TECHNICAL REPORT

Screening Level Human Health Risk Assessment of Recreational Use of Talfourd Creek, Ontario

Ontario Ministry of the Environment and Climate Change

Human Toxicology and Air Standards Section
Standards Development Branch

July 2014

Technical Report

Screening Level Human Health Risk Assessment of Recreational Use of Talfourd Creek, Ontario

Report Prepared by:

O.M. Pagliarulo, M.Sc.

Human Toxicology and Air Standards Section
Standard Development Branch
Ontario Ministry of the Environment and Climate Change

Toronto, Ontario

July 2014

Executive Summary

Talfourd Creek is a shallow creek flowing into the St. Clair River in southern Ontario. It is located south of Sarnia and crosses Aamjiwnaang First Nation (AFN) land.

The Ontario Ministry of the Environment and Climate Change's (MOECC's) Standards Development Branch carried out a screening level human health risk assessment (SLHRA) to assess the potential for adverse health effects from exposure to chemical contaminants in the sediment and water of the creek among people using the creek for recreation. The SLHRA is intended to inform the risk management of the creek for recreation.

Since the focus of this risk assessment was on the risks to people using the creek for recreation, it did not include consideration of risks from other activities or from sources other than the creek water and sediment. Additionally, consumption of fish (or other wildlife) caught in Talfourd Creek was not a part of this risk assessment since information on the creek indicated that sport fish spend most of their time in the St. Clair River. Therefore, risks associated with contaminants in these fish are related to the St. Clair River rather than contaminants present in Talfourd Creek. Residents consuming sport fish from Talfourd Creek should consult the consumption advice for the Upper St. Clair River in the Ontario Ministry of the Environment's Guide to Eating Ontario Sport Fish, available online at www.ontario.ca/environment-and-energy/guide-eating-ontario-sport-fish.

The SLHRA was carried out by first identifying the contaminants to be considered, who would likely be exposed, and how they would be exposed. Subsequently, the potential exposures and toxicities of those contaminants were assessed and used to characterize risk.

Contaminant concentrations in Talfourd Creek sediment and water were screened against health-based criteria and background concentrations, identifying cadmium (Cd), manganese (Mn), titanium (Ti), and polycyclic aromatic hydrocarbons (PAHs) as contaminants of concern (COCs). Two additional COCs, octachlorostyrene and 2,6-dichlorobenzyl chloride, were identified since no screening criteria were available to screen them out.

Conservative exposure estimates for these COCs were calculated by (1) developing a conservative "frequent recreator" exposure scenario involving frequent playing and/or fishing and infrequent accidental immersion in the creek, and (2) using maximum or 95th percentile concentrations of the contaminants in the Talfourd Creek sediment and water. A number of different age groups were included in this assessment but infants (0 to 6 months old) and toddlers (7 months to 4 years old) were not included since these age groups were not anticipated to wander around the creek unsupervised. The activities of the "frequent recreator" were modelled around the main recreational activities occurring along Talfourd Creek and were based on observations and discussions with Aamjiwnaang First Nation and on professional judgement; the activities involve frequent year-round playing and/or fishing and also involve occasional accidental immersion in the creek.

For the risk assessment, the frequent recreator was assumed to be exposed to the maximum or 95th percentile of measured concentrations of contaminants found in the creek. These estimated exposures were compared to reference exposures (toxicity reference values) that reflect negligible risk. From these estimated and reference exposures, Hazard Quotients (HQs)

and Incremental Lifetime Cancer Risks (ILCRs) were calculated for non-cancer and cancer effects, respectively. Calculated HQs less than or equal to 1 and ILCRs less than or equal to 1-in-a-million are considered to indicate negligible risk. HQs exceeding 1 and ILCRs exceeding 1-in-a-million would be flagged for further study.

The child (5-11 years old) was identified as the most sensitive age category since the calculated average daily doses (ADDs) exceeded those of other age categories. The vast majority of a child's exposure was estimated to be from oral and dermal contact with sediment from playing and/or fishing. Some exposure of the child is also estimated to be from dermal contact with sediment resulting from occasional accidental immersion in the creek. Exposures to contaminants in the water of Talfourd Creek were negligible compared to exposures to contaminants in sediment.

Under the conservative frequent recreator exposure scenario using upper estimate concentrations of contaminants, estimated HQs were at or below 0.2 for all COCs and all age categories. The estimated ILCR for polycyclic aromatic hydrocarbons (PAHs) falls at the 1-in-1,000,000 risk level and is thus considered to reflect negligible risk. The application of more realistic exposure assumptions (i.e., in a comprehensive human health risk assessment; HHRA) would result in even lower estimates of risk.

Based on the current SLHRA using conservative exposure assumptions, exposures to contaminants in Talfourd Creek have been estimated to reflect negligible additional risk to a frequent recreator.

It is important to note that this SLHRA considers only the additional or *incremental* risk a person would have from recreational use of Talfourd Creek, not the overall or *absolute* risk a person would have from all sources of exposure to a contaminant in Lambton County.

Plain Language Summary

Talfourd Creek is a shallow creek flowing into the St. Clair River in southern Ontario. It is located south of Sarnia and crosses Aamjiwnaang First Nation (AFN) land.

The Ontario Ministry of the Environment and Climate Change's (MOECC's) Standards Development Branch assessed the risk of exposure to contaminants in Talfourd Creek to people using the creek for recreational activities.

A human health risk assessment is a process for estimating the likelihood that people may experience adverse health effects from exposure to contaminants. Some are screening level risk assessments, relying on available information and using conservative assumptions about the way people are exposed. Others are comprehensive risk assessments, because they rely on more complex information and use more realistic (less conservative) exposure scenarios.

Because they use more conservative assumptions, screening level risk assessments tend to estimate higher risks than comprehensive assessments. Therefore, if a screening level risk assessment does not reveal any elevated risks, contaminants can be ruled out as concerns for human health effects. Contaminants that cannot be ruled out may be further investigated through additional study.

The risk assessment carried out by the MOECC to evaluate the risks to people using Talfourd Creek for recreation was a screening level risk assessment. Since the focus of this risk assessment was on the risks to people using the creek for recreation, it did not include consideration of risks from other activities or from sources other than the creek water and sediment. Additionally, consumption of fish (or other wildlife) caught in Talfourd Creek was not a part of this risk assessment since information on the creek indicated that sport fish spend most of their time in the St. Clair River. Therefore, risks associated with contaminants in these fish are related to the St. Clair River rather than contaminants present in Talfourd Creek. Residents consuming sport fish from Talfourd Creek should consult the consumption advice for the Upper St. Clair River in the Ontario Ministry of the Environment's Guide to Eating Ontario Sport Fish, available online at www.ontario.ca/environment-and-energy/guide-eating-ontario-sport-fish.

The following is a summary of the screening level risk assessment:

The MOECC reviewed available information to identify contaminants that should be considered in the risk assessment. Six contaminants were identified: cadmium (Cd), manganese (Mn), titanium (Ti), polycyclic aromatic hydrocarbons (PAHs), octachlorostyrene and 2,6-dichlorobenzyl chloride.

Subsequently, the MOECC considered which groups of people would likely be exposed and how they would be exposed. A scenario was developed for a hypothetical person who would frequently use the creek for recreation – a "*frequent recreator*" – and would be exposed to contaminants in Talfourd Creek sediment and water. A number of different age groups were included in this assessment but infants (0 to 6 months old) and toddlers (7 months to 4 years old) were not included since these age groups were not anticipated to wander around the creek unsupervised. The activities assumed for the "frequent recreator" were based on information

provided by AFN and professional judgement and included frequent year-round playing and/or fishing and occasional accidental immersion in the creek.

For the risk assessment, the frequent recreator was assumed to be exposed to maximum or close-to-maximum measured concentrations of the contaminants found in the creek. These estimated exposures were compared to reference exposures that reflect negligible risk. Exposures no greater than the reference exposures are considered to be of negligible risk and exposures higher than the reference exposures would be flagged for further study.

Based on the assessment in this screening level risk assessment, exposures to contaminants in Talfourd Creek reflect negligible additional risk to a frequent recreator.

It is important to note that this risk assessment reflects only the *additional* risk a person would have from recreational use of Talfourd Creek, not the total risk a person would have from all sources of exposure to a contaminant in Lambton County.

Table of Contents

1.0 Introduction

Talfourd Creek, located near Sarnia, Ontario, runs through Aamjiwnaang First Nation (AFN) land. Residents have expressed concern over potential environmental impacts to Talfourd Creek from industrial activity in the area. In response to AFN requests, the Ontario Ministry of the Environment (MOE) conducted ecological assessments of water and sediment quality in the Talfourd Creek watershed in 2005, 2007, 2008 and 2010.

In the mid-2000s, signs were posted within AFN land warning people to keep out of Talfourd Creek due to toxic substances and health risks. The current document discusses the human health risk assessment the Ontario Ministry of the Environment and Climate Change (MOECC) has undertaken in order to assess the risks associated with exposures to chemical contaminants in the creek to people using the creek for recreational activities. The assessment is intended to inform the risk management of the creek for recreation. In the context of this assessment, recreation is defined as activity that people do during their free time, which at Talfourd Creek involves mostly fishing, exploring the creek in various ways, and playing (children).

1.1 Description and Approach

A risk assessment was carried out in order to assess the potential for adverse health effects on people exposed to chemical contaminants in Talfourd Creek. Risk assessments are carried out by first scoping the question to be answered and by identifying the contaminants to be considered, who would likely be exposed, and how they would be exposed (problem formulation, Section 2.0). Subsequently, the potential exposures to and toxicities of those contaminants are assessed (Sections 3.0 and 4.0, respectively) and used to characterize risk (Section 5.0). The uncertainties and limitations associated with the SLHRA are discussed in Section 6.0.

This risk assessment was conducted as a “screening level human health risk assessment” (SLHRA), since it relied on available monitoring data for Talfourd Creek as well as anecdotal information regarding recreational use of the creek.

SLHRAs tend to be simpler and more conservative than comprehensive human health risk assessments (HHRAs), which use more complex, detailed, and realistic scenarios.

SLHRAs are used to rule out contaminants as concerns for human health effects. That is, by using conservative assumptions about exposure, the risks estimated in a SLHRA are higher than the risk that would be estimated using a more realistic HHRA process. Therefore, if no elevated risks are identified through the SLHRA, no further action is required; however, if an elevated risk is identified, additional study or analysis (e.g., a comprehensive HHRA or a biomonitoring study) may be carried out to more accurately characterize the risk.

1.2 Scope of the SLHRA

This SLHRA considers only the risks associated with exposure to contaminants in Talfourd Creek during recreational activities. It does not consider the exposure to these contaminants from other sources. That is, the risk assessed in this report is the additional or *incremental* risk a person would have from recreational use of Talfourd Creek, not the overall or *absolute* risk a person would have from other sources of exposure to contaminants in Lambton County.

Furthermore, this SLHRA considers risks associated with exposure to contaminants while fishing in Talfourd Creek but does not consider the exposure to contaminants through ingestion of the fish caught in Talfourd Creek. According to the information reviewed, Talfourd Creek does not support a large sport fishery and any sport fish found in Talfourd Creek are transient and reside mainly in the St. Clair River (MOE, 2011a). In other words, risks from consumption of fish caught in Talfourd Creek are assumed to be related to contaminant issues in the St. Clair River rather than being reflective of the contaminants present in Talfourd Creek. Residents consuming sport fish from Talfourd Creek should consult the consumption advice for the Upper St. Clair River in the Ontario Ministry of the Environment's Guide to Eating Ontario Sport Fish, available online at www.ontario.ca/environment-and-energy/guide-eating-ontario-sport-fish.

The current SLHRA is specific to the risks associated with recreational use of Talfourd Creek and is not reflective of any other risks from living in the Lambton County area.

1.3 Description of Talfourd Creek Area

Talfourd Creek is located in Lambton County in southwestern Ontario, just south of Sarnia. It discharges into the St. Clair River and drains an area of approximately 57 km^2, consisting of agricultural, residential, and heavy industrial land uses; these industries include petroleum refineries and organic chemical manufacturers (MOE, 2011a). Figure 1-1 shows a map of Talfourd Creek in Ontario, based on watershed delineation from MOE, 1992.

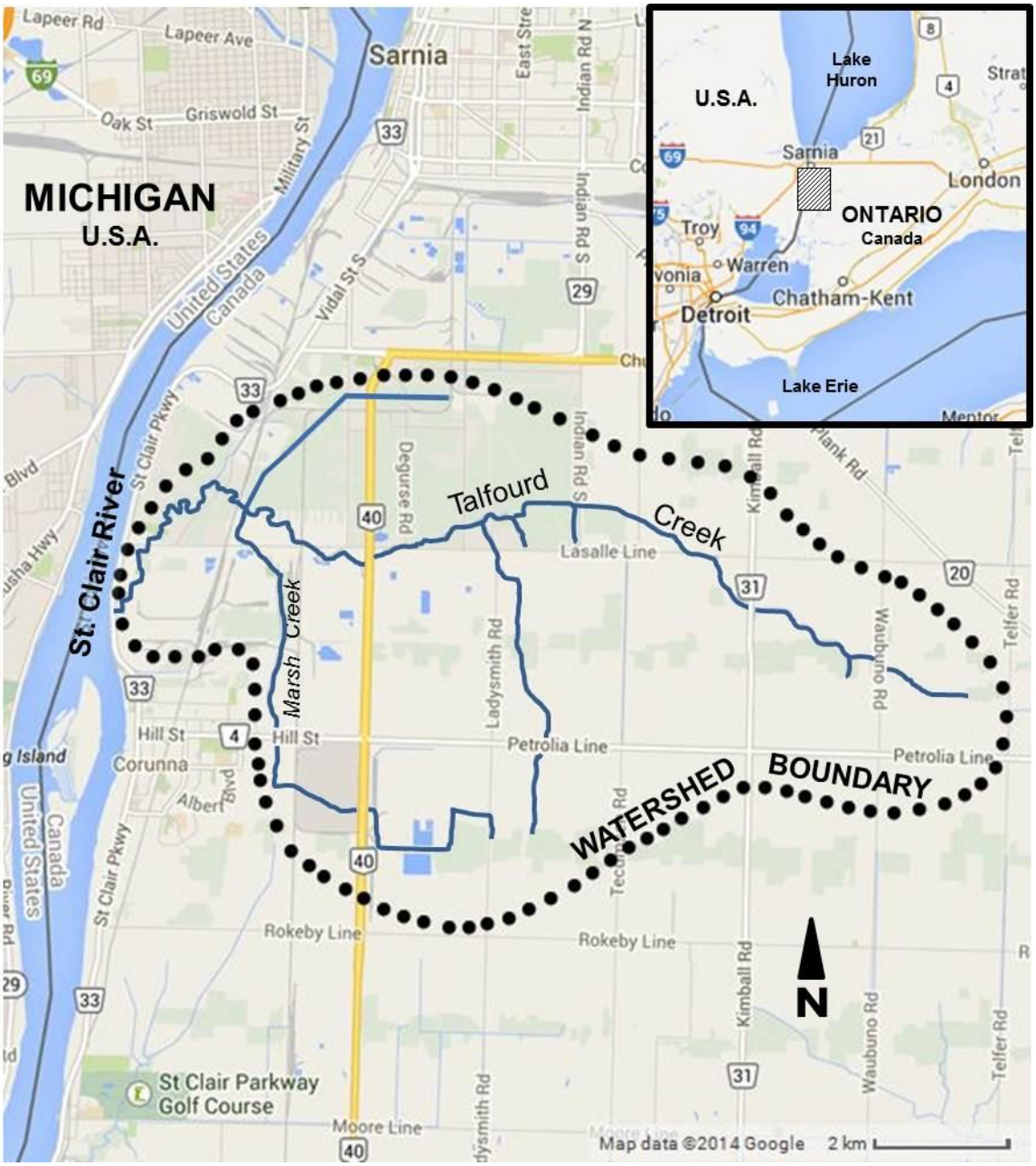
Sediments near the mouth of the creek are likely impacted by sediments moving in and out of the St. Clair River (MOE, 2007; 2009).

Talfourd Creek is generally very shallow and turbid. Several parts of the creek are easily accessible and a variety of types of recreational activities occur along the creek, such as rod fishing near the mouth of the creek and net fishing for bait in various segments of the creek. (However, as noted above, the consumption of fish or wildlife caught in or near Talfourd Creek is not included in this assessment.)

2.0 Problem Formulation

This section is the initial stage of a risk assessment where the contaminated area, the available contaminant data, the people potentially exposed, and the ways people could be exposed are described. Available information was reviewed to identify contaminants that should be considered (Section 2.1: Data Selection and Section 2.2: Contaminant Screening), groups of people who would likely be exposed (Section 2.3: Receptors), and how they would be exposed (Section 2.4 Identification of Exposure Pathways and Section 2.5: Conceptual Site Model).

Figure 1-1: Map of Talfourd Creek and Environs



2.1 Data Selection

Data on concentrations of organic and inorganic contaminants in Talfourd Creek surface sediment (0–3 cm to 0–10 cm) and water were compiled from reports published since 2004 (Atkinson Davies, 2004; Environment Canada, 2004; MOE, 2007; 2009; 2011a). The sample sizes (n) for each contaminant are reported in Tables 2-1 and 2-2. The most recent reports were used because water and sediment are mobile, resulting in changes in contaminant concentrations over time.

Additionally, because of the movement of sediment and water, samples collected from any location along the creek are relevant for use in assessing human health risks in this SLHRA. The limitations associated with using these data are discussed in section 6.0: Uncertainties and Limitations.

2.2 Contaminant Screening – Identification of Contaminants of Concern (COCs)

The process to identify contaminants of concern (COCs) is shown in Tables 2-1 and 2-2 for sediment and water contaminants, respectively, and is summarized as follows:

i. Data were compiled from the five documents (mentioned above) reporting contaminant concentrations in Talfourd Creek sediment and water. From these data, the maximum and 95th percentile concentrations for each contaminant were identified. Contaminants that were consistently reported as non-detects were not included. (Refer to section 6.0 for a discussion of the resultant limitations.) In cases where some but not all values were reported as non-detects, those non-detects were considered to be equal to ½ the detection limit; if the detection limit was not specified, the non-detects were considered to be equal to ½ the lowest detected value. **For the selected COCs, the concentrations used in the contaminant screening were then used in the exposure assessment** (as stated in section 3.1).

For each contaminant, if there were data from at least 20 samples (n ≥ 20), the 95th percentile concentration was used as the upper estimate; if there were data from fewer than 20 samples (n < 20), the maximum concentration was used as the upper estimate.[*]

The 95th percentile was used only where there were sufficient data. Use of a 95th percentile rather than a maximum is based on the assumption that the creek sediment and water are mobile (i.e., do not remain stationary over time) meaning that a receptor at a fixed location would not be exposed to the maximum concentration of a contaminant over the long term. Even if a receptor's exposure location is fixed over time, the contaminant concentrations at that location may shift over time with the movement of the water and sediment.

For non-cancer assessment of the polycyclic aromatic hydrocarbons (PAHs), the upper estimate concentration was determined for each individual PAH and these were summed to obtain an upper estimate concentration for total PAHs. For cancer assessment of PAHs, the upper estimate concentration was determined for each individual carcinogenic PAH and then multiplied by its toxic equivalence factor (TEF) (reported in Kalberlah *et al*., 1995, the source of TEFs used in deriving the MOE, 2011b soil and groundwater standards); these were then summed in order to obtain an upper estimate concentration for total carcinogenic PAHs in benzo(a)pyrene equivalents (BaP_{eq}). Further information regarding the application of TEFs to sum the carcinogenic PAHs is provided in sections 4.1.4.2 and 4.1.4.3.

ii. Contaminants that are either essential elements or are typically found in high concentrations in the earth's crust were not carried forward because of their generally low level of toxicity. These include calcium (Ca), iron (Fe), magnesium (Mg), nitrogen (N), phosphorus (P), potassium (K), sodium (Na), and sulphur (S).

iii. The maximum or 95th percentile contaminant concentrations were compared to human health-based criteria, which are set at concentrations that are protective against adverse health effects. However, for both sediment and surface water, reliable and complete sets of

[*] To compute a 95th percentile, 5% of samples need to be discarded; therefore, 5% of all samples must equal at least one sample. If 5% must be ≥ 1 sample, then the total number of samples (100%) must be ≥ 20 samples.

health-based criteria appropriate for contaminant screening were lacking. Therefore, health-based soil and groundwater (GW) criteria were used, respectively.

a. For sediment, health-based soil criteria were used. Upper estimate sediment contaminant concentrations from Talfourd Creek were screened against MOE (2011b) human health component values (HHCVs) for the S1 pathway (direct soil contact – oral and dermal routes of exposure). HHCVs are set based on 20% of the tolerable daily intake in order to account for potential exposures through other media (air, drinking water, diet, and consumer products). Where S1 HHCVs were lacking, US EPA Region III Risk-Based Concentrations (RBCs) were used (US EPA, 2013). RBCs were used because they are from a recognized health agency, they are derived following a clear and suitable health-based approach, and the list of RBCs is one of the most extensive lists of soil criteria. To be comparable to MOE's HHCVs, these RBCs were adjusted to 20% of the tolerable daily intake.

b. For water, health-based GW criteria were used. Upper estimate water contaminant concentrations from Talfourd Creek were screened against MOE (2011b, Appendix A3) "Groundwater Components for Potable Water Scenario". No values were available for aluminum. For aluminum, since Ontario's drinking water quality standard is not health-based, the drinking water guideline from California Environmental Protection Agency (Cal EPA, 2011) was selected because its derivation follows a clear and suitable health-based approach and all documentation is transparent.

iv. Contaminants that were not eliminated after screening against human health-based criteria were then screened against Ontario background concentrations. However, for both sediment and surface water, reliable and complete sets of background values were lacking and therefore, background data from soil and GW were used:

a. For sediment, MOE (2011b) Table 1 background soil concentrations for residential/parkland land use were used. For contaminants for which no Table 1 background soil value is reported, background soil values were obtained from MOE

(2011b) Table 8.2 (OTR_{98} Old Urban Parks). These values represent 97.5% of data in the "Ontario Typical Range" of background values.

b. For water, MOE (2011b) Table 1 background GW standards were used. For contaminants for which no Table 1 background GW value is reported, background GW values were obtained from MOE (2011b) Tables 8.4 and 8.5 (97.5^{th} percentile values).

Table 2-1 shows the upper estimate contaminant concentrations in Talfourd Creek sediment and the screening values used.

Table 2-1: Identification of Contaminants of Concern (COCs) for Sediment

Contaminant	i.			ii.	iii.	iv.
	n	Upper Estimate Sediment Concentration‡ (µg/g)		Essential Element or High Concentration in Earth's Crust	Human Health-Based Soil Criteria# (µg/g)	Background Soil@ (µg/g)
		95th percentile†	Maximum			
aluminum	34	18,350	24,000		*15,400*	*26,000*
antimony	16	nc	5		7.5	
arsenic	28	10	10.2		0.95	18
barium	37	90.2	100		3800	
beryllium	37	0.9	1.1		38	
boron	16	nc	24		4300	
cadmium	37	2.5	3.3		0.69	1.2
calcium	34	110,500	140,000	√		
chromium (total)	37	30.8	36		28,000	
cobalt	37	11.2	13		22	
copper	37	32.8	56		600	
iron	34	24,100	33,000	√		
lead	37	42.6	282		200	120
magnesium	34	31,350	43,000	√		
manganese	34	537.5	580		*360*	*1400*
mercury	41	0.8	18		9.8	0.27
molybdenum	37	5.2	9.7		110	
nickel	37	38.2	44		330	
nitrogen (Kjeldahl)	12	nc	1300	√		
phosphorus	25	558	580	√		
potassium	13	nc	4500	√		
selenium	28	5	5		110	
silver	16	nc	1		77	
sodium	13	nc	450	√		
strontium	34	113.5	150		*9400*	
sulphur	13	nc	8000	√		
thallium*	16	detected but not quantifiable*			0.29	1
tin	13	nc	2.5		*9400*	
titanium	34	260	280		no value	*4700*
vanadium	37	47	64		39	86
zinc	37	472	790		5600	

Contaminant	i.			ii.	iii.	iv.
	n	Upper Estimate Sediment Concentration‡ (µg/g)		Essential Element or High Concentration in Earth's Crust	Human Health-Based Soil Criteria# (µg/g)	Background Soil@ (µg/g)
		95th percentile†	Maximum			
cyanide (free)	3	nc	0.1		380	
pp-DDE	19	nc	0.048		2.3	
2,6-dichlorobenzyl chloride	8	nc	0.003		no value	no value
diethyl phthalate	3	nc	1.4		94,000	
di-n-butylphthalate	3	nc	0.22		*1220*	
hexachlorobenzene	15	nc	0.014		0.52	
hexachlorobutadiene	11	nc	0.05		7.1	
hexachloroethane	9	nc	0.05		21	
isophorone	3	nc	0.1		*2400*	
mirex	3	nc	0.002		*2.4*	
octachlorostyrene	11	nc	0.2		no value	no value
total PAHs	18-19	nc	5.44		0.078 (BaP)	0.3 (BaP)
carcinogenic PAHs in BaP_{eq}	18-19	nc	0.51		0.078 (BaP)	0.3 (BaP)
PCBs (total)	19	nc	0.11		0.35	
pentachlorobenzene	8	nc	0.005		*9.8*	
1,2,4,5-tetrachlorobenzene	8	nc	0.006		3.6	
1,2,4-trichlorobenzene	11	nc	0.05		210 (1,2,4-trichlorobenzene)	
1,3,5-trichlorobenzene	8	nc	0.005			
petroleum hydrocarbons - heavy oils (C>25)	3	nc	184		6,100	

‡ As described in text of section 2.2, for each contaminant, if n ≥ 20, then 95th percentile concentration was selected as upper estimate; if n < 20, then maximum concentration was selected as upper estimate. Upper estimate selected for each contaminant is underlined.

† nc = not calculated

* Of the 16 thallium sediment sample concentrations reported, 13 were reported as 5 µg/g (MOE, 2011a). A personal communication from Peter Drouin (Laboratory Services Branch, MOE) to Saloni Clerk (Environmental Monitoring & Reporting Branch, MOE) on Nov. 12, 2013 indicated that since the detection limit was 5 µg/g, these results should not be considered quantifiable. (The remaining 3 sample concentrations were reported as below the detection limit of 1 µg/g (Atkinson Davies, 2004)).

MOE (2011b) S1-HHCV; if in italics, criterion is *US EPA Region III Risk-Based Concentration at HQ of 0.2*

@ MOE (2011b) Table 1; if in italics, value is *MOE (2011b) Table 8.2 – Old Urban Parks, OTR98*

Yellow highlighting and **bold font** indicate contaminants identified as a COPCs.

Cadmium (Cd) and polycyclic aromatic hydrocarbons (PAHs) were identified as COCs for sediment because the upper estimate concentrations exceeded both human health-based criteria and background concentrations. Octachlorostyrene and 2,6-dichlorobenzyl chloride were also identified as COCs because no health-based or background screening values are available for these contaminants.

Lead (Pb) was not identified as a COC because the 95th percentile value was only 42.6 µg/g. The sample with the maximum Pb concentration in sediment was measured at 282 µg/g in 2004 immediately downstream of the Bear Park Bridge (Atkinson Davies, 2004). However,

subsequent sampling in the area (sampling sites TALC and TAL2A) reported sediment Pb concentrations in the range of 8 to 10 µg/g (MOE, 2009). Since sediment is mobile, contaminant concentrations are generally expected to move downstream, precluding exposure to any particular concentration over the long term. Further, of the 37 sediment samples collected, only one sample (282 µg/g) was above the background soil concentration of 120 µg/g.

Mercury (Hg) was screened out based on a 95th percentile sediment concentration of 0.8 µg/g. The maximum value was measured at 18 µg/g near the mouth of Talfourd Creek. This location was likely impacted by sediments washing in from the St. Clair River (MOE, 2007). Also, since sediment is mobile, contaminant concentrations are generally expected to move downstream, precluding exposure to any particular concentration over the long term. Further, of the 41 sediment samples collected, only one sample (18 µg/g) was above the soil human health-based criterion of 9.8 µg/g. (The second highest Hg concentration measured was 1.18 µg/g.)

Table 2-2 shows the upper estimate contaminant concentrations in Talfourd Creek water and the screening values used.

Table 2-2: Identification of Contaminants of Concern (COCs) for Water

Contaminant	i.			ii.	iii.	iv.
	n	Upper Estimate Water Concentration‡ (µg/L)		Essential Element or High Concentration in Earth's Crust	Human Health-Based GW Criteria# (µg/L)	MOE Background GW Concentration@ (µg/L)
		95th percentile†	Maximum			
aluminum	17	nc	588		*600*	*86.9*
arsenic	8	nc	0.005		25	
barium	17	nc	78.6		1000	
beryllium	8	nc	0.063		4	
cadmium	17	nc	1.15		5	
calcium	17	nc	108,000	√		
chromium (total)	8	nc	1.52		50	
cobalt	8	nc	1.21		3	
copper	17	nc	13.5		1000	
Iron	17	nc	3050	√		
lead	17	nc	4.25		10	
magnesium	17	nc	33,500	√		
manganese	17	nc	1380		no value	*717*
molybdenum	17	nc	3.82		70	
nickel	17	nc	3.14		100	
nitrogen (Kjeldahl)	17	nc	990	√		
phosphorus	17	nc	311	√		

Contaminant	i.			ii.	iii.	iv.
	n	Upper Estimate Water Concentration‡ (µg/L)		Essential Element or High Concentration in Earth's Crust	Human Health-Based GW Criteria# (µg/L)	MOE Background GW Concentration@ (µg/L)
		95th percentile†	Maximum			
strontium	17	nc	705		no value	*20,200*
titanium	17	nc	46		no value	*4.8*
vanadium	17	nc	3.39		6.2	
zinc	17	nc	42.2		5000	

‡ As described in the text of section 2.2, for each contaminant, if n ≥ 20, then the 95th percentile concentration was used as the upper estimate; if n < 20, then the maximum concentration was used as the upper estimate. The upper estimate used for each contaminant is underlined.

† nc = not calculated

MOE (2011b) GW Component for Potable Water, except the value in italics which was the drinking water guideline from Cal EPA (2011) for aluminum.

@ MOE (2011b) Table 1 Background Groundwater Standards, except values in italics which were MOE (2011b) 97.5th Percentile Background Groundwater Value from Tables 8.4 or 8.5

Yellow highlighting indicates contaminant was identified as a COPC.

Manganese (Mn) and titanium (Ti) were identified as COCs for water because the upper estimate concentrations exceeded background concentrations. (No human health-based water criteria were available for Mn or Ti.)

All COCs identified for either sediment (Table 2-1) or water (Table 2-2) were carried forward and assessed for exposure from both sediment and water; these are Cd, Mn, Ti, PAHs, octachlorostyrene, and 2,6-dichlorobenzyl chloride.

2.3 Identification of Receptors

A site visit was made to Talfourd Creek on August 23, 2013 to discuss the main recreational activities at the creek with an Aamjiwnaang First Nation Environment Committee staff person and to make observations of the creek. The possible recreational activities that could occur in or along Talfourd Creek are described in Table 2-3. This information was collected and compiled in order to inform the development of a recreational exposure scenario. Possible age categories initially considered were the infant (0 – 6 months), toddler (7 months – 4 years), child (5 – 11 years), teen (12 – 19 years), and adult (20+ years).

Table 2-3: Possible Types of Recreational Activities along Talfourd Creek

Age Category	Activity	Likely Frequency
Adult	Fishing with a rod by the mouth of the creek either sitting on the bank or using waders	Roughly twice per week although some fish almost every day
Adult	Bait-fishing with a net along the creek using waders	At most three times per week during the summer
Any age	Strolling along creek	Uncertain but possibly often
Child	Playing along creek	Uncertain but possibly often
Toddler (in daycare)	Looking for benthic organisms in the creek	Very low frequency: twice per year
Toddler	Playing in sediment	Not likely to occur
Any age	Swimming	Not likely to occur because water is generally turbid and shallow and because deeper areas are difficult to access readily.

2.3.1 The Frequent Recreator

Based on the types and frequencies of recreational activities occurring along the creek, a conservative "frequent recreator" was conceived as the hypothetical human receptor in a recreational exposure scenario. The activities of this receptor were based on observations during a site visit, on discussions with an Aamjiwnaang First Nation Environment Committee staff member, and on professional judgement in consideration of the main recreational activities which occur along Talfourd Creek.

The frequent recreator visits Talfourd Creek to play and/or fish along the creek on a frequent and regular basis: 5 days/week, all year round, from childhood through to and including adulthood. (Table 3-2 in the Exposure Assessment shows the parameters describing the frequent recreator.) To account for the possibility of accidental immersion in the creek during recreational activities, the frequent recreator scenario also includes an occasional accidental immersion event.

This receptor includes the child, teen, and adult; it does not include the infant and toddler, because the recreational use of Talfourd Creek by these age categories would be very

infrequent and they would not wander around the creek unsupervised; therefore, their exposures would be minimal compared to other age categories.

Due to the conservative assumptions used in this SLHRA, the calculated exposures of the frequent recreator are expected to exceed actual exposures to anyone partaking in any of the activities considered in Table 2-3. The main assumptions driving the conservative exposure estimates are described in the discussion following Table 3-2.

2.4 Identification of Exposure Pathways

A hypothetical conservative exposure scenario for the frequent recreator described above (a person who visits Talfourd Creek to play/fish in the creek 5 days/week, all year round, from age 5 to 80 years) was created to obtain upper-bound exposure estimates. The selected parameters and assumptions are based on two main activities considered for the frequent recreator:

1) **Playing/fishing along Talfourd Creek**
2) **Accidental immersion in Talfourd Creek while playing/fishing**

The following exposure pathways are assessed for the frequent recreator in this SLHRA:

a) incidental ingestion of sediment b) dermal contact with sediment c) dermal contact with water	Occurring on a frequent basis from **playing/fishing** activities
d) incidental ingestion of water e) dermal contact with water f) incidental ingestion of sediment suspended in water g) dermal contact with sediment suspended in water	Occurring infrequently from **accidental immersion in water**

Total exposure for each age category of the frequent recreator is the sum of all seven pathways described above.

Playing and fishing activities are assumed to occur mainly along the bank of the creek or in shallow water. During each playing/fishing event, the activities involve incidental ingestion of sediment and dermal exposure to sediment and water. In the summer months, playing/fishing

includes dermal exposures of the head, hands, feet, forearms, and lower legs to sediment and water which could occur while wading. During colder seasons, more clothing is expected and therefore less skin is exposed. Water ingestion is not included in the playing/fishing activity because this activity does not comprise frequent swimming in Talfourd Creek. This is considered suitable for the following reasons: (1) Swimming is unlikely to occur in Talfourd Creek based on observations and discussions during the site visit; (2) The accidental immersion event is based on parameters used for assessing exposures from swimming and thereby is also relevant for occasional swimming events; (3) Sediment ingestion rates estimated for in-water activities are much lower than rates for on-land activities (Wilson & Meridian, 2012), and contaminant concentrations in water are much lower than those in sediment; therefore, any additional exposures incurred via accidental ingestion of water would only marginally increase total exposures.

2.5 Conceptual Site Model

An exposure pathway is complete when contaminants from a site reach the receptor. If the pathway is missing either the source, release/transport mechanism, medium, exposure route, or the receptor itself, then it is incomplete and exposure does not occur. (For example, on a residential site, a vapour intrusion pathway would be incomplete if vapour barriers blocked the vapour transport from the ground into a house.) Complete pathways may be assessed quantitatively (using numerical estimates of exposure and toxicity) or qualitatively (using contaminant characteristics, ranges of values, or rankings of values). Incomplete pathways are not assessed since exposures via these pathways do not occur.

A conceptual site model (CSM) represents the exposure pathways by which receptors may be exposed to the COCs. The CSM for this SLHRA is shown in Figure 2-1. In this SLHRA, ingestion of sediment and water and dermal exposure to sediment and water are exposure pathways expected to be significant and are generally assessed quantitatively. Pathways assessed qualitatively are the ingestion of COCs in water from playing/fishing and the inhalation pathways (COCs in both sediment and water, both playing/fishing and accidental immersion). These are discussed following the CSM but are not carried through the risk assessment.

Figure 2-1: Conceptual Site Model (CSM)

PRIMARY SOURCE	RELEASE MECHANISM	EXPOSURE MEDIA	EXPOSURE ROUTE	RECEPTOR: Frequent Recreator	
				PLAYING / FISHING	ACCIDENTAL IMMERSION
historic & current industrial & agricultural activities, discharges of storm water & treated sewage effluent, industrial waste landfill →	discharges, runoff, leaching →	sediment →	ingestion →	√	√
			dermal →	√	√
			inhalation →	□	□
		water →	ingestion →	□	√
			dermal →	√	√
			inhalation →	□	□

Notes: √ = complete exposure pathway
□ = complete pathway but assessed qualitatively

The exposure pathway "ingestion of water from playing/fishing" was considered complete but was assessed qualitatively because the receptors are assumed to be sitting or standing along the bank of the creek or standing/wading in shallow water, rather than swimming or being immersed. Water ingestion while sitting, standing, or wading in shallow water would be minimal, especially with respect to the exposures to sediment quantified below, and would be very difficult to estimate. [Note that ingestion of water is assessed *quantitatively* as part of the occasional accidental immersion event; this exposure pathway is assessed using parameters based on swimming and thus would also be relevant for occasional swimming events.]

The inhalation pathways ("inhalation of contaminants in sediment" and "inhalation of contaminants in water") were considered complete exposure pathways but were assessed qualitatively because they were expected to be minor relative to the ingestion and dermal absorption pathways. Inhalation pathways may be significant in some conditions but these conditions are not present along Talfourd Creek. (e.g., low wind speeds and the presence of deep trenches (MOE, 2011b)). That is, for volatile contaminants (those that tend to partition to air), concentrations may be higher in a trench than in outdoor air at the surface due to reduced mixing with ambient air; for non-volatile contaminants, concentrations may be higher in areas where there is both sufficient water turbulence (causing increasing aerosolization of water

droplets) and decreased air exchange with ambient air. Since these conditions are not present along Talfourd Creek, contaminant air concentrations originating from creek water and/or sediment are not expected to be substantial and thus, inhalation exposure pathways are expected to be negligible with respect to ingestion and dermal pathways.

3.0 Exposure Assessment

This section describes how exposure to the COCs was estimated for each of the relevant sources (water and sediment) and pathways (ingestion, dermal and inhalation).

3.1 Exposure Media Concentrations

For the COCs identified in section 2.2, the upper estimate contaminant concentrations used in the contaminant screening were also used to estimate exposures. These values are shown in Table 3-1. [Note that PAHs are highly lipophilic (dissolve in oil); therefore, water concentrations of PAHs are generally low and water samples are not typically analyzed for PAHs.]

Table 3-1: Concentrations of COCs

Contaminant*		Upper Estimate Concentration	
		Sediment (µg/g)	Water (µg/L)
cadmium (Cd)		2.5	1.15
manganese (Mn)		538	1380
titanium (Ti)		260	46
polycyclic aromatic hydrocarbons (PAHs)	carcinogenic PAHs as BaP_{eq}	0.51	not reported
	total PAHs	5.4	not reported

Octachlorostyrene and 2,6-dichlorobenzyl chloride are not included here because they do not have suitable toxicity reference values (TRVs) to permit a typical quantitative assessment. Exposures to these contaminants will be assessed qualitatively.

3.2 Receptor Characterization – Assumptions and Parameters Used

The receptor assessed in this SLHRA is the frequent recreator, as described in section 2.2. The selected assumptions and receptor parameters are based on the two main activities for this

receptor: playing/fishing along Talfourd Creek and accidental immersion in the creek while playing/fishing.

All receptor parameters selected for this SLHRA are reported in Table 3-2, with the details and rationale pertaining to their selection described in sections 3.2.1 to 3.2.12. The age ranges and categories were based on those used in developing the MOE (2011b) soil and GW standards. (Section 5.1 discusses the pregnant female receptor and associated parameters used to assess risk of developmental toxicity.)

Table 3-2: Receptor Parameters

Parameter (unit)	Symbol	Child (5-11 y)	Teen (12-19 y)	Adult (20+ y)	Level of Conservatism[a]	Source
Body Weight (kg)	BW	32.9	59.7	70.7	CT	Richardson, 1997
Sediment Ingestion Rate (g/d)	SIR	0.12	0.04	0.04	C	Wilson & Meridian, 2011; 2012
Skin Surface Area[b] (cm^2)	SSA_1	2264	3364	3778	C	Richardson, 1997; professional judgement
	SSA_2	10,140	15,740	17,640	C	Richardson, 1997
Sediment Adherence Factor ($mg/cm^2/d$)	SAF	1.19	0.11	0.11	CT	US EPA, 2011; professional judgement
Exposure Frequency[b] (d/y)	EF_1	260			C	professional judgement
	EF_2	13			C	professional judgement
Water Ingestion Rate (L/d)	WIR	0.09	0.09	0.09	C	US EPA, 2011
Total Suspended Sediment (mg/L)	TSS	144			C	MOE, 2007
Exposure Duration (y)	ED	7	8	60	n/ap	MOE, 2011b; HC 2012
Averaging Time for non-cancer (y)	AT	7	8	60	n/ap	
Averaging Time for cancer (y)	$AT_{lifespan}$	80			n/ap	
Duration that skin remains wet (h/d)	t_{event}	6			C	professional judgement
Duration of sediment adherence to skin (h/d)	(not used)	24			C	US EPA, 2004
Relative Absorption Factor (unitless)	RAF	contaminant-specific				See Toxicity Assessment section
Dermal permeability coefficient in water (cm/h)	K_P	contaminant-specific				US EPA, 2004; see Toxicity Assessment section

[a] CT = central tendency; C = conservative; n/ap = not applicable
[b] Parameters have different values based on type of activity: (1) playing/fishing, or (2) immersion in creek.

The main assumptions driving the conservative exposure estimates for the frequent recreator in this SLHRA are as follows:

- Playing or fishing activities are assumed to occur along Talfourd Creek 5 days/week, all year round (52 weeks/year).
- During each playing/fishing event, the receptor ingests sediment and water and has all exposed body parts (body parts not covered by clothing) covered with sediment and water.
- One of every 20 playing/fishing events is accompanied by accidental immersion in the creek, involving additional exposures from ingestion and from whole-body dermal exposure.
- During every exposure to sediment and water, contaminants are at upper estimate concentrations (95th percentiles or maxima).

3.2.1 Body Weight (BW)

The body weights (BWs) used in this SLHRA are averages from Richardson (1997) and are specific to the Canadian population; they are the same BWs used in the development of the MOE (2011b) soil and groundwater (GW) standards. The selected central tendency BW values are 32.9, 59.7, and 70.7 kg for the child, teen, and adult, respectively.

3.2.2 Sediment Ingestion Rate (SIR)

Estimated rates of sediment ingestion for use in human health risk assessments (HHRAs) are limited in the literature. *Soil* ingestion rates have historically been used in risk assessments as a surrogate for sediment ingestion, but it is unclear whether this is a conservative assumption; on one hand sediment has greater skin adherence but on the other hand soil exposures are expected to have higher contact durations than sediment (Wilson & Meridian, 2011). Since soil ingestion rates are commonly used in HHRAs as a surrogate for sediment ingestion, data on both soil ingestion and sediment ingestion were considered here.

For the development of the MOE (2011b) soil standards, a soil ingestion rate of 200 mg/day was used for the toddler – a conservative value based on US EPA (1997; 2008) analyses of data from several tracer studies where 100 mg/day was considered a central tendency value.

For the adult, the MOE soil standards used 50 mg/day (based on US EPA, 1997 analyses); the adult rate was also used for the school-aged child (5 – 11 years old) and the teen based on the assumption that their behaviour and soil ingestion rates are more similar to an adult than a toddler. Soil ingestion rates for toddlers (7 months to 4 years old) are higher than for other age categories because they exhibit behaviours (e.g., hand-to-mouth activity) that increase exposure to media such as soil (MOE, 2011b).

Health Canada (2012) recommends residential soil ingestion rates of 20 mg/day for infants, children, teens, and adults and 80 mg/day for toddlers.

For soil and dust combined, US EPA (2011) recommends central tendency intake rates of 100 mg/day for the child and teen and 50 mg/day for the adult, based on an analysis of data from several tracer element studies; for the toddler, 3–5 years old, a general population upper percentile of 200 mg/day is recommended.

The only recommended sediment ingestion rates available are provided by Wilson & Meridian (2011; 2012) in contractor reports to Health Canada's Contaminated Sites Division. Wilson & Meridian (2011) developed central tendency sediment intake rates of 78, 25, and 28 mg/day for the child, teen, and adult, respectively, based on an analysis of estimated intakes from hand-to-mouth contact with sediment and incidental ingestion of water containing sediment. In a follow-up report to, Wilson & Meridian (2012) proposed average sediment ingestion rates of 90 – 120 mg/day for the child and 30 – 40 mg/day for the teen and adult. For contact with suspended sediment during in-water activities only, Wilson & Meridian (2012) proposed mean sediment ingestion rates of 10 – 20 mg/day for all age categories. The authors recommend that the high end of these ranges be used for beaches, tidal flats, and riverbeds that are known to be used regularly by the general public.

For the playing/fishing activity in this SLHRA – which is presumed to occur predominantly on land rather than in water – sediment ingestion rates (SIRs) of 120 mg/day (0.12 g/day) for the child and 40 mg/day (0.04 g/day) for the teen and adult were selected based on the high end of the ranges proposed by Wilson & Meridian (2012) for on-land activities. Although the SIR rates selected from Wilson & Meridian (2012) are those proposed for on-land activities, they are

within the ranges proposed for the combination of both on-land and in-water activities. For accidental immersion events, the pathway of ingestion of suspended sediment was estimated using site-specific data (See sections 3.2.7 and 3.3.2 for details.) rather than using the sediment ingestion rates proposed by Wilson & Meridian (2012) for in-water activities.

3.2.3 Skin Surface Area (SSA)

For residential exposures, US EPA (2004) recommends exposed skin surface area (SSA) to be limited to:

- child: head, hands, forearms, lower legs, and feet
- adult: head, hands, forearms, and lower legs

US EPA (2004) does not have similar recommendations for the teen. For all age categories in this SLHRA, the body parts available for dermal exposure are based on US EPA (2004) recommendations for the child resident and on anticipated clothing considerations and behaviour. Based on the seasons, the frequent recreator's body parts assumed to be exposed are as follows:

- summer: head, hands, forearms, lower legs, and feet
- spring and autumn: head, hands, and forearms
- winter: hands only

SSA values for child, teen, and adult receptors were obtained from Richardson (1997) and are specific to the Canadian population. Since Richardson (1997) does not report SSAs for the head, these were estimated: From the age of 2 years and older, the SSA of the head is roughly equivalent to that of one arm (US EPA, 2004). Furthermore, based on professional judgement, the forearm is assumed to be half of the arm and the lower leg is assumed to be half of the leg.

The resultant SSAs for playing/fishing activities are time-weighted mean SSAs based on year-round exposures; the calculation of these values is shown in Table 3-3.

Table 3-3: Calculation of Time-Weighted Average Skin Surface Areas

	Body Part	Skin Surface Area (cm^2) by Age Category		
		Child	**Teen**	**Adult**
Values obtained from Richardson (1997)	arms	1480	2230	2500
	hands	590	800	890
	legs	3070	4970	5720
	feet	720	1080	1190
Assumption-Based Values	*head*	740	1115	1250
	forearms	740	1115	1250
	lower legs	1535	2485	2860
Summer	head, hands, forearms, lower legs, & feet	**4325**	**6595**	**7440**
Spring	head, hands, forearms	**2070**	**3030**	**3390**
Autumn	head, hands, forearms	**2070**	**3030**	**3390**
Winter	hands only	**590**	**800**	**890**
Time-weighted average skin surface area for playing/fishing (cm^2)		**2264**	**3364**	**3778**

For the playing/fishing activity, the time-weighted average SSAs of 2264, 3364, and 3778 cm^2 were used for the child, teen, and adult, respectively. For dermal exposure to sediment and water from playing/fishing, these values may be considered to be conservative because they are based on the assumption that all uncovered body parts are in contact with sediment and water on each day of activity at the creek. For sediment, these values may also be considered conservative because they are based on the assumption that sediment remains adhered to all exposed skin for 24 hours on each day of activity at the creek.

For the immersion events, SSA of the whole body was used for all seasons of the year: 10,140 cm^2 (child), 15,740 cm^2 (teen), and 17,640 cm^2 (adult) (Richardson, 1997). For dermal exposure to water from accidental immersion in the creek, these values may be considered central tendency estimates because upon immersion in the creek, the whole body would be immersed and in contact with water. For dermal exposure to sediment from accidental immersion in the creek, these values may be considered conservative because clothing would block the skin from exposures to sediment and because they are based on the assumption that

sediment remains on the skin of the whole body for 24 hours on each day of activity at the creek.

3.2.4 Sediment Adherence Factor (SAF)

In order to calculate exposure to contaminants in sediment via dermal contact, it is necessary to estimate the amount of sediment which adheres to the skin, known as sediment loading or sediment adherence. As with sediment ingestion, information on both soil and sediment adherence were considered here.

For *soil* adherence to skin, Health Canada (2012) Guidance on Human Health Preliminary Quantitative Risk Assessment recommends 0.1 mg/cm^2/event for hands and 0.01 mg/cm^2/event for the rest of the body based on soil studies by Kissel *et al*. (1996; 1998).

Wet soil is roughly similar to sediment. For the child 8–12 years old playing in wet soil, US EPA (2004) guidance for dermal risk assessment recommends 0.2 mg/cm^2/day (geometric mean) based on studies of children playing with toys in wet soil (Kissel *et al*., 1998). For adult gardeners over 16 years old, US EPA (2004) recommends a *soil* adherence rate of 0.07 mg/cm^2/day (geometric mean).

For the child playing in sediment, US EPA's Exposure Factors Handbook (US EPA, 2011) recommends adherence factors of 0.040 to 21 mg/cm^2 (based on geometric means of children 7–12 years old playing in tidal flats; Shoaf *et al*., 2005), depending on the body part exposed. For the adult engaged in outdoor sports or activities with soil, US EPA (2011) recommends 0.0314 to 0.1595 mg/cm^2 (based on geometric means from Kissel *et al*., 1996 and Holmes *et al*., 1999).

For use in the current SLHRA, sediment adherence factors were weighted by body part and by season (Table 3-4). Adherence factors selected for the child were obtained from US EPA (2011) for children playing in sediment (based on Shoaf *et al*., 2005). Adherence factors selected for the adult were the higher of adults engaged in outdoor sports and adults engaged in

activities with soil (US EPA, 2011). The teen was assumed to have the same adherence factors as the adult.

Overall adherence factors were weighted by body part surface area to obtain season-specific adherence factors. For each age category, the season-specific adherence factors were averaged to obtain a time-weighted adherence factor. The resultant time-weighted sediment adherence factors used in this SLHRA are provided in Table 3-4. The current SLHRA uses the conservative assumption that recreators are barefooted during every day of recreation in the summer.

Table 3-4: Calculation of Time-Weighted Sediment Adherence Factors for Skin

		Sediment Adherence Factor (mg/cm^2) (*and surface area of body part: cm^2*)		
		Child	Teen	Adult
Adherence factors recommended by US EPA (2011), by body part	hands	0.49 (*590*)	0.1595 (*800*)	0.1595 (*890*)
	feet	21 (*720*)	0.1393 (*1080*)	0.1393 (*1190*)
	head	0.040 (*740*)	0.0314 (*1115*)	0.0314 (*1250*)
	forearms	0.17 (*740*)	0.0872 (*1115*)	0.0872 (*1250*)
	lower legs	0.70 (*1535*)	0.1223 (*2485*)	0.1223 (*2860*)
Adherence weighted by body part surface area	SUMMER: head, hands, forearms, lower legs + feet	3.85	0.11	0.11
	SPRING: head, hands + forearms	0.21	0.09	0.09
	AUTUMN: head, hands, + forearms	0.21	0.09	0.09
	WINTER: hands only	0.49	0.1595	0.1595
Time-Weighted Adherence Factors (weighted by body part & season)		**1.19**	**0.11**	**0.11**

3.2.5 Exposure Frequency (EF)

According to discussions with the Aamjiwnaang First Nation Environment Committee staff person, people generally fish in Talfourd Creek roughly 2 or 3 days per week, some almost every day (See Table 2-3). For this SLHRA, an exposure frequency of 5 days/week all year round was selected for the playing/fishing activity (EF_1), i.e., 260 days/year. Although this rate of activity may be attained by some people during the summer, it is unlikely during colder months. Also, as noted above, fishing activities in Talfourd Creek are observed to occur 2 or 3 times per week, on average. Therefore, an EF_1 of 260 days/year may be considered a conservative estimate

For the accidental immersion events, it was conservatively assumed that anyone engaging in the playing/fishing activity would be immersed in the creek once per 20 playing/fishing days. Since the selected EF_1 was 260 days/year, the selected exposure frequency for immersion in the creek (EF_2) was 13 days/year (all year round). Since accidental immersion events are not likely to occur that often, this may be considered a conservative assumption.

3.2.6 Water Ingestion Rate (WIR)

Each time a person is immersed in the creek, it is assumed that this person will ingest creek water. For water ingested while swimming, US EPA (2011) recommends means of 37 and 16 mL/event and upper percentiles of 90 and 53 mL/event for the child and adult, respectively, obtained from a study by Dufour *et al*. (2006) where participants swam for 45 minutes.

For this SLHRA, a water ingestion rate (WIR) of 90 mL/day (0.09 L/day) was selected for all age categories. The selected WIR may be considered conservative for two reasons: (1) It is based on an upper percentile WIR; (2) the WIR for a swimming event lasting 45 minutes is allotted to a brief immersion event, and (3) although 90 mL/day was selected for the adult, the maximum WIR for adults in the Dufour *et al*. (2006) study was only 53 mL/event.

3.2.7 Total Suspended Sediment (TSS)

Each time a person is immersed in the creek, it is assumed that this person will ingest creek water and the sediment which is suspended in that water. Two MOE monitoring studies (MOE, 2007; 2009) reported total suspended sediment (TSS) measured in Talfourd Creek. MOE (2007) reported TSS values from 9.8 to 144 mg/L (n=9). MOE (2009) reported TSS values from 3.9 to 24.5 mg/L (n=9). For this SLHRA, the maximum reported TSS of 144 mg/L was selected. Since this is a maximum value, it may be considered a conservative assumption. (A general rule of thumb is that water with a total suspended solids concentration of <20 mg/L is clear, 40 – 80 mg/L is cloudy, and >150 mg/L appears brown and opaque.)

3.2.8 Exposure Duration (ED) and Averaging Time (AT)

Exposures are considered to occur from childhood throughout the receptor's average lifetime of 80 years. According to standard risk assessment practice for estimating risks for non-cancer effects, the exposure duration (ED) and averaging time (AT) for each age category are based on the duration of the age category. This SLHRA uses the age categories, exposure durations, and averaging times that were used in the derivation of MOE's (2011b) soil and GW standards. [Note that since ED and AT have equal values for each age category, they negate each other in the calculations of average daily dose (ADD); they are thus not shown in Equations 1–8.]

To estimate cancer risks, the calculation of the lifetime average daily dose (LADD) requires the use of an AT for the entire lifespan. $AT_{lifespan}$ = 80 years based on Health Canada (2012) guidance on human health preliminary quantitative risk assessment.

ED and AT are fixed values because they are part of the definition of each age category. For example, a person is considered a teen when aged between 12 and 19 years old (MOE, 2011b; HC 2012); ED and AT for the teen are 8 years and do not change between HHRAs evaluating long-term exposures. Since these values are essentially fixed and based on the definitions of the age categories, they are not considered either central tendency or conservative assumptions.

3.2.9 Duration that Skin Remains Wet (t_{event})

Uncovered skin may be exposed to water in Talfourd Creek from playing/fishing or from accidental immersion in the creek. To assess dermal exposure to contaminants in water requires a duration that the skin remains wet. For this SLHRA, it is assumed that the skin remains wet for 6 hours/day when the activity or event occurs (professional judgement). It is possible that some exposed skin may remain wet for longer durations; however, a t_{event} of 6 hours/day may be considered conservative for playing/fishing and for accidental immersion because it assumes that the skin of every exposed body part remains wet for 6 hours each time the activity or event occurs.

3.2.10 Duration of Sediment Adherence to Skin

According to standard risk assessment practice, this SLHRA assumes that sediment adheres to the skin for 24 hours. Dermal absorption of a contaminant depends on time, but data in the scientific literature are insufficient to determine the kinetics of absorption over time; therefore site-specific exposure scenarios should not scale dermal absorption (US EPA, 2004). This assumption may be considered conservative, but it is supported by considerations that recreators may not have the opportunity for washing until well after their activity at Talfourd Creek. In some extreme cases, lodged sediment or sediment with strong adherence to the skin may not be eliminated until after a thorough wash.

3.2.11 Dermal Permeability Coefficient for Contaminants in Water (K_P)

Dermal permeability coefficients for contaminants in water (K_P) are contaminant-specific values related to the toxicokinetics of the contaminants. K_P is the rate at which a contaminant in water permeates the skin and is used to estimate a dermally absorbed dose for contaminants in water. The K_P value of a contaminant can be estimated using a contaminant's K_{OW} (oil/water partition coefficient) and molecular weight or can be estimated through experiment with human skin *in vitro* (US EPA, 2004). US EPA (2004) provides K_P values for several contaminants; for Cd, Mn, Ti, and PAHs, K_P is 1×10^{-3} cm/h.

3.2.12 Relative Absorption Factors (RAFs)

Absorption is the proportion of a contaminant contacted that gets absorbed. Absorption may change with the receptor (human or test animal), the contaminated medium, the route of exposure, and the contaminant itself. To make adjustments for these dissimilarities, a relative absorption factor (RAF) is calculated as a ratio comparing the absolute absorption of the contaminant in the exposure pathway being assessed with the absolute absorption in the key toxicity study that forms the basis of the selected TRV. Further information on RAFs is provided in Section 4.0: Toxicity Assessment.

3.3 Estimation of Exposure Rates

The total average daily dose (ADD) is the total exposure incurred by each receptor and includes exposures from several pathways from both the playing/fishing activity and the accidental immersion event. For each of the COCs and each receptor age category (child, teen, and adult), the ADD was estimated for each pathway of the playing/fishing activity and accidental immersion event. These ADDs were then summed to obtain a total ADD of each age category. The total ADD of each age category was used in the risk characterization section to estimate risk of non-cancer health effects. Lifetime average daily dose (LADD) was also calculated for the PAHs.

In all equations and scenarios that follow, parameter values are provided for the child as sample calculations. Calculations were repeated using the appropriate parameter values for the teen and adult receptor. Sample calculations are provided using manganese (Mn) for ADD calculations and polycyclic aromatic hydrocarbons (PAHs) for LADD calculations.

3.3.1 Calculation of ADD for Playing/Fishing in Talfourd Creek on a Regular Basis

Exposures to all COCs from playing/fishing in Talfourd Creek on a regular basis throughout the year were estimated for the child, teen, and adult using the following equations. Exposure pathways include incidental ingestion of sediment and dermal exposure to sediment and water.

$$ADD_{sed\text{-}oral} = \frac{conc_S \times SIR \times RAF_{OS} \times EF_1}{BW \times CF_a}$$ (Equation 1)

$$ADD_{sed\text{-}derm} = \frac{conc_S \times SAF \times SSA_1 \times RAF_D \times EF_1}{BW \times CF_a \times CF_b}$$ (Equation 2)

$$ADD_{water\text{-}derm} = \frac{conc_W \times K_P \times t_{event} \times SSA_1 \times RAF_D \times EF_1}{BW \times CF_a \times CF_c}$$ (Equation 3)

$$ADD_1 = ADD_{sed\text{-}oral} + ADD_{sed\text{-}derm} + ADD_{water\text{-}derm}$$ (Equation 4)

ADD_1 = total average daily dose of COC from **playing/fishing** (µg/kg/d)
$ADD_{sed\text{-}derm}$ = average daily dose from dermal exposure to sediment (µg/kg/d)
$ADD_{sed\text{-}oral}$ = average daily dose from incidental ingestion of sediment (µg/kg/d)
$ADD_{water\text{-}derm}$ = average daily dose from dermal exposure to water (µg/kg/d)
BW = body weight (for the child: 32.9 kg)
CF_a = unit conversion factor (365 d/y)
CF_b = unit conversion factor (1000 mg/g)
CF_c = unit conversion factor (1000 cm^3/L)
$conc_S$ = concentration of COC in sediment (for Mn: 538 µg/g)
$conc_W$ = concentration of COC in water (for Mn: 1380 µg/L)
EF_1 = exposure frequency for playing/fishing (260 d/y)
K_P = dermal permeability coefficient of COC in water (for Mn: 0.001 cm/h)
RAF_D = dermal relative absorption factor for COC (for Mn: 0.01, unitless)
RAF_{OS} = oral relative absorption factor for COC in sediment (for Mn: 1, unitless)
SAF = sediment adherence factor (for the child: 1.19 mg/cm^2/d)
SIR = sediment ingestion rate (for the child: 0.12 g/d)
SSA_1 = skin surface area exposed from playing/fishing (for the child: 2264 cm^2)
t_{event} = duration that skin remains wet from playing/fishing (6 h/d)

In Equation 4, all doses incurred from playing/fishing in Talfourd Creek on a regular basis were summed: oral exposure to sediment, dermal exposure to sediment, and dermal exposure to water. For the child engaged in playing/fishing in Talfourd Creek, exposures to Mn in sediment and water are summarized as follows:

$ADD_{sed\text{-}oral}$ = 1.40 µg/kg/d,

$ADD_{sed\text{-}derm}$ = 0.314 µg/kg/d

$ADD_{water\text{-}derm}$ = 0.0041 µg/kg/d

Total ADD for playing/fishing (Equation 4) is the sum of these ADD values: 1.7 µg/kg/d for Mn.

3.3.2 Calculation of ADD for Occasional Accidental Immersion in Talfourd Creek

Exposures to all COCs from occasional accidental immersion in Talfourd Creek throughout the year were estimated for the child, teen, and adult using the following equations. Exposure pathways include incidental ingestion of water and sediment and dermal exposure to water and sediment.

$$ADD_{water\text{-}oral} = \frac{conc_W \times WIR \times RAF_{OW} \times EF_2}{BW \times CF_a} \quad \text{(Equation 5)}$$

$$ADD_{sed\text{-}oral} = \frac{conc_S \times WIR \times TSS \times RAF_{OS} \times EF_2}{BW \times CF_a \times CF_b} \quad \text{(Equation 6)}$$

$$ADD_{water\text{-}derm} = \frac{conc_W \times K_P \times t_{event} \times SSA_2 \times RAF_D \times EF_2}{BW \times CF_a \times CF_c} \quad \text{(Equation 7)}$$

$$ADD_{sed\text{-}derm} = \frac{conc_S \times SAF \times SSA_2 \times RAF_D \times EF_2}{BW \times CF_a \times CF_b} \quad \text{(Equation 8)}$$

$$ADD_2 = ADD_{water\text{-}oral} + ADD_{sed\text{-}oral} + ADD_{water\text{-}derm} + ADD_{sed\text{-}derm} \quad \text{(Equation 9)}$$

ADD_2 = total average daily dose of COC from **accidental immersion in Talfourd Creek** (µg/kg/d)
$ADD_{sed\text{-}derm}$ = average daily dose from dermal exposure to sediment (µg/kg/d)
$ADD_{sed\text{-}oral}$ = average daily dose from incidental ingestion of sediment (µg/kg/d)
$ADD_{water\text{-}derm}$ = average daily dose from dermal exposure to water (µg/kg/d)
$ADD_{water\text{-}oral}$ = average daily dose from incidental ingestion of water (µg/kg/d)
BW = body weight (for the child: 32.9 kg)
CF_a = unit conversion factor (365 d/y)
CF_b = unit conversion factor (1000 mg/g)
CF_c = unit conversion factor (1000 cm^3/L)
$conc_S$ = concentration of COC in sediment (for Mn: 538 µg/g)
$conc_W$ = concentration of COC in water (for Mn: 1380 µg/L)
EF_2 = exposure frequency of accidental immersion in Talfourd Creek (13 d/y)
K_P = dermal permeability coefficient of COC in water (for Mn: 0.001 cm/h)
RAF_D = dermal relative absorption factor for COC (for Mn: 0.01, unitless)
RAF_{OS} = oral relative absorption factor for COC in sediment (for Mn: 1, unitless)
RAF_{OW} = oral relative absorption factor for COC in water (for Mn: 1, unitless)
SAF = sediment adherence factor (for the child: 1.19 mg/cm^2/d)
SSA_2 = skin surface area exposed from accidental immersion in Talfourd Creek (for the child: 2264 cm^2)

t_{event} = duration that skin remains wet from accidental immersion in Talfourd Creek (6 h/d)
TSS = total suspended sediment (144 $mg_{sediment}/L_{water}$)
WIR = water ingestion rate (0.09 L/d)

As shown in Equation 9, doses were summed for dermal and oral exposures to sediment and water incurred from occasional accidental immersion in Talfourd Creek. For the child accidentally immersing into Talfourd creek, exposures to Mn in sediment and water are summarized as follows:

$ADD_{water\text{-}oral}$ = 0.134 µg/kg/d,

$ADD_{sed\text{-}oral}$ = 0.0075 µg/kg/d,

$ADD_{water\text{-}derm}$ = 0.000 91 µg/kg/d

$ADD_{sed\text{-}derm}$ = 0.070 µg/kg/d

Total ADD from occasional accidental immersion in the creek (Equation 9) is the sum of these ADD values: 0.21 µg/kg/d for Mn.

3.3.3 Calculation of Total ADD for the Sum of Exposures

As shown in Equation 10, the total estimated ADD for activities at Talfourd Creek is the sum of the total ADD from playing/fishing and the total ADD from accidental immersion in creek. The total ADD for each COC was estimated for the child, teen, and adult using the following relationship:

$$\boxed{ADD_{Total} = ADD_1 + ADD_2} \qquad \text{(Equation 10)}$$

ADD_1 = total average daily dose of COC from playing/fishing in Talfourd Creek (µg/kg/d)
ADD_2 = total average daily dose of COC from accidental immersion in Talfourd Creek (µg/kg/d)
ADD_{Total} = total average daily dose of COC from both activities (µg/kg/d)

For the child exposed to Mn in Talfourd Creek sediment and water,

ADD_{Total} = 1.7 µg/kg/d + 0.21 µg/kg/d = 1.9 µg/kg/d.

The ADD_{Total} for each COC was estimated for each age category (child, teen, and adult).

3.3.4 Contribution of Various Exposure Pathways to Total Recreational Intake

In order to determine the relative contribution of each exposure pathway, the average daily dose (ADD) of each pathway is compared to the total ADD. Since the child category had the highest total ADD for each COC (as shown in Table 5-1 in the Risk Characterization section), the relative contribution of each pathway for the child was determined for cadmium (Cd), manganese (Mn), titanium (Ti), and polycyclic aromatic hydrocarbons (PAHs). These relationships are illustrated in Figures 3-1 to 3-4.

Figure 3-1: Contribution of Exposure Pathways to Recreational Intake of Cd – Child

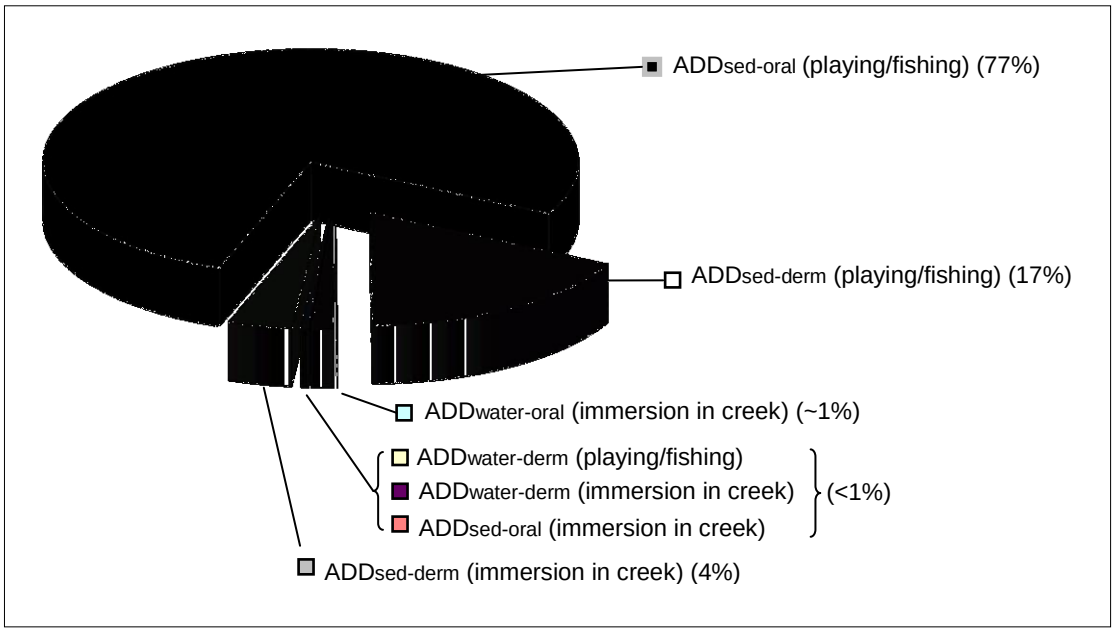


Figure 3-2: Contribution of Exposure Pathways to Recreational Intake of Mn – Child

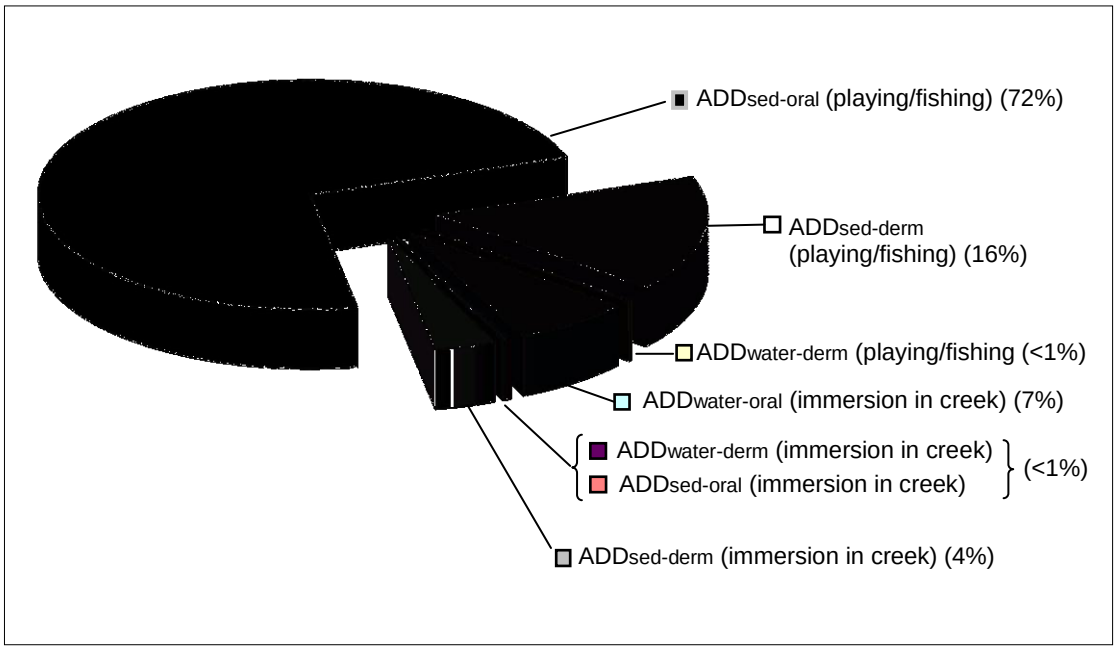


Figure 3-3: Contribution of Exposure Pathways to Recreational Intake of Ti – Child

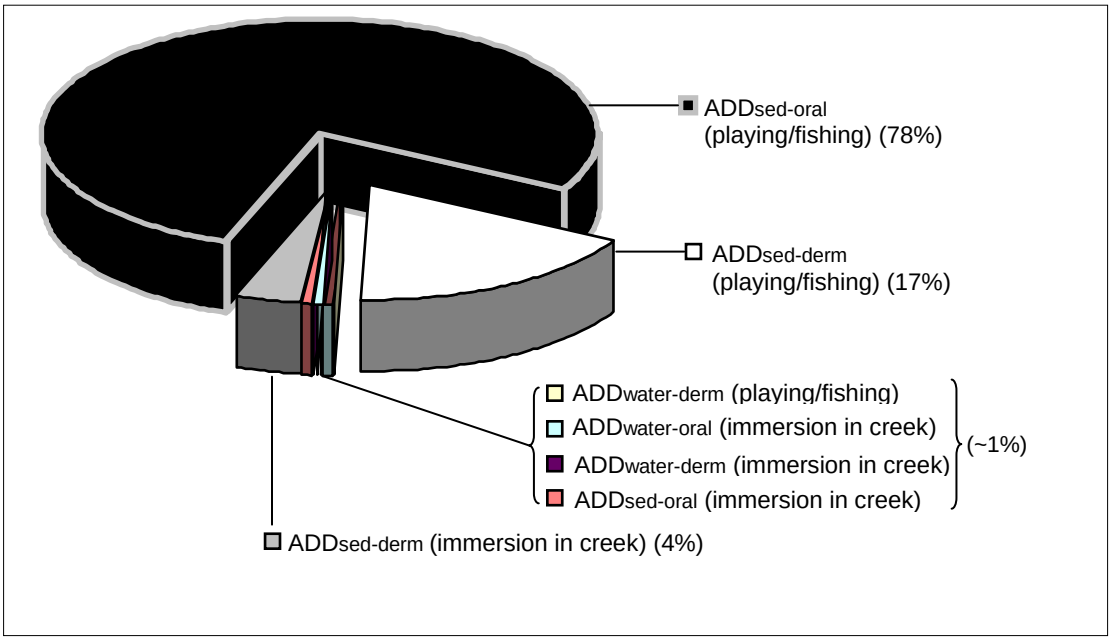
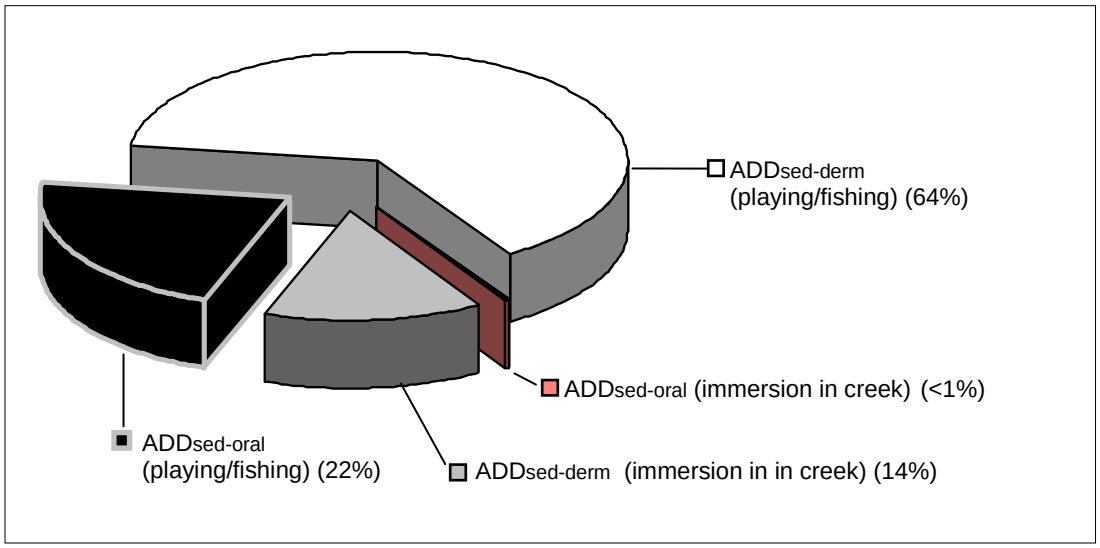


Figure 3-4: Contribution of Exposure Pathways to Recreational Intake of PAHs – Child



It is evident from Figures 3-1 to 3-4 that the majority of exposures to the child are from oral and dermal contact with sediment from playing/fishing. A moderate proportion of exposure to the child occurs from dermal contact with sediment from occasional accidental immersion in the creek. Oral and dermal exposures to contaminants in the water of Talfourd Creek are minimal compared to exposures to contaminants in sediment.

3.3.5 Calculation of Lifetime Average Daily Dose (LADD)

In order to estimate cancer risk, it is necessary to estimate a Lifetime Average Daily Dose (LADD) for each COC. The LADD is a time-weighted average of the ADDs over a lifetime. LADD was estimated for the polycyclic aromatic hydrocarbons (PAHs) because they are the only carcinogenic COC. A sample calculation for LADD is provided using PAHs. [Note: For cancer risk evaluation, PAHs are expressed as equivalents of benzo(a)pyrene (BaP_{eq}) as described in section 2.2.]

$$LADD = \frac{(ADD_{Total\text{-}Child} \times ED_{Child}) + (ADD_{Total\text{-}Teen} \times ED_{Teen}) + (ADD_{Total\text{-}Adult} \times ED_{Adult})}{AT_{lifespan}} \quad \text{(Equation 11)}$$

$ADD_{Total\text{-}Adult}$ = total average daily dose of COC for an adult (for PAHs in BaP_{eq}: 0.000 49 µg/kg/d)
$ADD_{Total\text{-}Child}$ = total average daily dose of COC for a child (for PAHs in BaP_{eq}: 0.005 24 µg/kg/d)
$ADD_{Total\text{-}Teen}$ = total average daily dose of COC for a teen (for PAHs in BaP_{eq}: 0.000 54 µg/kg/d)
$AT_{lifespan}$ = averaging time for total lifespan of receptor (80 y)
ED_{Adult} = exposure duration for adult age category (60 y)
ED_{Child} = exposure duration for child age category (7 y)
ED_{Teen} = exposure duration for teen age category (8 y)
LADD = lifetime average daily dose for receptor (µg/kg/d)

For frequent recreators exposed to polycyclic aromatic hydrocarbons (PAHs) in Talfourd Creek from regularly playing/fishing and occasional accidental immersion in the creek from childhood through to and including adulthood, the LADD is calculated as follows. Using Equations 1 – 10 (and using the concentration of total carcinogenic PAHs in BaP_{eq} as calculated in section 2.2), the ADD_{Total} for carcinogenic PAHs was estimated for each age category: 0.00524, 0.00054, and 0.00049 µg/kg/d for $ADD_{Total\text{-}Child}$, $ADD_{Total\text{-}Teen}$, and $ADD_{Total\text{-}Adult}$, respectively. Using Equation 11, a conservative estimate of LADD for PAHs in BaP_{eq} for the frequent recreator was estimated to be 0.0010 µg/kg/d. For the remaining COCs, oral cancer TRVs are either not available or considered not appropriate because of the lack of evidence of cancer by the oral route. (See the carcinogenicity discussions of each COC in section 4.0.) Thus, no other LADDs were calculated.

4.0 Toxicity Assessment

This section describes the adverse effects and degree of toxicity of each of the COCs.

For each COC, toxicity reference values (TRVs) for cancer and non-cancer effects and relative absorption factors (RAFs) were selected. A non-cancer TRV is a daily dose of a chemical that is considered to be without risk of adverse effects, i.e., an acceptable or tolerable daily intake. A cancer TRV is a value that reflects a relationship between cancer risk and exposure. TRVs are used as (or to set) target exposures that can be compared to estimated exposures in order to evaluate risk.

RAFs are related to a contaminant's toxicokinetics, in particular the proportion absorbed into the body upon exposure to the contaminant in sediment or water. The following sections discuss the selections of TRVs and RAFs for each of the COCs considered in this SLHRA.

4.1 Toxicology and Selection of TRVs

Sections 4.1.1 – 4.1.6 and Appendix A provide discussions of the toxicology and selected TRVs selected for the COCs. A summary of the TRVs selected is provided in Table 4-1.

Table 4-1: Summary of TRVs Selected

COC	Oral Chronic Non-Cancer TRV		Oral Cancer TRV	
	Value (µg/kg/d)	Reference	Value* (per µg/kg/d)	Reference
Cd	0.1	ATSDR, 2012a	n/a	See section 4.1.1
Mn	122	HC CSD, 2010	n/a	See section 4.1.2
Ti	3000	NSF, 2005	n/a	See section 4.1.3
PAHs (BaP)	0.3	US EPA, 2013 (draft)	1×10^{-3}	US EPA, 2013 (draft)
2,6-dichlorobenzyl chloride	n/a	See discussion	n/a	See section 4.1.5
octachlorostyrene	n/a	See discussion	n/a	See section 4.1.6

* n/a = TRV was not available or not appropriate for use

4.1.1 Toxicology and Selection of TRVs for Cadmium (Cd)

4.1.1.1 Non-Cancer Effects of Cd

For the general population, the diet is the most likely source of Cd exposure, and the most sensitive target organs for toxicity are the kidney and bone (Agency for Toxic Substances and Disease Registry (ATSDR), 2012a). Other effects include those on reproduction/development, the liver, haematology (blood), and the immune system (ATSDR, 2012a).

The earliest sign of kidney toxicity is an increased excretion of low molecular weight proteins, increased urinary levels of intracellular enzymes, and increased excretion of calcium and metallothione (ATSDR, 2012a). When the total Cd content in the renal cortex reaches 50 – 300 µg/g (wet weight), the amount of Cd not bound to metallothionein becomes sufficiently high to cause damage to the kidney tubules (ATSDR, 2012a). At higher exposure levels, decreases in glomerular filtration rate, increased risk of renal replacement therapy, and significant increases in the risk of deaths from renal disease have been observed (ATSDR, 2012a).

4.1.1.2 Carcinogenicity of Cd

The U.S. Environmental Protection Agency (US EPA, 1992) classified Cd as a probable human carcinogen based on all exposure routes although no positive studies of ingested Cd were found to be suitable for derivation of a cancer TRV. The health agency of the Netherlands (RIVM, 2001) concluded that evidence of carcinogenicity via the oral route is insufficient. Health Canada (1996) classified Cd as probably carcinogenic to humans. Based on inhalation studies, the International Agency for Research on Cancer (IARC, 2012) stated there is sufficient evidence of carcinogenicity in humans. Both Health Canada (1996) and the California Environmental Protection Agency (Cal EPA, 2009) derived oral cancer TRVs by route-to-route extrapolation from inhalation data because oral cancer data were insufficient. However, epidemiological studies of people chronically exposed to Cd via the diet as a result of environmental contamination have not shown an increased cancer risk (World Health Organization (WHO), 2011).

4.1.1.3 Selection of TRVs for Cd

An oral chronic non-cancer TRV was selected for Cd subsequent to comparing and contrasting the available TRVs from various agencies. Appendix A includes a table with these TRVs and describes the process for selecting 1×10^{-4} mg/kg/d (0.1 µg/kg/d) from ATSDR (2012a) as the most suitable oral chronic non-cancer TRV for use in this SLHRA.

The only oral cancer TRVs identified for Cd were those derived by Health Canada (1996) and Cal EPA (2009) through route-to-route extrapolation from inhalation data. Since the evidence for Cd carcinogenicity by the oral route is insufficient and since the identified oral cancer TRVs were derived by route-to-route extrapolation from inhalation data, these TRV derivations were not considered appropriate for selection. Consequently, no oral cancer TRV was selected for Cd.

4.1.2 Toxicology and Selection of TRVs for Manganese (Mn)

4.1.2.1 Non-Cancer Effects of Mn

Manganese (Mn) is an essential nutrient in humans and other animals (ATSDR, 2012b). For the general population, the primary sources of exposure to Mn are the diet and Mn-containing nutritional supplements (ATSDR, 2012b).

Reports of adverse health effects in humans from ingestion of excess Mn are limited; the limited human data and extensive animal data in the literature clearly identify neurobehavioral changes as the most sensitive effect from intermediate- and chronic-duration oral exposure to excess Mn; at higher doses in rodents, there is also evidence of reproductive, developmental, immunological and other effects (ATSDR, 2012b).

4.1.2.2 Carcinogenicity of Mn

US EPA (1996) classified Mn as “not classifiable as to human carcinogenicity” because existing studies were not adequate to assess the carcinogenicity of Mn. The results of *in vitro* studies show that some chemical forms of Mn may be mutagenic; however, as the results of *in vivo* studies in mammals are inconsistent, no overall conclusion can be made about the possible genotoxic hazard to humans from exposure to Mn (WHO CICAD, 1999).

4.1.2.3 Selection of TRVs for Mn

An oral chronic non-cancer TRV was selected for Mn subsequent to comparing and contrasting the available TRVs from various agencies. Appendix A includes a table with these TRVs and describes the selection process for identifying 0.122 mg/kg/d (122 µg/kg/d) from Health Canada (HC CSD, 2010) as the most suitable oral chronic non-cancer TRV for use in this SLHRA.

No oral cancer TRVs were identified for Mn from the authoritative bodies reviewed.

4.1.3 Toxicology and Selection of TRVs for Titanium (Ti)

4.1.3.1 Non-Cancer Effects of Ti

Ti is generally considered to have low toxicity. Evaluations of titanium dioxide (TiO_2) by several health agencies have concluded that there are no safety concerns associated with the use of TiO_2 as a food additive at levels ranging up to 3% (US EPA, 2005).

There are no experimental data by the oral route in humans (NSF, 2005). In a 3-generation reproductive study in mice and rats (using an unspecified form of Ti), statistically increased neonatal deaths and runts were seen in the second generation (Schroeder and Mitchener, 1971); however, this study was not conducted according to guidelines and details were insufficient for use in risk assessment (NSF, 2005). In female mice fed TiO_2 for 2 years, there was a statistically significant reduction in survival (NCI, 1978), but the study was of questionable significance (NSF, 2005).

TiO_2 fine particles have been considered as a low toxicity material; however, TiO_2 nanoparticles possess different physico-chemical properties which are expected to alter their biological properties (Shi *et al*., 2013). Animal studies imply that accumulation of TiO_2 nanoparticles in organs or tissues may occur with continuous exposure, although responses to this accumulation still require evaluation in further studies (Shi *et al*., 2013).

4.1.3.2 Carcinogenicity of Ti

NSF (2005) stated that there is inadequate information to assess the carcinogenic potential of Ti and TiO_2 to humans by the oral route; the concern associated with positive clastogenicity (disruption or breakage of chromosomes) data is reduced because Ti failed to induce neoplastic lesions following chronic oral exposure in rats and mice.

IARC (2010b) noted that studies do not suggest an association between occupational exposure to TiO_2 and cancer risk. In animal studies, administration of Ti by the oral, subcutaneous, and intraperitoneal routes did not produce a significant increase in frequency of any type of tumour in rats or mice, although inhalation exposure was associated with

occurrence of tumours in some rat studies (IARC, 2010b). Accordingly, IARC (2010b) concluded that there is sufficient evidence for the carcinogenicity of TiO_2 in experimental animals, but inadequate evidence in humans; IARC's overall evaluation is that TiO_2 is possibly carcinogenic to humans (Group 2B).

4.1.3.3 Selection of TRVs for Ti

An oral chronic non-cancer TRV was selected for Ti subsequent to comparing and contrasting the available TRVs from various agencies. Appendix A includes a table with these TRVs and describes the process for selecting 3 mg/kg/d (3×10^3 µg/kg/d) from NSF (2005) as the most suitable oral chronic non-cancer TRV for use in this SLHRA.

No oral cancer TRVs were identified for Ti from the authoritative bodies reviewed.

4.1.4 Toxicology and Selection of TRVs for Polycyclic Aromatic Hydrocarbons (PAHs)

4.1.4.1 Non-Cancer Effects of PAHs

In humans, exposure to benzo[a]pyrene (BaP) occurs in conjunction with other polycyclic aromatic hydrocarbons (PAHs). Human and animal studies have demonstrated associations between exposures to various PAHs and several effects including developmental, reproductive, and immunological (US EPA, 2013 draft).

4.1.4.2 Carcinogenicity of PAHs

IARC (2010a) evaluated the carcinogenicity of several PAHs and PAH mixtures; IARC stated that benzo(a)pyrene is carcinogenic to human. In addition, IARC considers some PAHs to be probably carcinogenic to humans, some to be possibly carcinogenic to humans, and some to be not classifiable as to their carcinogenicity to humans.

Studies in various animal species demonstrate that BaP is carcinogenic at multiple tumour sites by all routes of exposure; there is also strong evidence of carcinogenicity in occupations involving exposures to PAH mixtures containing BaP (US EPA, 2013 draft). Furthermore,

experimental evidence has shown that the carcinogenicity of PAHs is additive (RIVM, 2001). To assess cancer risk from exposure to PAH mixtures, two approaches have been developed. In the surrogate approach, the concentration and toxicity of one PAH (typically BaP) is considered to be representative of the toxicity of the mixture. In the relative potency approach, toxic equivalence factors (TEFs) are assigned to each carcinogenic PAH based on their cancer potency relative to BaP, which is assigned a TEF of 1; the TEF of each PAH is then used to express the concentration of each PAH as an equivalent concentration of BaP, i.e., in BaP equivalents (BaP_{eq}); these are summed to obtain a total concentration of carcinogenic PAHs.

4.1.4.3 Selection of TRVs and TEF Scheme for PAHs

When the ratios among individual PAHs within a mixture tend to vary, the relative potency approach is used to assess the carcinogenicity of the mixture. As described above, this approach requires a TEF scheme, several of which have been developed by various groups; of these, the TEF scheme of Kalberlah *et al*. (1995) used in the development of the MOE (2011b) soil and groundwater (GW) standards was selected for the current SLHRA. The corresponding TEF was applied to the upper estimate of each PAH in order to obtain a concentration of total PAHs expressed in BaP_{eq}. (See also section 2.2). For risk characterization, an oral cancer TRV for BaP was selected. Appendix A includes a table of the oral cancer TRVs available and describes the process for selecting 1.7 per mg/kg/d (1.7×10^{-3} per µg/kg/d) from Cal EPA DW (2010) as the most suitable oral cancer TRV for use in this SLHRA.

Surrogate and relative potency approaches are not available for assessing risk of *non*-cancer effects of PAH mixtures. Therefore, to assess non-cancer risk in this SLHRA, the lowest (most stringent) TRV was selected from among non-cancer TRVs for each PAH. Among the non-cancer TRVs selected for derivation of the MOE (2011b) soil and GW standards, the TRV selected for 1-/2-methylnaphthalene was the lowest at 4×10^{-3} mg/kg/d (US EPA, 2003). However, US EPA's (2013) draft oral chronic non-cancer TRV for BaP is even lower, at 3×10^{-4} mg/kg/d; this non-cancer TRV was selected for comparison with exposure to the sum of all PAHs. Since other PAHs have non-cancer TRVs which are one to three orders of magnitude greater (less stringent) than BaP, summing the PAHs to compare to the oral non-cancer TRV of BaP is a very conservative approach. Appendix A includes a table with the available oral

chronic non-cancer TRVs for BaP and describes the process for selecting 3 x 10^{-4} mg/kg/d (0.3 µg/kg/d) from US EPA (2013 draft) as the most suitable non-cancer TRV for BaP.

4.1.5 Toxicology and Selection of TRVs for 2,6-Dichlorobenzyl Chloride

As discussed in section 2.2, since no health-based or background screening values were available for 2,6-dichlorobenzyl chloride, it was identified as a COC.

4.1.5.1 Non-Cancer Effects of 2,6-Dichlorobenzyl Chloride

2,6-dichlorobenzyl chloride (also called α-2,6-trichlorotoluene) is a chlorinated toluene with three chlorine groups. There are no available studies on the effects of 2,6-dichlorobenzyl chloride in humans and studies in animals are limited. US EPA PPRTV (2005) discusses a 28-day rat study reporting increased liver enzymes at 46 mg/kg/d (Chu *et al*., 1984a); mild histopathological lesions in the liver, kidney, and thyroid were also reported but the doses at which they were produced were not identified. Chu *et al*. (1984a) reported similar findings for 2,3,6-trichlorotoluene. US EPA PPRTV (2005) also discusses a developmental toxicity study reported only as an abstract (Ruddick *et al*., 1982) where pregnant rats were given 2,6-dichlorobenzyl chloride at 0, 100, 200, or 400 mg/kg/d on gestation days 6–15; the authors reported statistically significant reductions in maternal weight gain at 200 and 400 mg/kg/d and liver lesions in rat pups at unspecified doses with the most severe effects at 400 mg/kg/d.

4.1.5.2 Carcinogenicity of 2,6-Dichlorobenzyl Chloride

Neither IARC nor the National Toxicology Program (NTP) evaluated the carcinogenicity of 2,6-dichlorobenzyl chloride (US EPA PPRTV, 2005). Data on the genotoxicity, mutagenicity, or carcinogenic potential of 2,6-dichlorobenzyl chloride are not available. Although mutagenicity and/or carcinogenicity have been shown with other chlorinated toluenes, cancer TRVs for these compounds would not be suitable for assessing carcinogenicity of 2,6-dichlorobenzyl chloride because their dissimilarities with this contaminant have not been quantified.

4.1.5.3 Selection of TRVs for 2,6-Dichlorobenzyl Chloride

No TRVs are available for 2,6-dichlorobenzyl chloride; therefore, exposures to this contaminant are assessed qualitatively.

US EPA PPRTV (2005) found the studies by Ruddick *et al*. (1982) and Chu *et al*. (1984a) to be inadequate for the derivation of TRVs. However, US EPA HEAST (1997) had previously derived an oral *sub*-chronic non-cancer TRV (which is no longer supported by US EPA) of 5 x 10^{-5} mg/kg/d (0.05 µg/kg/d) from the Chu *et al*. (1984a) study, identifying a LOAEL of 0.5 ppm (0.048 mg/kg/d) for liver, kidney, and thyroid lesions and a composite uncertainty factor of 1000 (presumably 10 for extrapolation from LOAEL to NOAEL, 10 for interspecies differences, and 10 for intraspecies differences). Although this TRV has low confidence and is not suitable for evaluating chronic exposure, it is considered for discussion purposes in the risk characterization (section 5.3.1).

4.1.6 Toxicology and Selection of TRVs for Octachlorostyrene

As discussed in section 2.2, octachlorostyrene was identified as a COC because no health-based or background screening values were available for this contaminant; therefore, exposures to this contaminant are assessed qualitatively.

4.1.6.1 Non-Cancer Effects of Octachlorostyrene

Octachlorostyrene causes liver, thyroid, kidney, and hematological effects in laboratory animals (NYS, 1998). In a 28-day feeding study by Chu *et al*. (1982), rats fed 5.0 ppm (0.34–0.43 mg/kg/d) or higher experienced pronounced liver and thyroid effects. In a 90-day feeding study by Chu *et al*. (1984b), rats fed 0.05 ppm (0.0036 mg/kg/d) or higher experienced decreased erythroid cell numbers in bone marrow smears and lesions in the thyroid, liver, and kidney.

In a 12-month feeding study by Chu *et al*. (1986), rats fed in the 0.005 to 0.5 ppm range experienced decreased serum glutamic oxaloacetic transaminase and mild liver changes; in the

thyroid, rats had mild reduction in colloid density and scattered collapse of follicles. According to NYS (1998), the authors report that the effects noted in these three studies (28-day, 90-day, and 12-month) in the 0.005 – 0.5 ppm range are considered to be mild and adaptive in nature and concluded that 0.5 ppm (0.03 mg/kg/d) is an overall NOAEL.

4.1.6.2 Carcinogenicity of Octachlorostyrene

No carcinogenicity evaluations were identified for octachlorostyrene from IARC or other authoritative bodies reviewed. Octachlorostyrene was not mutagenic in the reverse mutation test (Tarkpea *et al*., 1985); no other reports on mutagenicity or genotoxicity were available. In a 12-month rat study, no significant increases in tumour incidence associated with exposure to octachlorostyrene were reported (Chu *et al*., 1986).

4.1.6.3 Selection of TRVs for Octachlorostyrene

No TRVs are available for octachlorostyrene; therefore, exposures to this contaminant will be assessed qualitatively. The toxicity database is not considered sufficient for the derivation of an oral TRV with confidence. However, for the purpose of deriving a drinking water quality value, NYS (1998) derived an acceptable daily intake (ADI) of 0.00003 mg/kg/d (0.03 µg/kg/d) for octachlorostyrene based on a NOAEL of 0.3 mg/kg/d from Chu *et al*. (1986) and a composite uncertainty factor of 1000 (10 for intraspecies variability, 10 for the uncertainty of extrapolation across species, and 10 for the combination of less-than-lifetime duration and uncertainty over the severity of effects below 5 ppm). Although this TRV has low confidence, it may be considered for discussion purposes. In addition, available data on the mutagenicity and carcinogenicity of octachlorostyrene, albeit limited, indicate that it is unlikely to be carcinogenic.

4.2 Selection of Relative Absorption Factors (RAFs) for all COCs

Relative absorption factors (RAFs) are used in a risk assessment to account for the differences in the efficiency of contaminant absorption from the exposure media (e.g., sediment or water) by various exposure routes (e.g., ingestion or dermal) in a human exposure scenario as compared to the toxicity study on which the TRV is based. A RAF is the ratio of the fraction

of a contaminant absorbed in the human exposure scenario to the fraction absorbed in the key toxicity study from which the TRV is derived.

RAFs are contaminant-specific because they depend on unique physical-chemical properties of each contaminant. RAFs are also TRV-specific because they depend on the absolute absorption in the key study of the TRV.

For this SLHRA, each RAF is calculated as follows:

$$RAF = \frac{\text{absolute absorption in exposure scenario (from sediment or water)}}{\text{absolute absorption estimated for key study of TRV}}$$

A RAF of 1 (i.e., 100%) does NOT indicate complete absorption, but rather that absorption in the exposure scenario is considered equivalent to absorption in the key study of the TRV.

As a first step in the determination of RAFs for use in this SLHRA, estimates of absolute absorption were identified for the animal species, the route of exposure, and the medium used in the key study of each TRV selected. Subsequently, absolute absorption was estimated for the frequent recreator at Talfourd Creek for each of the COCs in sediment and water by the oral and dermal routes of exposure. RAFs were then determined by comparing the absorption estimates for the exposure scenario to the absorption estimates for the TRV.

Reviews from several agencies were used to estimate absorption. If absorption estimates were not sufficient or not available from reviews, primary literature was also consulted.

The RAFs selected for use in this SLHRA are shown in Table 4-2. A description of the contaminant-specific information considered for use in estimating these RAFs is presented in Tables B-1 and B-2 of Appendix B. Note that since data on absorption from sediment are lacking, soil absorption data were used to estimate absorption of contaminants from sediment; as advised by US EPA (2004), it was assumed that absorption from soil and sediment is similar.

Table 4-2: Summary of TRVs Selected for Use in the Current SLHRA

COC*	RAF_{OS} (oral relative absorption factor for COC in sediment)	RAF_{OW} (oral relative absorption factor for COC in water)	RAF_{D} (dermal relative absorption factor for COC in sediment and water)
Cd	1	1	0.01
Mn	1	1	0.01
Ti	1	1	0.01
PAHs (BaP)	1	1	0.13

* Exposures to 2,6-dichlorobenzyl chloride and octachlorostyrene will be assessed qualitatively; therefore, RAFs were not determined for these contaminants.

5.0 Risk Characterization

The risk characterization stage of a risk assessment determines whether the estimated COC exposures exceed the identified TRVs. Risks to human health from recreational exposure to contaminants in Talfourd Creek sediment and water were estimated for the frequent recreator scenario for both cancer and non-cancer effects. Section 3.0 describes how maximum and 95th percentile concentrations of COCs in Talfourd Creek sediment and water and were used to calculate conservative estimates of exposure. Section 4.0 describes how TRVs were selected for each of the COCs. In this section, risks are characterized by comparing estimated contaminant exposures to the TRVs.

5.1 Risk Characterization for Non-Cancer Effects

For non-cancer effects, the potential for adverse health effects is assessed by calculating a ratio between (a) the estimated average daily dose (ADD) for each age category of the frequent recreator and (b) the toxicity reference value (TRV). This ratio is termed the hazard quotient (HQ), i.e., $HQ = ADD \div TRV$.

In SLHRAs, the use of conservative assumptions in the exposure estimate generally results in higher HQ estimates than comprehensive risk assessments, but these do not necessarily indicate a greater likelihood of occurrence of adverse health effects than the same HQ values in

a comprehensive risk assessment. A calculated HQ >0.2 or >1 does not necessarily indicate that an elevated risk exists – only that further refinement in the screening level assessment and/or a more comprehensive risk assessment may be warranted.

In the current SLHRA, HQs were estimated for the child, teen, and adult for each COC. Table 5-1 shows the estimated HQs rounded to 1 significant digit and the ADDs and TRVs used to estimate them.

Table 5-1: Estimation of Hazard Quotients (HQs)

Contaminant	Receptor Age Category	Total ADD (µg/kg/d)	Oral Chronic Non-Cancer TRV (µg/kg/d)	HQ
Cd	child	0.0084	0.1	**0.08**
	teen	0.0014		**0.01**
	adult	0.0012		**0.01**
Mn	child	1.9	122	**0.02**
	teen	0.37		**0.003**
	adult	0.31		**0.003**
Ti	child	0.87	3000	**0.0003**
	teen	0.14		**0.000 05**
	adult	0.12		**0.000 04**
Total PAHs	child	0.065	0.3	**0.2**
	teen	0.0065		**0.02**
	adult	0.0059		**0.02**

Based on the results presented in Table 5-1, the child was identified as the “most sensitive receptor” since the HQs for each contaminant were higher for the child than for the teen or adult. For all contaminants, receptor sensitivity decreased in the following order: child > teen > adult.

Under the conservative frequent recreator exposure scenario, estimated HQs were at or below 0.2 for all COCs and all age categories, indicating that no adverse non-cancer health effects are expected to result from recreational use of Talfourd Creek.

Regarding BaP, the selected oral chronic non-cancer TRV was based on a study reporting developmental effects as the critical endpoint. (See details in Appendix A.) Although dosing in the study occurred post-natally, it is possible that individuals are susceptible to developmental effects of BaP during the pre-natal period as well. To address this possibility, the inclusion of a pregnant female receptor would be required. Basically, the main differences in the adult receptor and the pregnant female receptor are exposure frequency (EF) (due to the absence of dose amortizing when assessing risk of developmental effects), body weight (BW), and skin surface area. HQ for the pregnant female can be roughly estimated from the HQ for the adult as follows:

$$HQ_{\text{adult female}} = HQ_{\text{adult}} \times \frac{EF_{\text{adult female}}}{EF_{\text{adult}}} \times \frac{BW_{\text{adult}}}{BW_{\text{adult female}}}$$

$$= 0.020 \times \frac{365\ \text{d/y}}{260\ \text{d/y}} \times \frac{70.7\ \text{kg}}{63.1\text{kg}}$$

$$HQ_{\text{adult female}} = 0.031$$

As a result, the HQ for PAHs for the pregnant female receptor is still well below 0.2. The adult female body weight of 63.1 kg is from Richardson (1997) and was used in the development of the MOE (2011b) soil and groundwater (GW) standards. Note that since skin surface area is greater for the adult receptor than for the adult female receptor, the exclusion of skin surface area from this calculation renders the estimate more conservative.

5.2 Risk Characterization for Cancer Effects

For cancer risk, the potential for adverse health effects is assessed by multiplying the estimated lifetime average daily dose (LADD) for the composite receptor (an estimate of average exposure over a lifetime) by the cancer TRV (a cancer slope factor). The resulting value is expressed as the Incremental Lifetime Cancer Risk (ILCR) which is in the form of a risk level per population, i.e., *ILCR = LADD x cancer TRV*.

In SLHRAs, the use of conservative assumptions in the exposure estimate generally results in higher ILCR estimates than comprehensive risk assessments, but these do not necessarily indicate a greater likelihood of occurrence of adverse health effects than the same ILCR values in a comprehensive risk assessment. A calculated ILCR in the range of 1×10^{-6} to 1×10^{-4} (1 cancer incident per 1,000,000 to 10,000 people exposed; or a cancer incidence probability of 0.0001% to 0.01%) does not necessarily indicate that an elevated risk exists – only that further refinement in the screening level assessment and/or a more comprehensive risk assessment may be warranted.

The ILCR was estimated for the composite receptor for PAHs. Since oral cancer TRVs are not available or not appropriate for use for the remaining COCs, no other ILCRs were calculated. Table 5-2 shows the estimated ILCR (rounded to 1 significant digit) with the LADD and TRV used to estimate it.

Table 5-2: Estimation of Incremental Lifetime Cancer Risk (ILCR)

Contaminant	LADD (µg/kg/d)	Oral Cancer TRV (per µg/kg/d)	ILCR
PAHs (BaP)	0.001	1×10^{-3}	**1×10^{-6}**

Under the conservative exposure scenario developed for recreational exposure, the estimated ILCR for PAHs was 1×10^{-6}, which can also be expressed as a cancer incidence rate of 1 in a million or as a probability of 0.000 1%. An ILCR of 1×10^{-6} is considered to be negligible by MOECC. Therefore, the excess cancer risk resulting from recreational exposure to PAHs in Talfourd Creek is considered to be negligible – even under the conservative frequent recreator scenario used in this SLHRA.

5.3 Risk Characterization for Remaining COCs

2,6-dichlorobenzyl chloride and octachlorostyrene do not have suitable TRVs to permit a typical quantitative risk analysis as conducted for the other COCs. However, based on available information, risks from exposure to these COCs are qualitatively evaluated here.

5.3.1 Risk Characterization for 2,6-Dichlorobenzyl Chloride

US EPA HEAST (1997) derived an oral *sub-chronic* TRV of 0.05 µg/kg/d (which is no longer supported by US EPA) for 2,6-dichlorobenzyl chloride which includes a composite uncertainty factor of 1000 applied to the LOAEL of 48 µg/kg/d. This TRV has low confidence and is not suitable for evaluating chronic exposure; it is included to assist in giving a rough estimate of the level of toxicity of this contaminant.

The maximum concentration of 2,6-dichlorobenzyl chloride found in Talfourd Creek sediment was 0.003 µg/g. At a sediment ingestion rate of 0.12 g/day and BW of 32.9 kg for the child, the contaminant intake rate would be 0.000 01 µg/kg/d, which is over 1000-fold lower than US EPA's oral sub-chronic TRV 0.05 µg/kg/d. Based on available information, no adverse effects from exposure to 2,6-dichlorobenzyl chloride are expected in recreational users of Talfourd Creek.

5.3.2 Risk Characterization for Octachlorostyrene

NYS (1998) derived an oral chronic TRV of 0.03 µg/kg/d for octachlorostyrene which includes a composite uncertainty factor of 1000 applied to the NOAEL of 0.3 mg/kg/d. This TRV has low confidence because the toxicity database is not considered sufficient for the derivation of a reliable oral TRV and may not suitable for evaluating chronic exposure; it is included to assist in giving a rough estimate of the level of toxicity of this contaminant.

The maximum concentration of octachlorostyrene found in Talfourd Creek sediment was 0.2 µg/g. At a sediment ingestion rate of 0.12 g/day and body weight of 32.9 kg for the child, the contaminant intake rate would be 0.0007 µg/kg/d, which is over 40-fold lower than NYS's oral chronic TRV of 0.03 µg/kg/d. Based on available information, no adverse effects are expected from exposure to octachlorostyrene in recreational users of Talfourd Creek.

6.0 Uncertainties and Limitations

SLHRAs typically use conservative assumptions to over-estimate risk. In the current SLHRA, conservative exposure assumptions based on frequent recreational use of Talfourd Creek were used to estimate upper-bound contaminant exposures and the associated risks. Several assumptions were made to fill data gaps and missing information required to estimate the potential exposures and associated risks. The uncertainties associated with these assumptions are briefly discussed below. The results and conclusions reported in this SLHRA should be interpreted in light of these uncertainties.

- The conservative exposure scenario described for the frequent recreator playing and/or fishing at Talfourd Creek was based on information gathered during a site visit. A more comprehensive approach would involve developing a more realistic, albeit conservative, exposure scenario based on more accurate data on recreational uses of the creek. Given the intensity of use assumed for the frequent recreator in the current SLHRA, it is likely that the inclusion of more accurate information on recreational use of the creek would lead to a decrease in estimated exposures and risks. The frequent recreator scenario is sufficiently conservative (e.g., playing/fishing 5 days/week, activity all year round, all exposed body parts covered with sediment and water with every visit to the creek) that it would still be considered protective if actual rates of recreational activity were to increase due to the removal of the posted warning signs along the creek.

- Risks to infants (0 – 6 months old) or toddlers (7 months – 4 years old) were not assessed in this SLHRA since they are not anticipated to wander around the creek unsupervised. Although a toddler has a lower body weight and a higher estimated sediment ingestion rate than other age categories, the increased exposure incurred during a single visit would be outweighed by their very low anticipated overall frequency of exposure (days/week and weeks/year) at the creek. This is especially true for infants because their sediment ingestion rates are even lower than other age categories.

- In the absence of applicable human health-based sediment criteria, sediment concentrations were screened against human health-based soil criteria. Given that exposures to soil and

sediment have some differences, it is possible that the soil criteria are not sufficiently protective when used to screen sediment. In the screening step, some contaminants may not have been screened out if a reliable and complete set of human health-based criteria were available; however, given the conservative approach of the current assessment, any such contaminants would likely have fallen within the risk estimates of the COCs that were carried forward.

- Since sediment contaminants are somewhat mobile as sediment moves downstream over time, contaminant exposures are not likely to be constant during long-term activities at any particular location along Talfourd Creek; a receptor at a fixed location would not be exposed to the upper estimate concentration of a contaminant over the long term. The assumption of long-term exposures to the upper estimate concentration of each contaminant is likely to have overestimated actual exposures.

  Regarding Pb and Hg, maximum sediment concentrations were 282 and 18 µg/g, respectively, but sufficient sediment data were available to allow for the calculation of 95th percentile concentrations. Since contaminant concentrations at any particular location would shift over time with the mobility of the sediment, a human receptor would not be exposed to the maximum concentrations of Pb and Hg over the long term.

- The contaminant concentrations used in the SLHRA were obtained from studies published since 2004. The concentrations of some contaminants may have been lower or higher in data reports before 2004, but those data would not be representative of current conditions of the creek especially because of the mobility of water and sediment of the creek.

- If additional sediment or water sampling were to be undertaken in Talfourd Creek, it is possible that higher concentrations could be found because there have been changes over time in the primary contaminant sources. However, based on the sediment and water data available, it is difficult to delineate any overall trends in the contaminant concentrations. Nonetheless, in a comprehensive risk assessment with an appropriately planned sampling strategy and a more realistic exposure scenario, the calculated risks for all contaminants would likely be lower than the risks calculated in the current SLHRA.

- The data in the source documents were collected generally for the purpose of monitoring biota in Talfourd Creek, rather than for estimating human health risks. The suite of measured contaminants differs between reports and differed between sediment and water within reports. Also, contaminant concentrations from some of the source documents were below the method detection limits (MDLs); in some cases, MDLs were not reported. Any contaminants where all data were consistently below the MDL were not included in this SLHRA which creates some limitations. Firstly, the true concentrations of these contaminants remain unknown. Secondly, it is unknown if the concentrations are below health-based concentrations. However, the wide range of data on concentrations of inorganic and organic contaminants used in this SLHRA is sufficient to support the SLHRA conclusions. Additional data would not be likely to change the SLHRA conclusions.

- Additional pathways of exposure from recreation were not assessed in the current SLHRA but are theoretically possible, such as inhalation of contaminants in sediment or water. However, as discussed in section 2.5, inhalation of sediment and water particles resuspended in air is expected to be negligible compared to oral and dermal exposures from recreation at Talfourd Creek.

- Site-specific bioavailability and speciation data were not available for the contaminants evaluated; as is commonly done in risk assessments, data obtained from the scientific literature were used in lieu of site-specific data. These assumptions generally contributed additional conservatism to the SLHRA. For example, the oral bioavailabilities of heavy metals in sediment and water were generally assumed to be equivalent to the highly bioavailable forms normally used in toxicity studies.

- The derivation of TRVs typically incorporates a considerable degree of uncertainty. In the derivation of cancer TRVs, the linear extrapolation of data in the low-dose region of the dose-response curve is assumed to be sufficiently conservative to account for uncertainties related to the TRV. In the derivation of non-cancer TRVs, the application of uncertainty factors conservatively addresses the various areas of uncertainty in the TRV.

- The selection of only one non-cancer TRV to assess all PAHs introduces some uncertainty in the risk assessment, but since the most stringent TRV was selected from all the PAHs, the uncertainty errs on the side of conservatism. Another layer of conservatism is added by summing all the PAH exposures together even though it is not likely that they would all have the effect on which the selected oral non-cancer TRV is based.

- There is some uncertainty in the SLHRA from the lack of toxicological information available for 2,6-dichlorobenzyl chloride and octachlorostyrene. However, based on available information and the upper estimate concentrations reported in Talfourd Creek, adverse health effects from these contaminants are not likely.

- It is important to note that the scope of this SLHRA was limited to exposures only from *recreational use* of Talfourd Creek. Potential exposures to COCs from other pathways such as fish consumption were not evaluated. Therefore, this SLHRA is not directly applicable to other uses of Talfourd Creek (such as the consumption of fish or wildlife) or to any other potential exposures from living in the Lambton County area.

7.0 Conclusions and Recommendations

Based on a conservative exposure scenario for frequent recreation that likely overestimates actual recreational exposures along Talfourd Creek, the predicted exposures to COCs from incidental ingestion and dermal contact with contaminants in sediment and water were relatively low. Estimated HQs were at or below 0.2 for all COCs; the estimated ILCR for PAHs, the only carcinogenic COC, was at the 1×10^{-6} risk level (i.e., 0.0001% probability of cancer incidence).

The results obtained in this SLHRA indicate that adverse health effects are not likely to occur from contact with sediment and water from recreational use of Talfourd Creek. Within the limits of the current SLHRA, health risks associated with recreational exposure to contaminants in the sediment and water of Talfourd Creek under the exposure routes and pathways described are negligible. The application of more realistic exposure assumptions (i.e., in a comprehensive HHRA) would result in even lower estimates of risk.

The most sensitive receptor is the most impacted receptor with the highest calculated risk values. The child is identified as the most sensitive receptor because exposures and calculated risk incurred by the child for all COCs are greater than those incurred by the teen and adult. The most significant exposure pathways for the child are oral and dermal contact with sediment incurred from playing/fishing.

Based on the current SLHRA using conservative exposure assumptions, frequent recreational use of Talfourd Creek and associated exposures to Talfourd Creek sediment and water are unlikely to cause any adverse health effects to any age group.

It should be clearly stated that this SLHRA was designed to assess potential health effects only from recreational use of Talfourd Creek. Consequently, the results are not directly applicable to any other risks associated with living in the Lambton County area (including consumption of fish or other wildlife).

8.0 References

Atkinson Davies. 2004. Report on Sediment Sampling Program - Aamjiwnaang First Nation, Sarnia, Ontario. Southern First Nations Secretariat, Bothwell, Ontario. Ref.: 5-1680-1. Atkinson, Davies Inc. Consulting Engineers. London, Ontario. August 18, 2004.

ATSDR. 2007. Toxicological Profile for Lead. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Atlanta, GA, USA. August 2007.

ATSDR. 2012a. Toxicological Profile for Cadmium. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Atlanta, GA, USA. September 2012.

ATSDR. 2012b. Toxicological Profile for Manganese. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. September 2012.

Cal EPA. 2009. Technical Support Document for Cancer Potency Factors: Methodologies for Derivation, Listing of Available Values, and Adjustments to Allow for Early Life Stage Exposures. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Air Toxics Hot Spots Program. Sacramento, California. May 2009.

Cal EPA DW. 2010. Public Health Goals for Chemicals in Drinking Water – Benzo(a)pyrene. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. September 2010.

Cal EPA. 2011. All Public Health Goals. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. All PHGs developed as of July 2011. http://oehha.ca.gov/water/phg/allphgs.html (Last accessed Oct. 23, 2013).

CCME. 1999. Canadian Sediment Quality Guidelines for the Protection of Aquatic Life - Mercury. Canadian Council of Ministers of the Environment. Excerpt from Publication No. 1229; ISBN 1-896997-34-1.

CCME. 2012. Canadian Soil Quality Guidelines for Lead: Human Health. Scientific Supporting Document. Prepared by National Guidelines and Standards Office, Environment Canada and Contaminated Sites Division, Health Canada. January 2012 draft.

Chu I, Secours VE, Villeneuve DC, Valli VE. 1982. Acute and Subacute Toxicity of Octachlorostyrene in the Rat. J Toxicol Environ Health. 10:285-296.

Chu I, Shen SY, Villeneuve DC, Secours VE, Valli VE. 1984a. Toxicity of Trichlorotoluene Isomers: A 28-Day Feeding Study in the Rat. *J Environ Sci Health* B19:183-191.

Chu I, Villeneuve DC, Secours VE, Yagminas A, Reed B, Valli VE. 1984b. Octachlorostyrene: A 90-day Toxicity Study in the Rat. *Fundam Appl Toxicol* 4:547-557.

Chu I, Villeneuve DC, Secours VE, Valli VE, Leeson S, Shen SY. 1986. Long-term Toxicity of Octachlorostyrene in the Rat. *Fundam Appl Toxicol* 6:69-77.

Dufour AP, Evans O, Behymer TD, Cantu R. 2006. Water Ingestion during Swimming Activities in a Pool: A Pilot Study. *J Water Health* 4:425-430.

Environment Canada. 2004. Results of Follow-Up Sediment Quality Investigations in Lake Erie Watersheds, 2003. Ecosystem Health Division, Environmental Conservation Branch, Ontario Region, Environment Canada. February 2004.

Health Canada. 1996. Priority Substances List Assessment Report: Cadmium and its Compounds. Second Priority Substances List (PSL2) Assessments. Canadian Environmental Protection Act (CEPA). Environment Canada and Health Canada. Ottawa, ON.

HC CSD. 2010. Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors, Version 2.0. Prepared by: Contaminated Sites Division, Safe Environments Directorate. Health Canada. September 2010.

Health Canada. 2012. Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA), Version 2.0. Federal Contaminated Site Risk Assessment in Canada. Contaminated Sites Division, Health Canada. ISBN 978-1-100-17671-0. September 2010. Revised 2012.

Holmes KK Jr, Shirai JH, Richter KY, Kissel JC. 1999. Field Measurement of Dermal Soil Loadings in Occupational and Recreational Activities. *Environ Res* 80:148-157, section A.

IARC. 2010a. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans – Volume 92: Some Non-heterocyclic Polycyclic Aromatic Hydrocarbons and Some Related Exposures. International Agency for Research on Cancer, World Health Organization. Geneva, Switzerland.

IARC. 2010b. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans – Volume 93: Carbon Black, Titanium Dioxide, and Talc. International Agency for Research on Cancer, World Health Organization. Geneva, Switzerland.

IARC. 2012. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans – Volume 100F: Chemical Agents and Related Compounds. International Agency for Research on Cancer, World Health Organization. Geneva, Switzerland.

Kalberlah F, Frijus-Plessen N, Hassauer M. 1995. Toxicological Criteria for the Risk Assessment of Polyaromatic Hydrocarbons (PAH) in Existing Chemicals. Part 1: The Use of Equivalency Factors. Altlasten-Spektrum. 5 pp. 231-237. As cited in: WHO, 1998.

Kissel JC, Richter KY, Fenske RA. 1996. Field Measurement of Dermal Soil Loading Attributable to Various Activities: Implications for Exposure Assessment. *Risk Analy* 16:115-125.

Kissel JC, Shirai JH, Richter KY, Fenske RA. 1998. Investigation of Dermal Contact with Soil in Controlled Trials. *J Soil Contam* 7:737-752.

MOE. 1992. Talfourd Creek Contaminant Survey. R.W. Bilyea, Great Lakes Section, Water Resources Branch, Ontario Ministry of the Environment. In support of the St. Clair River Remedial Action Plan. March 1992.

MOE. 2007. Biomonitoring Assessment of Talfourd Creek, Sarnia, Ontario, 2005. R. Fletcher, S. Baker, and J. Martherus, Environmental Monitoring and Reporting Branch, Ontario Ministry of the Environment. October 2007.

MOE. 2009. Talfourd Creek, 2007-2008 Monitoring Study. Memorandum from Wolfgang Scheider, Environmental Monitoring and Reporting Branch, Ontario Ministry of the Environment (MOE) to Annette Verhagen, South Western Region, MOE. September 3, 2009.

MOE. 2011a. Assessment of Sediment and Biota from Talfourd Creek, 2010. Emily Awad, Biomonitoring Section, Environmental Monitoring and Reporting Branch, Ontario Ministry of the Environment. November 2011.

MOE. 2011b. Rationale for the Development of Soil and Ground Water Standards for Use at Contaminated Sites in Ontario. Standards Development Branch, Ontario Ministry of the Environment. PIBS 7386e01. April 15, 2011.

NCI. 1978. Bioassay of Titanium Dioxide for Possible Carcinogenicity. National Cancer Institute. Bethesda, MD, USA. NTIS PB288780. Technical Report 97.

NSF. 2005. Titanium and Titanium Dioxide. CAS# 7440-32-6 and 13463-67-7. Oral Risk Assessment Document. National Sanitation Foundation International. Ann Arbor, MI, USA. 46 pp. June 2005.

NYS. 1998. New York State Human Health Fact Sheet – Ambient Water Quality Value for Protection of Sources of Potable Water. Substance: Octachlorostyrene. New York State. 12 pp. March 12, 1998.

Richardson GM. 1997. Compendium of Canadian Human Exposure Factors for Risk Assessment. O'Connor Associates Environmental Inc. Ottawa, Ontario, Canada.

RIVM. 2001. Re-Evaluation of Human Toxicological Maximum Permissible Risk Levels. Rijksinstituut voor Volksgezondheid en Milieu (National Institute of Public Health and the Environment). Baars AJ, Theelen RMC, Janssen PJCM, Hesse JM, Apeldoorn ME van, Meijerink MCM, Verdam L, Zeilmaker MJ. Bilthoven, The Netherlands. RIVM Report No. 711701025. March 2001.

Ruddick JA, Villeneuve DC, Secours VE, Valli VE. 1982. A Transplacental and Teratological Evaluation of Three Trichlorotoluene Congeners in the Rat. *Teratology* 25:72A-73A.

Schroeder HA, Mitchener M. 1971. Toxic effects of trace elements on the reproduction of mice and rats. Arch Environ Health 23: 102-106.

Shi H, Magaye R, Castranova V, Zhao J. 2013. Titanium Dioxide Nanoparticles: A Review of Current Toxicological Data. Part Fibre Toxicol 10:15. 33 pp.

Shoaf MB, Shirai JH, Kedan G, Schaum J, Kissel JC. 2005. Child Dermal Sediment Loads Following Play in a Tide Flat. *J Expo Analy Environ Epidemiol* 15:407-412.

Tarkpea M, Hagen I, Carlberg GE, Kolsaker P, Storflor H. 1985. Mutagenicity, Acute Toxicity, and Bioaccumulation Potential of Six Chlorinated Styrenes. *Bull Environ Contam Toxicol* 35:525-530.

US EPA. 1992. Cadmium (CASRN 7440-43-9). Integrated Risk Information System. U.S. Environmental Protection Agency. Washington, DC, USA. www.epa.gov/iris/subst/0141.htm (Last accessed Oct. 31, 2013).

US EPA. 1995. Mercuric Chloride (CASRN 7487-94-7). Integrated Risk Information System. U.S. Environmental Protection Agency. Washington, DC, USA. www.epa.gov/iris/subst/0692.htm (Last accessed Sept. 30, 2013).

US EPA. 1996. Manganese (CASRN 7439-96-5). Integrated Risk Information System. U.S. Environmental Protection Agency. Washington, DC, USA. http://www.epa.gov/iris/subst/0373.htm (Last accessed March 17, 2014).

US EPA. 1997. Exposure Factors Handbook. U.S. Environmental Protection Agency. EPA/600/P-95/002Fa. August 1997.

US EPA. 2001. Methylmercury (CASRN 22967-92-6). Integrated Risk Information System. U.S. Environmental Protection Agency. Washington, DC, USA. www.epa.gov/iris/subst/0073.htm (Last accessed Sept. 30, 2013).

US EPA IRIS. 2003. 2-Methylnaphthalene. (CASRN 91-57-6). Integrated Risk Information System. U.S. Environmental Protection Agency. Washington, DC, USA. www.epa.gov/iris/subst/1006.htm (Last accessed Nov. 19, 2013).

US EPA. 2004. Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Final. U.S. Environmental Protection Agency. EPA/540/R/99/005. July 2004.

US EPA. 2005. Memorandum: Reassessment of the Exemptions from the Requirement of a Tolerance for Titanium Dioxide. June 28, 2005. From Kerry Leifer, Inerts Team Leader, Minor Use, Inerts and Emergency Response Branch; to Dan Rosenblatt, Chief, Minor Use, Inerts, and Emergency Response Branch. Office of Prevention, Pesticides and Toxic Substances, U.S. Environmental Protection Agency.

US EPA. 2008. Child-Specific Exposure Factors Handbook. U.S. Environmental Protection Agency. WPA/600/R/06/096F. September 2008.

US EPA. 2009. Toxicological Review of Thallium and Compounds (CAS No. 7440-28-0). In Support of Summary Information in the Integrated Risk Information System (IRIS). U.S. Environmental Protection Agency. Washington, DC, USA. September 2009.

US EPA. 2011. Exposure Factors Handbook: 2011 Edition. National Center for Environmental Assessment, U.S. Environmental Protection Agency. Washington, DC, USA. EPA/600/R-09/052F. September 2011.

US EPA. 2013. Regional Screening Level (RSL) Residential Soil Table (TR = 1E-6, HQ=0.1). U.S. Environmental Protection Agency, Region III. May 2013.

US EPA. 2013 (draft). Toxicological Review of Benzo[a]pyrene (CASRN 50-32-8). In Support of Summary Information on the Integrated Risk Information System (IRIS). U.S. Environmental Protection Agency. Washington, DC. Public Comment Draft. August 2013.

US EPA HEAST. 1997. Health Effects Assessment Summary Tables, FY 1997 Update. Solid Waste and Emergency Response, U.S. Environmental Protection Agency. EPA-540-R-97 036. July 1997.

US EPA PPRTV. 2005. Provisional Peer Reviewed Toxicity Values for α-2,6-Trichlorotoluene. (CASRN 2014-83-7). Superfund Health Risk Technical Support Center, Office of Research and Development, U.S. Environmental Protection Agency. Cincinnati, OH, USA. September 23, 2005.

WHO. 1998. Selected Non-Heterocyclic Polycyclic Aromatic Hydrocarbon. Environmental Health Criteria 202. International Program on Chemical Safety, United Nations Environmental Program, World Health Organization. Geneva, Switzerland.

WHO CICAD. 1999. Manganese and its Compounds – Concise International Chemical Assessment Document 12. World Health Organization. Geneva, Switzerland.

WHO. 2011. Cadmium in Drinking-Water. Background Document for Development of WHO Guidelines for Drinking-Water Quality. World Health Organization. Geneva, Switzerland. WHO/SDE/WSH/03.04/80/Rev/1.

Wilson & Meridian. 2011. Development of Sediment Ingestion Rates for Use in Human Health Based Sediment Quality Guidelines and Contaminated Sites Human Health Risk Assessment. Wilson Scientific Consulting Inc. and Meridian Environmental Inc. Submitted to Contaminated Sites Division, Environmental Health Programs Directorate, Health Canada. Call-up Reference No. 4500243691. March 15, 2011.

Wilson & Meridian. 2012. Analysis of Sensitivity of Sediment Ingestion Rates for Human Health Risk Assessment of Contaminated Sites. Wilson Scientific Consulting Inc. and Meridian Environmental Inc. Submitted to Contaminated Sites Division, Environmental Health Programs Directorate, Health Canada. Final Report. December 2012.

Wilson R, Richardson GM. 2013. Lead (Pb) is Now a Non-Threshold Substance: How Does this Affect Soil Quality Guidelines? Human Ecol Risk Assess 19:1152-1171.

Appendix A: Selection of Toxicity Reference Values (TRVs)

MOECC TOXICITY REFERENCE VALUE (TRV) SELECTION RATIONALE DOCUMENT

Benzo[a]pyrene (BaP)

CAS # 50-32-8

Oral Chronic Non-Cancer

Agency	TRV (mg/kg/d)	Point of Departure		Uncertainty Factors							Notes
		Dose (mg/kg/d)	Basis	A	H	L	S	D	X	Total	
Cal EPA DW 2010	1.7E-03	5	LOAEL	10	10	10	10			3000	• Knuckles *et al.*, 2001 • Supporting studies: MacKenzie & Angevine, 1981; Kristensen *et al*., 1995; De Jong *et al.*, 1999; Kroese *et al.*, 2001; Saunders *et al*., 2006: subchronic & acute LOAELs in range of 10–25 mg/kg/d for various effects • M/F F-344 rats fed 0, 5, 50, 100 mg_{BaP}/kg_{BW}/d in diet, for up to 90 d • Critical effect: dose-dependent kidney abnormalities in males at all doses • Composite UF of 10,000 was limited to max of 3000
US EPA IRIS 2013 (draft)	3E-04	0.09	BMDL	10	10			3		3000	• Chen *et al.*, 2012 • M/F Sprague-Dawley rats, 10/sex/dose, early post-natal exposure • Doses: 0, 0.02, 0.2, 2 mg_{BaP}/kg/d by gavage PNDs 5–11 • Critical effect: Dose-dependent neurobehavioural changes (altered anxiety-like behaviour) during adulthood after exposure as pups at all doses (Developmental effects) • From various studies, US EPA derived TRVs of 3 x 10^{-4} mg/kg/d for developmental effects, 4 x 10^{-4} mg/kg/d for reproductive effects, and 2 x 10^{-3} mg/kg/d for immunological effects. ▪ Developmental toxicity was chosen as basis for proposed overall RfD • US EPA states that the overall RfD is derived to be *protective of all types of effects* for a given exposure duration and is *intended to protect the population as a whole* including potentially susceptible subgroups.

Selection: 3E-04 mg/kg/d: US EPA IRIS (2013 draft)

Rationale: The TRV derived by US EPA IRIS (2013 draft) was based on a more sensitive endpoint; also, BMD modelling is considered to be a more robust method of selecting a POD. Furthermore, the TRV derived by Cal EPA incorporated a composite UF of 10,000 – limited to a maximum composite UF of 3000 – which is indicative of a larger magnitude of uncertainty than the derivation by US EPA. For these reasons, the TRV derivation by US EPA IRIS (2013 draft) was preferred and selected.

Although this TRV is based on developmental effects, it is applicable to all age categories because it was co-derived with other candidate TRVs in the same range of values (from 6 x 10^{-5} mg/kg/d to 5 x 10^{-3} mg/kg/d) based on a variety of effects.

Oral Slope Factor

Agency	Oral Slope Factor $(mg/kg/d)^{-1}$	Extrapolation Method	Notes

US EPA IRIS 1994	7.3	various (See Notes)	• 4 CSFs calculated: 3 from mouse data (Neal and Rigdon, 1967) & 1 from rat data (Brune *et al.*, 1981)
			
			Neal and Rigdon, 1967 [Additional control data from Rabstein *et al*., 1973 (mice; oral, diet)]
			• CFW-Swiss Mice (M/F), 17-180 d old, 9–73 mice/group (& 289 controls); up to 197 d
			• Doses: 0, 1, 10, 20, 30, 40, 45, 50, 100, 250 ppm_{BaP} in diet
			• Critical effect: dose-dependent incidence of forestomach tumours
			• 3 modelling procedures applied to data to derive 3 CSFs:
			  ○ 2-stage response model to derive CSF of 5.9 $(mg/kg/d)^{-1}$
			  ○ linear extrapolation from 10% response point to background response to derive 9.0 $(mg/kg/d)^{-1}$
			  ○ Weibull-type model to reflect less-than-lifetime exposure to derive a CSF of 4.5 $(mg/kg/d)^{-1}$
			
			Brune *et al.*, 1981:
			• Sprague Dawley rats, 32/sex/grp, treated until moribund or dead (approx. 2 y)
			• Fed 0.15 mg/kg in diet or in 1.5% caffeine solution either every 9^{th} d or 5 d/wk ≈ 0, 6, 39 mg_{BaP}/kg/yr
			• Critical effect: tumours of forestomach, esophagus, & larynx
			• Linearized multistage model applied to data to derive CSF of 11.7 $(mg/kg/d)^{-1}$
			
			• CSF of 7.3 $(mg/kg/d)^{-1}$ is geometric mean of 4 CSFs

Agency	Oral Slope Factor $(mg/kg/d)^{-1}$	Extrapolation Method	Notes
RIVM 2001	0.2	Linear non-threshold	• Kroese *et al.*, 1999 • M/F Wistar rats, 52/sex/dose, 5 d/wk, for 2 y • Doses: 0, 3, 10, 30 mg_{BaP}/kg_{BW}/d by oral gavage • Critical effect: dose-dependent tumours most prominent in liver & forestomach, but also in skin, auditory canal, oesophagus, mammary gland, small intestine, & kidney
Cal EPA ATH 2009	12	Linearized multistage procedure	• Neal and Rigdon, 1967 (same study as US EPA IRIS, 1994) • Critical effect: gastric tumours (papillomas & squamous cell carcinomas) in M & F mice • CSF rounded from 11.5 per mg/kg/d
HC CSD 2010	2.3	Linear extrapolation	• Neal and Rigdon, 1967 (same study as US EPA IRIS, 1994) • Critical effect: gastric tumours (mostly squamous cell papillomas, with a few carcinomas) • Included surface area correction
Cal EPA DW 2010	2.9	Multi-stage Weibull-in-time model	• 5 CSFs calculated: 1 from mouse data (Culp *et al*., 1998) & 4 from rat data (Kroese *et al*., 2001) Culp *et al.*, 1998 • Female B6C3F1 mice, 48/dose, for 2 y • Only females because of their low spontaneous liver tumour incidence & lower ability to conjugate BaP reactive metabolites than males • Doses: 0, 5, 25, 100 ppm_{BaP} in diet ≈ 0.0, 0.65, 3.5, 15.2 mg/kg/d • Critical effect: combined tumours of forestomach, tongue, & esophagus • CSF of 1.7 $(mg/kg/d)^{-1}$ associated with LED_{10} Kroese *et al*., 2001 (same study as RIVM, 2001) • Critical effect: increases in liver tumours & combined tumours of oral cavity & forestomach • CSFs of 0.21, 0.10, 0.36, & 0.33 $(mg/kg/d)^{-1}$ associated with LED_{10} calculated for liver tumors in males & females, & for forestomach/oral cavity tumours in males & females, respectively • Most health-protective CSF of 1.7 $(mg/kg/d)^{-1}$ selected from mouse data (Culp *et al*., 1998); CSF multiplied by Age Sensitivity Factor of 1.7 to account for higher sensitivity of children to carcinogens

Agency	Oral Slope Factor $(mg/kg/d)^{-1}$	Extrapolation Method	Notes
US EPA IRIS 2013 (draft)	1	Linear extrapolation from $BMDL_{10}$ using multistage Weibull model	• 3 CSFs calculated: 1 from mouse data (Beland & Culp, 1998) & 2 from rat data (Kroese *et al*., 2001) Beland & Culp, 1998 (same study as Culp *et al*., 1998 used by Cal EPA DW, 2010, though reported separately) • Doses: 0, 5, 25, 100 ppm_{BaP} in diet ≈ 0, 0.7, 3.3, 16.5 mg/kg/d • Critical effect: dose-dependent increase in alimentary tract tumours (forestomach, esophagus, tongue, larynx) in female mice at ≥0.7 mg/kg/d; human equivalent CSF = $0.1/BMDL_{10HED}$ = 1 $(mg/kg/d)^{-1}$ Kroese *et al*., 2001 (same study as RIVM, 2001) • Critical effect: combined tumours of forestomach, oral cavity, liver, jejunum/duodenum, kidney, skin, & mammary glands; human equivalent CSF = 0.5 $(mg/kg/d)^{-1}$ • Critical effect: combined tumours of forestomach, oral cavity, liver, & jejunum/duodenum; human equivalent CSF = 0.3 $(mg/kg/d)^{-1}$ • No single CSF is supported as most relevant for extrapolating to humans, thus selected most health protective CSF derived: 1 $(mg/kg/d)^{-1}$ from mouse data (Culp *et al*., 1998)

Selection: 1 $(mg/kg/d)^{-1}$: US EPA IRIS (2013 draft)

Rationale: The CSFs identified were derived from among only four studies. The two most recent of these studies (Beland & Culp, 1998 / Culp *et al*., 1998 and Kroese *et al*., 2001) were conducted in accordance with Good Laboratory Practice (as established by the Organization for Economic Co-operation and Development: OECD); these studies included histological examinations for tumours in many different tissues, contained three exposure levels and controls, contained adequate numbers of animals per dose group (~50/sex/group), treated animals for up to 2 years, and included detailed reporting of methods and results, including individual animal data (US EPA IRIS, 2013). [Note that Beland & Culp (1998) and Culp *et al*. (1998) are two reports of the same study.]

The other two of the four studies (Neal & Rigdon, 1967 and Brune *et al.*, 1981) have been criticized for qualities that make them less optimal for use in CSF derivation. Although the Neal and Rigdon (1967) study is a controlled, multiple-dose, repeating-dosing study, most animals were treated <1 year, which is less optimal for extrapolating to lifetime exposure (US EPA IRIS, 2013). Furthermore, this study was deficient because combined groups of males and females were employed, the number of animals in each group was variable, treatment began at difference ages among the animals, and treatment occurred for different time intervals (Cal EPA DW, 2010). The study by Brune *et al*. (1981) has been criticized for its non-standard treatment protocol in comparison to the GLP studies conducted by Beland & Culp (1998) / Culp *et al*. (1998) and by Kroese *et al*. (2001). Accordingly, the CSFs derived from the studies by Neal and Rigdon, 1967 and Brune *et al.*, 1981 (US EPA IRIS, 1994; Cal EPA ATH, 2009; HC CSD, 2010) were not considered further.

The CSF derived by RIVM (2001) was based on a suitable study (Kroese *et al*., 1999), but is less robust than the derivations by Cal EPA DW (2010) and US EPA IRIS (2013), each of which was based on several datasets from two studies (Beland & Culp *et al*., 1998 / Culp *et al*., 1998 and Kroese *et al*., 2001). In addition, derivation details are not very extensive in the RIVM (2001) documentation. For these reasons, RIVM (2001) was not considered further.

The CSFs derived by Cal EPA DW (2010) and US EPA IRIS (2013) are based on the same two studies, Beland & Culp (1998) / Culp *et al*. (1998) and Kroese *et al*. (2001). The values of the CSFs derived and the derivation methods used by these agencies are comparable, but Cal EPA applied an age sensitivity factor to their CSF. To ensure a CSF is appropriate for use in a wide variety of HHRAs and standards development, it is best to select and apply any applicable age sensitivity factors, if necessary, in the risk calculations along with the CSF. Therefore, the CSF of 1 $(mg/kg/d)^{-1}$ derived by US EPA IRIS (2013 draft) was selected.

References for Benzo[a]pyrene TRVs:

Beland F, Culp S. 1998. Chronic Bioassay of Two Composite Samples from Selected Manufactured Gas Plant Waste Sites [Unpublished report]. Division of Biochemical Toxicology, National Center for Toxicological Research, Jefferson, Arkansas, USA. Technical Report 6722.02.

Brune H, Deutsch-Wenzel RP, Habs M, Ivankovic S, Schmahl D. 1981. Investigation of the Tumorigenic Response to Benzo(a)pyrene in Aqueous Caffeine Solution Applied Orally to Sprague-Dawley Rats. *J Cancer Res Clin Oncol* 102:153-157.

Cal EPA ATH. 2009. Technical Support Document for Cancer Potency Factors: Methodologies for Derivation, Listing of Available Values, and Adjustments to Allow for Early Life Stage Exposures. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Air Toxics Hot Spots Program. Sacramento, California. May 2009.

Cal EPA DW. 2010. Public Health Goals for Chemicals in Drinking Water – Benzo(a)pyrene. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. September 2010.

Chen C, Tang Y, Jiang X, Qi Y, Cheng S, Qiu C, Peng B, Tu B. 2012. Early Postnatal Benzo(a)pyrene Exposure in Sprague-Dawley Rats Causes Persistent Neurobehavioral Impairments that Emerge Postnatally and Continue into Adolescence and Adulthood. *Toxicol Sci* 125:248-261.

Culp SJ, Gaylor DW, Sheldon WG, Goldstein LS, Beland FA. 1998. A Comparison of the Tumors Induced by Coal Tar and Benzo[a]pyrene in a 2-year Bioassay. *Carcinogen* 19:117-124.

HC CSD. 2010. Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors, Version 2.0. Prepared by: Contaminated Sites Division, Safe Environments Directorate. Health Canada. September 2010.

Knuckles ME, Inyang F, Ramesh A. 2001. Acute and Subchronic Oral Toxicities of Benzo[a]pyrene in F-344 Rats. *Toxicol Sci* 61:382-388.

Kristensen P, Eilertsen E, Einarsdottir E, Haugen A, Skaug V, Ovrebo S. 1995. Fertility in Mice after Prenatal Exposure to Benzo[a]pyrene and Inorganic Lead. *Environ Health Perspect* 103:588-590.

Kroese ED, Muller JJA, Mohn GR, Dortant PM, Wester PW. 1999. Tumorigenic Effects in Wistar Rats Orally Administered Benzo(a)pyrene for Two Years (Gavage Studies). Implication for Human Cancer Risk Associated with Oral Exposure to Polycyclic Aromatic Hydrocarbons. RIVM: Rijksinstituut voor Volksgezondheid en Milieu (National Institute of Public Health and the Environment). Bilthoven, Netherlands. Report No 6658603 010. July 1999 draft report.

Kroese ED, Muller JJA, Mohn GR, Dortant PM, Wester PW. 2001. Tumorigenic Effects in Wistar Rats Orally Administered Benzo(a)pyrene for Two Years (Gavage Studies). Implication for Human Cancer Risk Associated with Oral Exposure to Polycyclic Aromatic Hydrocarbons. RIVM: Rijksinstituut voor Volksgezondheid en Milieu (National Institute of Public Health and the Environment). Bilthoven, Netherlands. Report No 6658603 010.

MacKenzie KM, Angevine DM. 1981. Infertility in Mice Exposed *in utero* to Benzo(a)pyrene. *Biol Reprod* 24:183-191.

Neal J, Rigdon RH. 1967. Gastric Tumors in Mice Fed Benzo(a)pyrene: a Quantitative Study. *Tex Rep Biol Med* 25:553-557.

Rabstein LS, Peters RL, Spahn GJ. 1973. Spontaneous Tumors and Pathologic Lesions in SWR-J Mice. *J Natl.Cancer Inst* 50:751-758.

RIVM. 2001. Re-Evaluation of Human Toxicological Maximum Permissible Risk Levels. Rijksinstituut voor Volksgezondheid en Milieu (National Institute of Public Health and the Environment). Baars AJ, Theelen RMC, Janssen PJCM, Hesse JM, Apeldoorn ME van, Meijerink MCM, Verdam L, Zeilmaker MJ. Bilthoven, The Netherlands. RIVM Report No. 711701025. March 2001.

Saunders CR, Das SK, Ramesh A, Shockley DC, Mukherjee S. 2006. Benzo(a)pyrene-induced Acute Neurotoxicity in the F-344 Rat: Role of Oxidative Stress. *J Appl Toxicol* 26:427-438.

US EPA IRIS. 1994. Benzo[a]pyrene (CASRN 50-32-8). Integrated Risk Information System. U.S. Environmental Protection Agency. Washington, DC, USA. www.epa.gov/iris/subst/0136.htm (Last accessed Oct. 1, 2013).

US EPA IRIS. 2013 draft. Toxicological Review of Benzo[a]pyrene (CASRN 50-32-8). In Support of Summary Information on the Integrated Risk Information System (IRIS). U.S. Environmental Protection Agency. Washington, DC. Public Comment Draft. August 2013.

MOECC TOXICITY REFERENCE VALUE (TRV) SELECTION RATIONALE DOCUMENT

Cadmium (Cd)

CAS # 7440-43-9

Oral Chronic Non-Cancer

Agency	TRV (mg/kg/d)	Point of Departure		Uncertainty Factors							Notes
		Dose (mg/kg/d)	Basis	A	H	L	S	D	X	Total	
US EPA IRIS 1994	5E-04 (water)	5.0E-03 (water)	NOAEL		10					10	• US EPA, 1985; based on Friberg *et al*., 1974 • Critical effects: significant proteinuria (water); human studies involving chronic exposures (food) • 200 μg_{Cd}/g human renal cortex (wet weight) is highest concentration not associated with proteinuria • TK model determined daily intake of 0.352 mg_{Cd}/d (0.005 mg/kg/d in 70kg adult) for 50 y results in renal cortex concentration of 200 μg_{Cd}/g • TK model assumes 0.01% of Cd body burden eliminated per day & 2.5% absorption from food & 5% absorption from water
	1E-03 (food)	1.0E-02 (food)									
RIVM 2001	5E-04	1E-03	LOAEL						2	2	• Jarup *et al*.,1998; Nogawa *et al*., 1989 & other human datasets not specified • Critical effect: renal tubular dysfunction (initially increased urinary excretion of low molecular weight proteins) • Adverse effects detected in ~4% of the general population (equivalent urinary excretion 2.5 $\mu g_{Cd}/g_{creatinine}$) • 50 μg_{Cd}/g in renal cortex likely reached after 40–50 y intake of 1 µg/kg/d • UF_X 2 to ensure individual is below population-based LOAEL • TWI of 3.5 μg_{Cd}/kg/wk estimated intake rate without appreciable risk

Agency	TRV (mg/kg/d)	Point of Departure		Uncertainty Factors							Notes
		Dose (mg/kg/d)	Basis	A	H	L	S	D	X	Total	
Cal EPA ChRD 2005	1.1E-05	1E-03	LOAEL		10	3			3	90	• Buchet *et al*., 1990 • Epidemiological study (1699 people); 20-80 y old • Critical effect: Renal tubular dysfunction (associated with Cd body burden) • Critical effect occurs at urinary excretion rate ≥ 2 μg_{Cd}/d • Assuming 5% oral absorption & 0.005% daily excretion rate of body burden, estimated urinary excretion rate corresponds to 50 μg_{Cd}/g in renal cortex • In non-smokers, 50 μg_{Cd}/g reached after 50 y intake of 1 μg_{Cd}/kg/d (LOAEL) • UF_X 3 for age specific differences in biokinetics
WHO JECFA 2005	1E-03	See notes									• WHO, 1972; WHO JECFA, 1988 • PTWI of 400-500 μg_{Cd}/person (~ 7 $\mu g/kg_{BW}$/wk) estimated intake rate without appreciable risk • Based on PTWI, estimated levels in renal cortex not to exceed 50 μg_{Cd}/g (assuming 5%absorption rate & 0.005% daily excretion of body burden); total intake not to exceed 1 µg/kg/d continuously for 50 yrs (WHO, 1992)
Cal EPA DW 2006	6.3E-06	3.17E-04	NOAEL		5				10	50	• Occupational & environmental exposure studies (Jarup *et al*., 1998; Jarup *et al.,* 1995; Buchet *et al*., 1980; Chia *et al.,* 1992; Cai *et al.,* 1998; Nogawa *et al.,* 1979; Elinder *et al.,* 1985; Bernard *et al*., 1990; Ellis *et al.,* 1979; Nakadaira and Nishi, 2003; Fels *et al.,* 1994; Roels *et al.,* 1993; Noonan *et al.,* 2002) • Critical Effect: renal toxicity (increased excretion of urinary proteins) • Urinary Cd of 1 $\mu g/g_{creatinine}$ not to result in critical effect • TK model used to determine that 19ug_{Cd}/d corresponds to urinary Cd level of 1$ug_{Cd}/g_{creatinine}$ • POD calculation: chronic oral intake ((19μg_{Cd}/d) ÷ 60 kg (female BW) • UF_H 5 for limited TK information on Cd, particularly in women • UF_X 10 for potential carcinogenicity

Agency	TRV (mg/kg/d)	Point of Departure		Uncertainty Factors							Notes
		Dose (mg/kg/d)	Basis	A	H	L	S	D	X	Total	
HC CSD 2010	1.0E-03	See notes								1	• HC DW, 1986 (WHO, 1972, Friberg *et al.,* 1971) • Occupational exposure studies, primarily inhalation (Cd oxide dusts &/or fumes) • Critical effect: renal tubular dysfunction (proximal tubule epithelial cell damage), manifested as low molecular weight proteinuria • 200 $\mu g_{Cd}/g$ human renal cortex (wet weight) is highest concentration not associated with proteinuria • PTWI of 400-500 μg_{Cd}/person (~ 7 µg/kg/wk) leads to 0.1% population reaching 200 $\mu g_{Cd}/g$ in renal cortex after 50 y • NOAEL of 2.5 $\mu g_{Cd}/g_{creatinine}$ in urine associated with chronic oral intake of 0.5-2.0 µg/kg/d • PTWI of 7 µg/kg/wk (1 µg/kg/d) retained • TRV is provisional
ATSDR 2012	1E-04	3.3E-04	$UCDL_{10}$		3					3	• Buchet *et al*., 1990; Jarup *et al*., 2000; Suwazono *et al*., 2006 • Meta-analysis of 7 environmental exposure studies (including Jin *et al*., 2004; Kobayashi *et al*., 2006; Shimizu *et al*., 2006; Wu *et al*., 2001) • Critical effect: urinary Cd level resulting in 10% increase in β2-microglobulin proteinuria (UCD_{10}) • $UCDL_{10}$ of 0.5 $\mu g_{Cd}/g_{creatinine}$ • 0.5 $\mu g/g_{creatinine}$ corresponds to dietary Cd intakes of 0.33 µg/kg/d (females) & 0.70 µg/kg/d (males) by 55 y of age • POD is dietary intake in females associated with $UCDL_{10}$ • UF_H 3 for increased sensitivity of diabetics (Akesson *et al*., 2005; Buchet *et al*., 1990)

Selection: 1E-04 mg/kg/d: ATSDR (2012)

Rationale: Documentation on the TRV derivations from RIVM (2001), WHO JECFA (2005), and HC CSD (2010) was limited, but were interpreted to be based on post hoc evaluations of the WHO (1972) provisional tolerable weekly intake rate (PWTI). The WHO (1972) PWTI and US EPA IRIS (1994) TRVs used the Cd concentration in the renal cortex as an indicator of Cd renal toxicity to estimate a NOAEL for proteinuria. However, a urinary Cd excretion concentration is a more sensitive indicator of the onset of renal toxicity then a renal cortex Cd concentration. Therefore,

there is greater confidence in the derivations of Cal EPA DW (2006) and ATSDR (2012) as they used the initial biomarkers of urinary Cd excretion to estimate the daily Cd intake. Consequently, the derivations by US EPA IRIS (1994), RIVM (2001), WHO JECFA (2005) and HC CSD (2010) were not considered further.

The ATSDR (2012) TRV derivation was selected as its derivation is based on more recent studies and applied the preferred dose-response benchmark meta-analysis to estimate the internal dose corresponding to a 10% excess risk of low molecular weight proteinuria, as compared to the NOAEL approach used by Cal EPA DW (2006).

References for Cadmium TRVs:

Åkesson A, Lundh T, Vahter M, Bjellerup P, Lidfeldt J, Nerbrand C, Samsioe G, Stromberg U, Skerfbing S. 2005. Tubular and Glomerular Kidney Effects in Swedish Women with Low Environmental Cadmium Exposure. *Environ Health Perspect* 113:1627-1631.

ATSDR. 2012. Toxicological Profile for Cadmium. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. September 2012.

Bernard AM, Roels H, Cardenas A, Lauwerys R. 1990. Assessment of Urinary Protein 1 and Transferrin as Early Markers of Cadmium Nephrotoxicity. *Br J Ind Med* 47:559-565.

Buchet JP, Lauwerys R, Roels H, Bernard A, Bruaux P, Claeys F, Ducoffre G, De Plaen P, Staessen J, Amery A, Lijnene P, Thijs L, Rondia D, Sartor F, Saint Remy A, Nick L. 1990. Renal Effects of Cadmium Body Burden of the General Population. *Lancet* 336:699-702.

Buchet JP, Roels H, Bernard A, Lauwerys R. 1980. Assessment of Renal Function of Workers Exposed to Inorganic Lead, Calcium or Mercury Vapor. *J Occup Med* 22:741-750.

Cai S, Yue L, Jin T, Nordberg G. 1998. Renal Dysfunction from Cadmium Contamination of Irrigation Water: Dose-Response Analysis in a Chinese Population. *Bull World Health Organ* 76:153-159.

Cal EPA chRD. 2005. Development of Health Criteria for School Site Risk Assessment Pursuant to Health and Safety Code Section 901(g): Child-Specific Reference Doses (chRDs) for School Site Risk Assessment - Cadmium, Chlordane, Heptachlor, Heptachlor Epoxide, Methoxychlor, and Nickel. Integrated Risk Assessment Branch, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Final. December 2005.

Cal EPA DW. 2006. Public Health Goal for Chemicals in Drinking Water: Cadmium. California Public Health Goal (PHG). Pesticide and Environmental Toxicology Branch, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Sacramento, CA, USA.

Chia KS, Tan AL, Chia SE, Ong CN, Jeyaratnam J. 1992. Renal Tubular Function of Cadmium Exposed Workers. *Ann Acad Med Singapore* 21:756-759.

Elinder CG, Edling C, Lindberg E, Kagedal B, Vesterberg O. 1985. Beta 2-Microglobulinuria among Workers Previously Exposed to Cadmium: Follow-up and Dose-Response Analyses. *Am J Ind Med* 8:553-564.

Ellis KJ, Vartsky D, Zanzi I, Cohn SH, Yasumura S. 1979. Cadmium: *In Vivo* Measurement in Smokers and Nonsmokers. *Science* 205:323-325.

Fels LM, Bundschuh I, Gwinner W, Jung K, Pergande M, Graubaum HJ. 1994. Early Urinary Markers of Target Nephron Segments as Studied in Cadmium Toxicity. *Kidney Int Suppl* 47:S81-S88.

Friberg L, Piscator M, Nordberg G. 1971. Cadmium in the Environment. The Chemical Rubber Co (CRC) Press, Cleveland, OH, USA.

Friberg L, Piscator M, Nordberg GF, Kjellstrom T. 1974. Cadmium in the Environment, 2nd ed. CRC Press Inc. Boca Raton, Florida, USA.

HC CSD. 2010. Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors, Version 2.0. Prepared by: Contaminated Sites Division, Safe Environments Directorate. Health Canada. September 2010.

HC DW. 1986. Guidelines for Canadian Drinking Water Quality: Supporting Documentation: Cadmium. Drinking Water Quality in Canada. Ministry of Public Works and Government Services. Ottawa, ON, Canada.

Järup L, Persson B, Elinder CG. 1995. Decreased Glomerular Filtration Rate in Solderers Exposed to Cadmium. *Occup Environ Med* 52:818-22.

Järup L, Berglund M, Elinder CG, Nordberg G, Vahter M. 1998. Health Effects of Cadmium Exposure - a Review of the Literature and a Risk Estimate. *Scand J Work Environ.Health* 24(Suppl.1):1-51.

Järup L, Hellstrom L, Alfven T. 2000. Low Level Exposure to Cadmium and Early Kidney Damage: The OSCAR study. *Occup Environ Med* 57:668-672.

Jin T, Kong Q, Ye T. 2004. Renal Dysfunction of Cadmium-Exposed Workers Residing in a Cadmium-Polluted Environment. *Biometals* 17:513-518.

Kobayashi E, Suwazono Y, Uetani M. 2006. Estimation of Benchmark Dose as the Threshold Levels of Urinary Cadmium, Based on Excretion of Total Protein, β2-microglobulin and N-acetyl-β-D-glucosaminidase in Cadmium Nonpolluted Regions in Japan. *Environ Res* 101:401-406.

Nakadaira H, Nishi S. 2003. Effects of Low-dose Cadmium Exposure on Biological Examinations. *Sci Total Environ* 308:49-62.

Nogawa K, Kobayashi E, Honda R. 1979. A Study of the Relationship between Cadmium Concentrations in Urine and Renal Effects of Cadmium. *Environ Health Perspect* 28:161-168.

Nogawa K, Honda R, Kido T, Tsuritani I, Yamada Y, Ishizaki M, Yamaya H. 1989. A Dose-Response Analysis of Cadmium in the General Environment with Special Reference to Total Cadmium Intake Limit. Environ Res 48:7-16.

Noonan CW, Sarasua SM, Campagna D, Kathman SJ, Lybarger JA, Mueller PW. 2002. Effects of Exposure to Low Levels of Environmental Cadmium on Renal Biomarkers. *Environ Health Perspect* 110:151-155.

RIVM. 2001. Re-Evaluation of Human Toxicological Maximum Permissible Risk Levels. Rijksinstituut voor Volksgezondheid en Milieu (National Institute of Public Health and the Environment). Baars AJ, Theelen RMC, Janssen PJCM, Hesse JM, Apeldoorn ME van, Meijerink MCM, Verdam L, Zeilmaker MJ. Bilthoven, The Netherlands. RIVM Report No. 711701025. March 2001.

Roels H, Bernard AM, Cardenas A, Buchet JP, Lauwerys RR, Hotter G, Ramis I, Mutti A, Franchini I, Bundschuh I. 1993. Markers of Early Renal Changes Induced by Industrial Pollutants. III. Application to Workers Exposed to Cadmium. *Br J Ind Med* 50:37-48.

Shimizu A, Kobayashi E, Suwazono Y. 2006. Estimation of Benchmark Doses for Urinary Cadmium based on β2 -microglobulin Excretion in Cadmium-polluted Regions of the Kakehashi River Basin, Japan. *Int J Environ Health Res* 16:329-337.

Suwazono Y, Sand S, Vahter M, Filipsson AF, Skerfving S, Lidfeldt J, Akesson A. 2006. Benchmark Dose for Cadmium-induced Renal Effects in Humans. *Environ Health Perspect* 114:1072-1076.

US EPA. 1985. Drinking Water Criteria Document on Cadmium. Office of Drinking Water, U.S. Environmental Protection Agency. Washington, DC, USA.

US EPA IRIS. 1994. Cadmium (CASRN 7440-43-9). Integrated Risk Information System. U.S. Environmental Protection Agency. Washington, DC, USA. www.epa.gov/iris/subst/0141.htm (Last accessed Jan. 25, 2013)

WHO. 1972. Long Term Programme in Environmental Pollution Control in Europe. The Hazards to Health of Persistent Substances in Water. Annexes to a Report on a Working Group. Technical Documents on Arsenic, Cadmium, Lead, Manganese and Mercury. Copenhagen: World Health Organization.

WHO JECFA.1988. Evaluation Performed by the Joint FAO/WHO Expert Committee on Food Additives. Geneva, Switzerland. TRS776 JECFA 33/28.

WHO JECFA. 2005. Cadmium. Summary of Evaluation Preformed by the Joint FAO/WHO Expert Committee on Food Additives. Food and Agriculture Organization of the United Nations. Geneva, Switzerland.

Wu X, Jin T, Wang Z. 2001. Urinary Calcium as a Biomarker of Renal Dysfunction in a General Population Exposed to Cadmium. *J Occup Environ Med* 43:898-904.

MOECC TOXICITY REFERENCE VALUE (TRV) SELECTION RATIONALE DOCUMENT

Manganese (Mn)

CAS # 7439-96-5

Oral Chronic Non-Cancer

Agency	TRV (mg/kg/d)	Point of Departure		Uncertainty Factors							Notes
		Dose (mg/kg/d)	Basis	A	H	L	S	D	X	Total	
US EPA IRIS 1996a	1.4E-1 (for diet)	1.4E-1 (10 mg/d)	NOAEL							1	• Freeland-Graves et al., 1987; NRC, 1989; WHO, 1973 • NOAEL of 10 mg/d (0.14 mg/kg/d for 70-kg adult) for chronic human consumption in diet, based on composite of data from several studies • Critical endpoint: CNS effects, although no LOAEL was identified by US EPA • UF of 1 because NOAEL was identified from large populations consuming normal diets over an extended period of time with no adverse effects • UF_X of 3: Modifying factor of 3 applied when assessing risk from Mn in DW or soil for various reasons: ▪ TK data: no significant difference in Mn absorption from food vs. water, but Mn uptake from water is greater in fasting individuals; ▪ Epidemiologic study of Mn in DW (Kondakis et al., 1989) raises concerns of possible neurotoxicity at doses near essential range; ▪ Neonates absorb more Mn from GIT & excrete less absorbed Mn ▪ Mn in infant formula much higher than human or cow milk; reconstituting with DW represents additional source of Mn for a potentially sensitive population
US EPA IRIS 1996b; US EPA HESD 2003; US EPA DW 2004	4.7E-02 (for DW & soil)								3	3	

Agency	TRV (mg/kg/d)	Point of Departure		Uncertainty Factors							Notes
		Dose (mg/kg/d)	Basis	A	H	L	S	D	X	Total	
HC CSD 2010	1.22E-01 to 1.56E-01	11 mg/d	NOAEL							1	• IOM, 2001; Greger, 1999; Davis & Greger, 1992 Davis & Greger, 1992: • 47 adult women; oral Mn supplements; 15 mg/d or placebo; 124 d • Critical effect: significant increases in lymphocyte Mn-dependent superoxide dismutase activity after 90 d; 15 mg/d considered LOAEL Greger, 1999 • Determined that individuals consuming Western diets consume up to 10mg_{Mn}/d, considered a NOAEL
ATSDR 2012	1.6E-01										• IOM: UF of 1 for of lack of evidence of human toxicity from doses <11 mg/d • IOM: TRVs for life stages before adult were adjusted for relative BW • HC CSD: Adjusted IOM TRVs for life stage & BW; TRV range is for various age categories • ATSDR: applied BW of 70 kg to IOM TRV of 11 mg/d to obtain interim guidance value
WHO DW 2011	6E-02	1.8E-01 (11 mg/d)	NOAEL							3	• Greger, 1999 (same key study as HC CSD, 2010); IOM, 2002 • Critical effect: neurological impairment, although no LOAEL was identified by WHO • Applied BW of 60 kg to NOAEL • UF_X of 3 for possible increased bioavailability of Mn from water

Selection: 1.22E-01 mg/kg/d: HC CSD (2010)

Rationale: All the TRVs identified were based on studies identifying NOAELs of 10 or 11 mg/d (converted to mg/kg/d by applying a BW). Derivations by HC CSD and ATSDR identified both a NOAEL and a LOAEL and were thus preferred to the remaining TRV derivations that identified solely a NOAEL. Of these two agencies, HC CSD (2010) adjusted the TRVs in consideration of age-specific body weights. Thus the low end of the TRV range – 0.122 mg/kg/d – from HC CSD (2010) was selected.

References for Manganese TRVs:

ATSDR. 2012. Toxicological Profile for Manganese. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. September 2012.

Davis CD, Greger JL. 1992. Longitudinal Changes of Manganese-Dependent Superoxide Dismutase and Other Indices of Manganese and Iron Status in Women. *Am J Clin Nutr* 55:747-752.

Freeland-Graves JH, Bales CW, Behmardi F. 1987. Manganese Requirements of Humans. In: Nutritional Bioavailability of Manganese, Kies C, Ed. American Chemical Society, Washington, DC. p. 90-104.

Greger JL. 1999. Nutrition Versus Toxicology of Manganese in Humans: Evaluation of Potential Biomarkers. *Neurotoxicol* 20: 205-212.

HC CSD. 2010. Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors, Version 2.0. Prepared by: Contaminated Sites Division, Safe Environments Directorate. Health Canada. September 2010.

IOM. 2001/2002. Manganese. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc (2000). A Report of the Panel on Micronutrients, subcommittees on upper reference levels of nutrients and of interpretation and uses of dietary reference intakes, and the standing committee on the scientific evaluation of dietary reference intakes. Washington, DC: Food and Nutrition Board. Institute of Medicine. National Academy Press, 394-419.

Kondakis XG, Makris N, Leotsinidis M, Prinou M, Papapetropoulos T. 1989. Possible Health Effects of High Manganese Concentration in Drinking Water. *Arch Environ Health* 44:175-178.

NRC. 1989. Recommended Dietary Allowances, 10th ed. Food and Nutrition Board, National Research Council, National Academy Press. p. 230-235. Washington, DC, USA.

WHO. 1973. Trace Elements in Human Nutrition: Manganese. Report of a WHO expert committee, World Health Organization. Technical Report Series, No. 532, pp. 34–36. Geneva, Switzerland.

WHO DW. 2011. Manganese in Drinking-Water. Background Document for Development of WHO Guidelines for Drinking-Water Quality. World Health Organization. Geneva, Switzerland. WHO/SDE/WSH/03.04/104/Rev/1.

US EPA DW. 2004. Drinking Water Health Advisory for Manganese. Office of Water (4304T), U.S. Environmental Protection Agency. EPA-822-R-04-003. January 2004.

US EPA HESD. 2003. Health Effects Support Document for Manganese. Office of Water (4304T), U.S. Environmental Protection Agency. EPA-822-R-03-003. February 2003.

US EPA IRIS. 1996. Manganese (CASRN 7439-96-5). Integrated Risk Information System. U.S. Environmental Protection Agency. Washington, DC, USA. www.epa.gov/iris/subst/0373.htm (Last accessed Sept. 30, 2013).

MOECC TOXICITY REFERENCE VALUE (TRV) SELECTION RATIONALE DOCUMENT

Titanium (Ti)

CAS # (various)

Oral Chronic Non-Cancer

Agency	TRV (mg/kg/d)	Point of Departure		Uncertainty Factors							Notes
		Dose (mg/kg/d)	Basis	A	H	L	S	D	X	Total	
RIVM 2004	12	1250	NOAEL							100	• NCI, 1978 • Fischer rats & B6C3F1 mice, 50/sex, fed Ti dioxide for 2 y • Doses: 25 000, 50 000 mg_{TiO2}/kg_{diet} ≈ 1250, 2500 mg/kg/d • Critical effect: None observed (no increased mortality, carcinogenicity, or adverse effects in rats) • Composite UF of 100 presumably UF_A of 10 & UF_H of 10
NSF 2005	3	2,680	NOAEL	10	10			10		1000	• NCI, 1978 (same key study as RIVM, 2004) • NSF calculated rat doses ≈ 1340, 2680 mg/kg/d • Critical effect: None observed (no significant adverse responses in rats at tested doses) • UF_D of 10 for lack of developmental toxicity studies in 2 species & a 2-generation reproduction study, & for possibility that reproductive toxicity may occur with oral exposure to Ti [Schroeder & Mitchener (1971) reported statistically increased neonatal deaths & runts in 2nd generation in a 3-generation reproduction study with unspecified form of Ti]

Selection: 3 mg/kg/d: NSF (2005)

Rationale: The two TRV derivations identified for Ti were based on the same key study, but differed in their selections of a NOAEL point of departure and UFs. Since neither treatment dose in the key study resulted in adverse health effects, the higher of the two doses is a more appropriate NOAEL for use as a POD, as selected by NSF (2005). In addition, the UF_D of 10 applied by NSF (2005) is justified given the potential for reproductive toxicity and the overall paucity in Ti toxicity studies, as described by NSF (2005). For these reasons, the TRV derived by NSF (2005) was preferred and selected.

References for Titanium TRVs:

NCI. 1978. Bioassay of Titanium Dioxide for Possible Carcinogenicity. National Cancer Institute. Bethesda, MD, USA. NTIS PB288780. Technical Report 97.

NSF. 2005. Titanium and Titanium Dioxide. CAS# 7440-32-6 and 13463-67-7. Oral Risk Assessment Document. National Sanitation Foundation International. Ann Arbor, MI, USA. 46 pp. June 2005.

Schroeder HA, Mitchener M. 1971. Toxic effects of trace elements on the reproduction of mice and rats. Arch Environ Health 23: 102-106.

RIVM. 2004. Oriënterende Evaluatie Gezondheidsrisico Metalen in Tatoeages [Preliminary Health Risk Assessment of Metals in Tattoos]. Janssen PJCM, Baars AJ. Rijksinstituut voor Volksgezondheid en Milieu (National Institute for Public Health and the Environment). The Netherlands. RIVM Report No. 320105001/2004. (In Dutch).

Appendix B: Selection of Relative Absorption Factors (RAFs)

Table B-1: Oral Relative Absorption Factors for Soil/Sediment (RAF_{OS}) & Water (RAF_{OW})

COC	Estimate of absolute oral absorption for species used in key study of TRV						RAF_{OS} : Estimate of oral soil RAF (oral absorption of COC from soil for humans relative to absorption in key study of TRV)		RAF_{OW} : Estimate of oral water RAF (oral absorption of COC from soil for humans relative to absorption in TRV key study)	
	TRV agency & year	type of TRV	details of key study		estimate of absolute oral absorption in key study		notes	RAF	notes	RAF
			species	dosing regimen	notes	%				
Cd	ATSDR 2012a	non-cancer	human	environ-mental exposure	General population is exposed to Cd mainly through food & tobacco smoke (Cal EPA DW, 2006). Human studies indicate absolute absorption of Cd consumed with food ranges 1 - 11% (Cal EPA DW, 2006).	1 – 11%	Studies suggest moderate reductions in bioavailability from soil compared to soluble forms (NEPI 2000a). NEPI (2000a) cites studies reporting relative bioavailability of Cd in soil of 43% & 62–85%. Schroder *et al*. (2003) report 10–116% for relative bioavailability of Cd from soils in juvenile swine. Since relative bioavailability is generally in high percentages, 100% RAF is selected.	1	US EPA (2004, Exhibit 4-1) estimates absolute oral Cd absorption from water to be 5% in humans. Since this is roughly within the range assumed for the key study, 100 % RAF is selected.	1
Mn	HC CSD 2010	non-cancer	human	supple-ments & diet	Oral absorption of Mn from food ranges from 0.6% to 16% - but generally around 5%, although some studies have shown infant absorption up to 41% & absorption with Fe deficiency to be up to 45.5% (US HESD, 2003).	~5% (range 0.6 – 16%)	No studies were identified which measured oral absorption of Mn from soil. Thus, it is assumed that absorption from soil is the same as that from food: RAF of 100% is selected.	1	Oral absorption of Mn from water is generally greater than from food (ATSDR, 2012b). One study found the range of oral absorption of Mn to be 7.74-10.24% in water & 1.71-5.20% in food (US HESD, 2003). Although absorption from water may be slightly higher than absorption from food, the range of absorption for Mn in water is still within the range for Mn in food. Thus, 100% RAF is selected.	1
Ti	NSF 2005	non-cancer	rat	Ti dioxide in diet	Oral Ti absorption is low but is shown to occur from diet in mice (Sugibayashi *et al*., 2008). Oral absorption occurs for Ti in water or in 0.5% hydroxypropylmethylcellulose including mineral form of Ti (DEPA, 2013). Oral absorption of TiO_2 is low, only ~6% for bulk form (DEPA, 2013).	<10%	Since absorption of Ti in soil relative to diet is not clear, assume 100% RAF.	1	Oral absorption is low but is shown to occur from water, 0.5% hydroxypropylmethylcellulose (DEPA, 2013), & diet (Sugibayashi *et al*., 2008). Since absorption of Ti in water relative to diet is not clear, assume 100% RAF.	1

COC	Estimate of absolute oral absorption for species used in key study of TRV						RAF_{OS} : Estimate of oral soil RAF (oral absorption of COC from soil for humans relative to absorption in key study of TRV)		RAF_{OW} : Estimate of oral water RAF (oral absorption of COC from soil for humans relative to absorption in TRV key study)	
	TRV agency & year	type of TRV	details of key study		estimate of absolute oral absorption in key study		notes	RAF	notes	RAF
			species	dosing regimen	notes	%				
PAHs	US EPA IRIS 2013	non-cancer	rat	BaP in peanut oil, by oral gavage	US EPA (2004, Exhibit 4-1) estimates absolute oral absorption of 58% for PAHs in rats dosed via starch solution.	58%	NEPI (2000b) reports absorption in the range of 25-90% for PAHs (data from Stroo *et al*., 1999). 7-76% (Magee *et al*., 1996). Few studies exist on absorption of PAHs from soil matrix; those located report a similar range of absorption efficiencies as diet, thus 100% RAF is assumed.	1	US EPA (2004, Exhibit 4-1) estimates absolute oral absorption of 58% for PAHs in rats dosed via starch solution.	1
	US EPA IRIS 2013	cancer	mice	in diet	US EPA (2004, Exhibit 4-1) estimates absolute oral absorption of 58–89% for PAHs, based on rats dosed via diet or starch solution.	58 – 89%				

Table B-2: Dermal Relative Absorption Factors (RAF_D)

COC	Estimate of absolute oral absorption for species used in key study of TRV						RAF_{OS} : Estimate of dermal RAF (dermal absorption of COC from soil or water for humans relative to absorption in key study of TRV)	
	TRV agency & year	type of TRV	details of key study		estimate of absolute oral absorption in key study		notes	RAF
			species	dosing regimen	notes	%		
Cd	ATSDR 2012a	non-cancer	human	environ-mental exposure	General population is exposed to Cd mainly through food & tobacco smoke (Cal EPA DW, 2006). Human studies indicate absolute absorption of Cd consumed with food ranges 1 - 11% (Cal EPA DW, 2006).	1 – 11%	US EPA (2004, Exhibit 3-4) suggests 0.1% absolute dermal absorption for Cd. Thus, the RAF with respect to oral absorption from diet is ~1%.	0.01
Mn	HC CSD 2010	non-cancer	human	supple-ments & diet	Oral absorption of Mn from food ranges from 0.6% to 16% - but generally around 5%, although some studies have shown infant absorption up to 41% & absorption with Fe deficiency to be up to 45.5% (US HESD, 2003).	~5% (range 0.6 – 16%)	There are few reports of dermal exposure to Mn; uptake across intact skin is expected to be limited (WHO CICAD, 1999). Workers dermally exposed to organic Mn after a spill experienced headache & paresthesia (WHO CICAD, 1999) which are symptoms of manganism. Dermal absorption appears to be evident only when doses are very high, thus dermal absorption rates are likely to be much lower than oral absorption rates. Therefore, a dermal RAF of 1% is selected. [Furthermore, NHDES (2006) also expects skin absorption to be <1%.]	0.01

COC	Estimate of absolute oral absorption for species used in key study of TRV						RAF_{OS} : Estimate of dermal RAF (dermal absorption of COC from soil or water for humans relative to absorption in key study of TRV)	
	TRV agency & year	type of TRV	details of key study		estimate of absolute oral absorption in key study		notes	RAF
			species	dosing regimen	notes	%		
Ti	NSF 2005	non-cancer	rat	Ti dioxide in diet	Oral Ti absorption is low but is shown to occur in diet in mice (Sugibayashi *et al.*, 2008). Oral absorption occurs for Ti in water or in 0.5% hydroxypropylmethylcellulose including mineral form of Ti (DEPA, 2013). Oral absorption of TiO_2 is low, only ~6% for bulk form (DEPA, 2013).	<10%	The weight of evidence indicates that dermally applied TiO_2 particles do not reach viable skin cells, although there are findings that suggest penetration through the stratum corneum & openings of hair follicles, and that skin penetration may be affected by dispersing vehicles & skin conditions (NICNAS, 2013). Since dermal absorption is minimal & perhaps negligible, a RAF of 1% is selected.	0.01
PAHs	US EPA IRIS 2013	non-cancer	rat	BaP in peanut oil, by oral gavage	US EPA (2004, Exhibit 4-1) estimates absolute oral absorption of 58% for PAHs in rats dosed via starch solution.	58%	US EPA (2004, Exhibit 3-4) recommends absolute dermal absorption of 13% based on Wester *et al.* (1990). Since estimate of absolute absorption in key study is assumed to be >50% it is assumed to be complete. Thus dermal RAF is 13%.	0.13
	US EPA IRIS 2013	cancer	mice	in diet	US EPA (2004, Exhibit 4-1) estimates absolute oral absorption of 58–89% for PAHs, based on rats dosed via diet or starch solution.	58 – 89%		

References for Oral and Dermal Relative Absorption Factors (RAFs):

ATSDR. 2012a. Toxicological Profile for Cadmium. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. September 2012.

ATSDR. 2012b. Toxicological Profile for Manganese. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. September 2012.

Cal EPA DW. 2006. Public Health Goal for Chemicals in Drinking Water: Cadmium. California Public Health Goal (PHG). Pesticide and Environmental Toxicology Branch, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Sacramento, CA, USA.

Cal EPA DW. 2010. Public Health Goals for Chemicals in Drinking Water – Benzo(a)pyrene. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. September 2010.

DEPA. 2013. Systemic Absorption of Nanomaterials by Oral Exposure. Part of the "Better Control of Nano" Initiative 2012-2015. Danish Ministry of the Environment, Environmental Protection Agency. Environmental Project No. 1505, 2013. Copenhagen, Denmark.

HC CSD. 2010. Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors, Version 2.0. Prepared by: Contaminated Sites Division, Safe Environments Directorate. Health Canada. September 2010.

Magee B, Anderson P, Burmaster D. 1996. Absorption Adjustment Factor (AAF) Distributions for Polycyclic Aromatic Hydrocarbons (PAHs). *Human Ecol Risk Assess* 2:841-873. Cited in NEPI, 2000b.

NEPI. 2000a. Assessing the Bioavailability of Metals in Soil for Use in Human Health Risk Assessments. Bioavailability Policy Project Phase II – Metals Task Force Report. National Environmental Policy Institute. Washington, DC, USA. Summer 2000.

NEPI. 2000b. Assessing the Bioavailability of Organic Chemicals in Soil for Use in Human Health Risk Assessments. Bioavailability Policy Project Phase II – Organics Task Force Report. National Environmental Policy Institute. Washington, DC, USA. Fall 2000.

NHDES. 2006. Environmental Fact Sheet – Manganese: Health Information Summary. New Hampshire Department of Environmental Services. Concord, New Hampshire, USA. ARD-EHP-15.

NICNAS. 2013. Nano Titanium Dioxide Technical Information Sheet. Appendix: Nano Titanium Dioxide Toxicology Information and References. National Industrial Chemicals Notification & Assessment Scheme, Department of Health, Australian Government. Online at nicnas.gov.au

NSF International. 2005. Titanium and Titanium Dioxide. CAS # 7440-32-6 and 13463-67-7. Oral Risk Assessment Document. June 2005. Ann Arbor, MI, USA. 46 pp.

Schröder JL, NT Basta, J Si, SW Casteel, T Evans, M Payton. 2003. *In Vitro* Gastrointestinal Method to Estimate Relative Bioavailable Cadmium in Contaminated Soil. *Environ Sci Tech* 37:1365-1370.

Stroo HF, Jensen R, Loehr RC, Nakles DV, Fairbrother A, Liban CB. 2000. Environmentally Acceptable Endpoints for PAHs at a Manufactured Gas Plant Site. Environ Sci Tech 34:3831-3836. Cited in NEPI, 2000b.

Sugibayashi K, Todo H, Kimura E. 2008. Safety Evaluation of Titanium Dioxide Nanoparticles by their Absorption and Elimination Profile. *J Toxicol Sci* 33:293-298.

US EPA. 2004. Risk Assessment Guidance for Superfund Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). US Environmental Protection Agency. EPA/540/R/99/005. July 2004.

US EPA IRIS. 2013. Toxicological Review of Benzo[a]pyrene (CASRN 50-32-8). In Support of Summary Information on the Integrated Risk Information System (IRIS). U.S. Environmental Protection Agency. Washington, DC. Public Comment Draft. August 2013.

US EPA HESD. 2003. Health Effects Support Document for Manganese. Office of Water (4304T), U.S. Environmental Protection Agency. EPA-822-R-03-003. February 2003.

Wester RC, Maibach HI, Bucks DA, Sedik L, Melendres J, Liao C, Dizio S. 1990. Percutaneous Absorption of [^{14}C]DDT and [^{14}C]Benzo(a)pyrene in Soil. *Fund Appl Toxicol* 15:510–516.

WHO CICAD. 1999. Manganese and its Compounds – Concise International Chemical Assessment Document 12. World Health Organization. Geneva, Switzerland.

Appendix C: Glossary of Terms, Abbreviations, & Acronyms

The purpose of this glossary is to provide the reader with definitions of many of the technical terms, abbreviations, and acronyms which are used in this risk assessment. Where possible, the terms are defined in the context of how they are used in this particular risk assessment.

Absolute absorption
The fraction or percentage of a contaminant which is ingested, inhaled, or applied onto the skin surface that actually is absorbed and reaches the bloodstream. See also *RAF*.

ADD
See *Average Daily Dose*.

Adult
In this document, an adult is any person over the age of 20 years.

ATSDR
Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services.

Average Daily Dose (ADD)
In this document, the ADD is the contaminant exposure acquired through recreational activity at the creek. For each *COC* and each receptor age category (child, teen, and adult), the ADD was estimated for each pathway of the playing/fishing activity and accidental immersion event. These ADDs were then summed to obtain a total ADD of each age category.

BaP
Benzo(a)pyrene, one of the polycyclic aromatic hydrocarbons *(PAHs)*.

BaP_{eq}
Benzo(a)pyrene equivalents. For cancer assessment of PAHs, the upper estimate concentration was calculated for each individual carcinogenic PAH and then multiplied by its toxic equivalence factor (TEF) (reported in Kalberlah et al., 1995, the source of TEFs used in deriving the MOE, 2011b soil and groundwater standards); these were then summed in order to obtain an upper estimate value for total carcinogenic PAHs in (BaPeq).

Benchmark Dose (BMD)
It is the dose of a substance that is expected to result in a prespecified level of effect.

Bioavailability
The fraction or percentage of a contaminant which is ingested, inhaled, or applied onto the skin surface that reaches the bloodstream.

BMD
See *Benchmark Dose*.

BMDL
The lower confidence limit of a one-sided 95% confidence interval on the BMD.

BW
Body weight.

Cal EPA ATH
California Environmental Protection Agency, Air Toxics Hotspots Program.

Cal EPA ChRD
California Environmental Protection Agency, Child-Specific Reference Dose.

Cal EPA DW
California Environmental Protection Agency, Public Health Goals (PHGs) for Chemicals in Drinking Water.

Cancer Slope Factor (CSF).
See *Toxicity Reference Value*.

Central tendency (CT) estimate
An estimate which is an average, median, most likely, or most common value. See also *conservative estimate*.

Cd
Cadmium

Child
In this document, a child is any person from 5 to 11 years old.

CNS
Central nervous system.

COC
See *Contaminant of concern*.

Composite receptor
A receptor which is a composite of all relevant life stages for which exposure to a carcinogen will be evaluated. Since the development of cancer is a long-term process that may take many years to manifest, exposure to carcinogens is commonly assessed over a lifetime. This requires the use of a composite receptor.

Comprehensive human health risk assessment
A human health risk assessment with a high level of detail and complexity. Comprehensive risk assessments generally incorporate an extensive sampling plan, extensive site characterization, and site-specific characterization of receptors. The advantages of comprehensive assessments are (1) a general reduction in the degree of uncertainty in the conclusion, (2) a more accurate, realistic, reliable, and defensible quantification of human health risks, and (3) usefulness as a critical tool in the identification of complex remedial and risk management alternatives. Screening level and comprehensive risk assessments represent opposite ends of a continuum of complexity. Compare with *screening level human health risk assessment*.

Conservative estimate
An estimate that is cautious, but within reason, in that it produces a higher estimate of health effects in a risk assessment. See also *central tendency estimate*.

Contaminant of Concern (COC)
A contaminant which is present in the creek sediment or water at concentrations which exceed the concentrations used for screening and require further evaluation in the risk assessment.

CSF
Oral cancer slope factor. See *Toxicity Reference Value*.

CT
See *central tendency estimate*.

DEPA
Danish Ministry of the Environment, Environmental Protection Agency.

DW
Drinking water.

Exposure frequency (EF)
A single value that represents the rate at which exposure events occur within an exposure duration.

Exposure pathway
The pathway that a contaminant may take to cause exposure to the receptor. Exposure pathways link the source of the contaminant to its entry into the body. See also *exposure route*.

Exposure route
The route by which a contaminant can enter the body. Inhalation (breathing), ingestion (eating), and dermal contact (contact with skin) are exposure routes by which environmental contaminants can enter the body. See also *medium* and *exposure pathway*.

Exposure scenario
A hypothetical situation evaluated in a risk assessment. It incorporates a combination of exposure pathways to which a receptor may be subjected.

Frequent recreator
The hypothetical receptor evaluated in this document. It is described in detail in Section 2.3.1.

GIT
Gastro-intestinal tract

GLP
Good Laboratory Practice refers to quality practices for regulated non-clinical research and development.

GW
Groundwater

Hazard Quotient (HQ)
The ratio of estimated site-related exposure to a single contaminant over a specified duration to the exposure estimate at which no adverse health effects are expected. It is essentially the ratio between the ADD to the non-cancer TRV. If a HQ ≤ 1, then no adverse health effects are expected as a result of exposure. If a HQ > 1, then adverse health effects are possible. However, a HQ cannot be translated to a probability of the likelihood of adverse health effects and it is unlikely to be proportional to risk. It is also important to note that a HQ exceeding 1 does not necessarily mean that adverse health effects will occur.

HC CSD
Health Canada, Contaminated Sites Division

HC DW
Health Canada, Guidelines for Canadian Drinking Water Quality

HED
Human Equivalent Dose.

HHRA
See *Human Health Risk Assessment*.

HQ
See Hazard Quotient.

Human Health Risk Assessment (HHRA)
A tool or process for estimating potential human health risks to a defined set of individuals from exposure to particular contaminants. See also *Comprehensive Human Health Risk Assessment* and *Screening Level Human Health Risk Assessment*.

IARC
International Agency for Research on Cancer, World Health Organization.

ILCR
See *incremental lifetime cancer risk*.

In vitro
In an artificial environment outside a living organism. In this document, *in vitro* refers to studies conducted in a laboratory setup that does not use live animals.

In vivo
Within a living organism. In this document, *in vivo* refers to studies conducted using live animals.

Incremental lifetime cancer risk (ILCR)
The estimated probability or risk of developing cancer sometime during a lifetime as a result of site-related or activity-related exposure to a particular contaminant. The term "incremental" refers to the increased risk associated with a specific site or activity, over and above the risks experienced by the general population due to background environmental exposures. The ILCR is calculated by multiplying the *lifetime average daily dose* (LADD) by the *cancer slope factor* (CSF). The ILCR (an estimated cancer risk) is compared to a target cancer risk level, e.g., 1-in-a-million.

Infant
In this document, an infant is any person from birth to 6 months old.

IOM
Institute of Medicine of the National Academies

Key study
The toxicological or epidemiological study which forms the basis of the derivation of the toxicity reference value (TRV).

LADD
See *lifetime average daily dose*.

LED_{10}
Lower limit on effective dose 10%. In a toxicological study, it is the 95% lower confidence limit of the dose needed to produce an adverse effect in 10% of those exposed to the chemical, relative to control.

Level of conservatism
See *conservative estimate*.

Lifetime average daily dose (LADD)
The average daily dose for the composite receptor, averaged over a lifetime. It is the exposure rate used in the assessment of cancer risk.

Lipophilic
The ability or affinity of a chemical to dissolve in fats, oils, lipids, and non-polar solvents. Lipophilic substances tend to dissolve in other lipophilic substances, but not in hydrophilic substances such as water.

LOAEL
Lowest observed adverse effect level. The LOAEL in a study is the lowest dose of a contaminant that produce statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Medium (plural *media*)
Environmental media are parts of the environment that contain contaminants. The environmental media considered in this document are sediment and water from Talfourd Creek. Other examples of environmental media that exist are soil, house dust, air, tap water, and food. See also *exposure route*.

Mn
Manganese

MOE
Ontario Ministry of the Environment. See *MOECC*.

MOECC
Ontario Ministry of the Environment and Climate Change, formerly Ontario Ministry of the Environment

NCI
National Cancer Institute.

NEPI
National Environmental Policy Institute.

NHDES
New Hampshire Department of Environmental Services.

NICNAS
National Industrial Chemicals Notification & Assessment Scheme, Department of Health, Australian Government.

NOAEL
No observed adverse effect level. The NOAEL in a study is the dose of contaminant at which there are no statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control group. Effects may be produced at this dose, but they are not considered to be adverse.

NSF
National Sanitation Foundation International, USA.

NTP
National Toxicology Program. An inter-agency program run by the United States Department of Health and Human Services.

NYS
New York State.

PAH
Polycyclic aromatic hydrocarbon. See also *toxic equivalence* factor.

Pathway
See *exposure pathway*.

PND
Post-natal day. Indication of day occurring shortly after birth where PND 0 is the day of birth, e.g., PND 20 occurs 20 days after birth.

POD
See *point of departure*.

Point of departure (POD)
In a toxicological study, the POD is the dose-response point that marks the beginning of a low-dose extrapolation. This point can be the lower bound on dose for an estimated incidence (e.g., a NOAEL or LOAEL) or a change in response from a dose-response model (e.g., a BMDL).

Provisional tolerable weekly intake (PTWI)
A type of non-cancer TRV that is expressed as a weekly exposure rate and is considered provisional by the deriving agency.

ppm
A unit of measure expressed as parts per million. Equivalent to 1×10^{-6}.

PTWI
See *provisional tolerable weekly intake*.

Qualitative Assessment
Assessment based on contaminant characteristics or attributes, ranges of values, or rankings of values rather than on fixed numerical values. This approach is often used when *quantitative* assessment is not possible or not appropriate.

Quantitative Assessment
Assessment based on numerical estimates of exposure and toxicity.

RAF
See *relative absorption factor*.

Receptor
A hypothetical individual that could come into contact with contaminants. A receptor's exposure may not reflect that of a specific person because it is modelled on conservative potential exposures rather than on anyone's actual exposures.

Recreational use
In this document, recreation is defined as activity that people engage in during their free time for the purpose of amusement, which involves mostly playing and fishing.

Relative absorption factor (RAF)
Absorption of a contaminant varies based on the *medium*, the *receptor*, and the *exposure route*. Therefore, when estimating exposure to a contaminant, it is important to consider the contaminant absorption in the *exposure scenario* of interest compared to the absorption in the *key study* which forms the basis of the *TRV* used in the risk assessment. A RAF is a ratio of the contaminant absorption in the *exposure scenario* being assessed to the absorption estimated or assumed for the *key study* of the *TRV*.

RfD
Reference Dose. An RfD is the term used by *US EPA* for an oral non-cancer *TRV*.

RIVM
National Institute of Public Health & Environmental Protection, The Netherlands.

Route of exposure
See *exposure route*.

Route-to-route extrapolation
A prediction of the amount of contaminant intake occurring by one *exposure route* that would produce the same toxic response as a given amount of contaminant intake by another *exposure route*.

Scenario
See *Exposure scenario*.

Screening Level Human Health Risk Assessment (SLHRA)
A human health risk assessment with a limited level of detail and complexity. SLHRAs generally incorporate limited site-specific data, limited site characterization, and a receptor characterization that is limited to standard, conservative assumptions. The advantage of a SLHRA is that it is the simplest and most streamlined form of risk assessment. A SLHRA may be used to highlight the priority issues of concern at a site or to inform a more comprehensive risk assessment. Since it is based on maximal exposure, a SLHRA can identify if no unacceptable human health risks exist. However, if a SLHRA concludes that unacceptable human health risks may exist, it may be appropriate to undertake a comprehensive risk assessment prior to defining remedial or risk management options. Screening level and comprehensive risk assessments represent opposite ends of a continuum of complexity. Compare with *comprehensive human health risk assessment*.

SLHRA
See *Screening Level Human Health Risk Assessment*.

TEF
See *toxic equivalence factor*.

Teen
In this document, a teen is any person from 12 to 19 years old.

Ti
Titanium.

Time-weighted average
A weighted average is an average in which each quantity to be averaged is assigned a weight; these weightings determine the relative importance of each quantity on the overall average. A time-weighted average is an average in which each quantity to be averaged is assigned a weight based on time. In this document, some parameters are time-weighted averages with equal weighting given to each of the four seasons of the year in order to calculate the averages.

TiO_2
Titanium dioxide.

TK
See *toxicokinetics*.

Toddler
In this document, a toddler is any person from 7 months to 4 years old.

Toxic Equivalence Factor (TEF)
Using TEFs is a method of evaluating structurally related compounds (such as *PAHs*) which share a common mechanism of action. For the *PAHs*, benzo(a)pyrene (the reference standard) is assigned a TEF of 1. Based on carcinogenic potency, the values of the TEFs of each individual PAH indicates how toxic they are relative to benzo(a)pyrene. The carcinogenic potency of the PAHs is also considered additive.

Toxicity Reference Value (TRV)
A TRV is a toxicological index that, when compared with exposure, is used to qualify or quantify a risk to human health. A TRV for assessing threshold (non-cancer) effects is an estimate of continuous exposure that is likely to be without a considerable risk of adverse health effects. A TRV for assessing non-threshold (cancer) effects from oral exposure is a cancer slope factor (CSF). It is an estimate of increased cancer incidence per unit of exposure to a contaminant. A CSF is usually expressed as a proportion of a population affected per mg of contaminant per kg of body weight per day.

Toxicokinetics
The study of a contaminant's entry into the body and what happens to it once it is in the body.

Transient
Pass through or by a place with only a brief stay, as opposed to being resident; short in its duration or stay.

TRV
See *Toxicity Reference Value*.

$UCDL_{10}$
The 95% lower confidence limit of the Urinary *Cd* Level associated with a 10% extra risk of low molecular weight proteinuria.

UF
See *uncertainty factor*.

Uncertainty Factor (UF)
A factor used in calculating a non-cancer *TRV* from experimental data. UFs are typically used to account for various types of uncertainties in the experimental data used:

- UF_A – interspecies: uncertainty in extrapolating from animal to human data
- UF_H – intraspecies: variation in sensitivity within the human population
- UF_L – uncertainty in using a *LOAEL* rather than a no-effect level
- UF_S – uncertainty in extrapolating data obtained from a study that covers less than the full life of the exposed animal or human
- UF_D – uncertainty associated with the adequacy of the database of experimental data
- UF_X – any remaining areas of uncertainty

US EPA
United States Environmental Protection Agency.

US EPA DW
United States Environmental Protection Agency, Drinking Water Health Advisory.

US EPA HEAST
United States Environmental Protection Agency, Superfund Health Effects Assessment Summary Tables.

US EPA HESD
United States Environmental Protection Agency, Health Effects Support Document.

US EPA IRIS
United States Environmental Protection Agency, Integrated Risk Information System.

US EPA PPRTV
United States Environmental Protection Agency, Provisional Peer Reviewed Toxicity Value.

WHO
World Health Organization.

WHO CICAD
World Health Organization, Concise International Chemical Assessment Document.

WHO JECFA
World Health Organization, Joint FAO/WHO Expert Committee on Food Additives.