipitation), ion-exchanges, adsorption using various adsorbents, membrane filtration (UF, RO, NF, and electrodialysis), CF, flotation and electrochemical methods (Fu and Wang 2011). However, the applications of these methods specific to OSPW treatment have not been studied previously.

Recently, Mahdavi et al. (2013) identified an indigenous green micro-alga (*Parachlorella kessleri*) in OSPW that is responsible for the removal of various metals (i.e., nickel, copper, arsenic, strontium, molybdenum, barium, zinc, manganese, chromium, and cobalt), while the removal rate was dependent on water sources and nutrient concentrations. As mentioned previously, PC has shown promise for the removal of organics from OSPW, but leaches metals into the OSPW (Pourrezaei et al. 2014). Pourrezaei et al. (2014) recently used zero valent iron (ZVI) alone or combined with PC (CZVI), demonstrating an effective removal of vanadium, manganese, nickel, molybdenum, arsenic, cadmium, cobalt, and strontium. Although the removal mechanisms of heavy metals from OSPW by ZVI have not been elucidated, research using groundwaters suggested that the reduction of metals might be based on their adsorption onto the surface of ZVI and co-precipitation with the iron oxy/hydroxides during the oxidation of ZVI (Abedin et al. 2011, Mak and Lo 2011).

Few of the physical, chemical, and biological processes discussed above have been tested at bench- or pilot- scales for OSPW treatment. The challenge for scale-up of these candidate technologies is to meet industrial needs in terms of volumetric flow rates, treatment efficiencies, and overall minimum operating and maintenance costs[4]. In addition, it may be necessary to develop an integrated approach to treat OSPW involving different processes working in parallel or sequentially (e.g., the previously mentioned ozonation/biodegradation combination). In contrast to the limited process scale-up of the previously mentioned methods, the use of *in situ* remediation of large volumes OSPW has been ongoing for decades. Remediation using dry and

[3] OSRIN will be releasing a report on the potential to use forward osmosis for OSPW desalination.
[4] See the following report for more discussion on treatment costs – Godwalt, C., P. Kotecha and C. Aumann, 2010. Oil Sands Tailings Management Project. OSRIN Report No. TR-7. 64 pp. http://hdl.handle.net/10402/era.22536

wet landscapes has been proposed as an option for handling the enormous amount of OSPW. In these two processes, mature fine tailings (MFTs; oil sands process sludges containing 30% to 35% wt% solids) and associated OSPW are used. The wet landscape consists of series of interconnected wetlands that ultimately drain into the end-pit lakes that are created by filling the mined pit with OSPW and freshwater (Allen 2008b, Kennedy 2012). The goal of constructing reclamation wetlands is the production of viable and self-sustaining aquatic ecosystems with functionality equivalent to natural water bodies (Han et al. 2009, Leung et al. 2003). While reduced NAs levels and associated toxicity in OSPW collected from various wetlands have been reported and discussed in the present review, the detoxification process is incomplete and slow. Therefore, the long-term toxicity, among other OSPW contamination issues, need to be taken into consideration when evaluating the practicality of this option at a full scale (Kennedy 2012). However, in the absence of adequate development and scale up of other technologies, wet landscapes are reasonable options due to their economic feasibility and minimal impacts produced on the environment (Allen 2008b).

5 CONCLUSIONS AND RECOMMENDATIONS

5.1 Conclusions

Toxicity to organisms in receiving environments must be taken into account prior to discharging OSPW. OSPW has shown to be toxic to a variety of organisms including microorganisms, invertebrates, fish, amphibians, birds, and mammals, although the exact causative agent(s) and mechanism(s) of toxicity are unknown. OSPW has a variable composition with many organic and inorganic constituents that may play a role in the overall toxicity. Although not the only source, NAs are the most widely reported compounds implicated in the toxicity of OSPW. However, other environmental pollutants of concern include PAHs, BTEX, phenols, dissolved ions and trace metals, may also contribute to and/or affect OSPW toxicity.

The concentrations of BTEX, phenols, and trace metals in OSPW are low; however, they have exceeded CCME water quality guidelines in many cases. While information on toxicity specific to these compounds for OSPW is limited, these compounds have been associated with a wide range of biological dysfunctions in fish and/or mammals, such as alterations in CYP enzyme activities and immune responses, mutagenicity and carcinogenicity, as well as endocrine disrupting effects. Elevated levels of TDS and conductivity in OSPW are also of concern given their ability to induce ionic imbalance in exposed organisms, causing osmotic stress and even leading to mortality.

5.2 Recommendations

OSPW is an extremely complex mixture and more research is needed to evaluate the additive, synergistic, and/or antagonistic effects caused by its many different constituents.

Further study of the potential for OSPW contaminants to biomagnify in the foodchain is needed given the potential threat to the health of higher organisms, including humans.

To date, the aging of tailings ponds and wet landscape remediation relying on natural biodegradation are the only economically-viable methods capable of handling the extremely large volumes of OSPW. However, these options are slow and lead to the incomplete detoxification of OSPW, making them essentially pre-treatment methods prior to further development of other processes. Some alternative technologies for OSPW treatment have been shown to be effective for the rapid removal of target contaminants from OSPW; however, they have only been tested at a small scale. Further work is required to demonstrate whether they will be effective in full-scale applications and how their resulting by-products will affect the receiving environments.

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7 GLOSSARY

7.1 Acronyms

AEF	Acid Extractable Fraction
AhR	Aryl-Hydrocarbon Receptor
AIFs	Apoptosis-Inducing Factors

API	American Petroleum Institute
AR	Androgen Receptor
ATP	Adenosine Triphosphate
ATSDR	Agency for Toxic Substances and Disease Registry
BGL	Brain-Gonad-Liver
BOD	Biological Oxygen Demand
bw	Body Weight
CCME	Canadian Council of Ministers of the Environment
CF	Coagulation Flocculation
CFS	Coagulation Flocculation Sedimentation
COD	Chemical Oxygen Demand
cP	Centipoise
CSS	Cyclic Steam Stimulation
DNA	Deoxyribonucleic Acid
DOC	Dissolved Orgnic Carbon
EC_{50}	Half Maximal Effective Concentration
EDC	Endocrine Disrupting Compound
EPA	Environmental Protection Agency
ER	Estrogen Receptor
EROD	Ethoxyresorufin O-Deethylase
GC	Gas Chromatography
GSI	Gonadosomatic Index
GSHPx	Glutathione Peroxidase
GST	Glutathione S-Transferase
$IC_{25\ (or\ 50)}$	Inhibition Concentration (the level at which the organisms exhibit 25% (or 50%) reduction in a biological measurement such as reproduction or growth)
ICP-MS	Ion Coupled Plasma – Mass Spectrometry
LC_{50}	Lethal Concentration (required to kill 50% of the population)
LD_{50}	Lethal Dose (required to kill 50% of the population)

MFO	Mixed Function Oxygenase
MFT	Mature Fine Tailings
MLSB	Mildred Lake Settling Basin
NF	Nano Filtration
NTU	Nephelometric Turbidity Units
OSPW	Oil Sands Process-Affected Water
OSPW-OF	OSPW Organic Fraction
PC	Petroleum Coke
PRPs	Pattern Recognition Proteins
PRRs	Pattern Recognition Receptors
PW	Produced Water (by-product of offshore oil production)
RO	Reverse Osmosis
ROS	Reactive Oxygen Species
SAGD	Steam Assisted Gravity Drainage
TDS	Total Dissolved Solids
ToF-MS	Time of Flight – Mass Spectroscopy
TS	Total Solids
UF	Ultrafiltration
UV	Ultraviolet
VTG	Vitellogenin
v/v	volume/volume
ZVI	Zero Valent Iron
Λ	Conductivity

7.2 Chemicals

11-KT	11-ketotestosterone
3-MC	3-methylcholanthrene
APs	Alkylated Phenols
BaP	Benzo[a]pyrene
BbF	Benzo[b]fluoranthene
BTEX	Benzene, Ethylbenzene, Toluene, Xylene

-COOH	Carboxylic Acid
C-NAs	Commercial NAs
DB[a,h]A	Dibenz[a,h]anthracene
DBC	Dibenzo[def,p]chrysene
DMBA	7,12-dimethylbenz[a]anthracene
E2	Estradiol
HCO_3^-	Bicarbonate
Na_2SO_4	Sodium Sulphate
NaCl	Sodium Chloride
NADP/NADPH	Nicotinamide-Adenine Dinucleotide Phosphate
$NaHCO_3$	Sodium Bicarbonate
NaOH	Sodium Hydroxide
NAs	Naphthenic Acids
NH_4^+	Ammonium
OCPs	Organochlorine Pesticides
PAEs	Phthalic Acid Esters
PAHs	Polyaromatic Hydrocarbons
S/N	Sulphur/Nitrogen
SO_4^{2-}	Sulphate
T	Testosterone
TCDD	2,3,7,8-tetrachlorodibenzo-p-dioxin

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