TECHNICAL REPORT

Sediment Guidelines for Recreational Use of the St. Lawrence River Waterfront at Cornwall, Ontario

Ontario Ministry of the Environment, Conservation and Parks

Report prepared by:
Marco Pagliarulo, M.Sc.

Human Toxicology & Air Standards Section
Technology Assessment & Standards Development Branch

June 2019
FINAL

Plain Language Summary

Cornwall is located in eastern Ontario along the north shore of the St. Lawrence River. Concerns have been expressed regarding risks from exposures to contaminants in St. Lawrence River sediment at Cornwall during recreational activities.

The Technology Assessment and Standards Development Branch (TASDB) of the Ontario Ministry of the Environment, Conservation and Parks (MECP) developed Cornwall-specific recreational sediment guidelines to inform and guide risk management of the Cornwall waterfront in support of the Cornwall Sediment Strategy. These guidelines are based on human health risks associated with potential exposures to sediment contaminants during recreational activities, including swimming and wading.

Caveats regarding these recreational sediment guidelines:

- They are not standards and they are non-regulatory.
- They are based on exposures from *recreation* along the Cornwall waterfront and are therefore **not appropriate for application to other exposure scenarios.**
- **Consumption of St. Lawrence River fish was not a part of this assessment**. For guidance on consuming fish from the St. Lawrence River, see the ministry's Guide to Eating Ontario Sport Fish, available online at www.ontario.ca/page/eating-ontario-fish-2017-18.
- **Since they have been developed specifically for the Cornwall waterfront, these guidelines may not be appropriate for other sites in Ontario.**
- The conclusions and recommendations presented here are based on current science and on the site description at the time of preparation of this report. New sediment data may indicate contaminant concentrations higher than those used in this assessment or the presence of contaminants not evaluated at all in this assessment. In either of these cases, re-assessment may be warranted. Furthermore, to keep up-to-date with current science, revisiting the current approach and input parameter values is recommended for the future.

Contaminant concentrations in Cornwall waterfront sediments were screened against health-based criteria and background concentrations, identifying three contaminants requiring the most attention: lead, mercury, and benzo[a]pyrene (B[a[P). (B[a]P is a member of a group of compounds called polycyclic aromatic hydrocarbons (PAHs) and is used as an indicator of other PAHs.) These are the contaminants for which the sediment guidelines were developed. Though some other contaminants were above the screening criteria, their maximum concentrations were not excessive, so risks from exposure to those contaminants are likely to be minimal.

Cornwall-specific sediment guidelines were developed using information on (i) how people may be exposed to sediment contaminants along the Cornwall waterfront, (ii) toxicity information for the three contaminants, and (iii) benchmarks of acceptable risk.

The Cornwall waterfront sediment guidelines were derived mainly based on small children because their behaviours lead to greater exposures than older children or adults.

The resultant recreational guidelines for lead, mercury, and B[a]P in sediment along the Cornwall waterfront are provided in Table A. To inform risk management, this table also provides guidance regarding risk levels and risk management.

Table A: Recreational Sediment Guidelines for the Cornwall Waterfront

Sediment Guidelines (ppm)			**Risk Management (RM)**	
Lead	**Mercury**	**B[a]P[#]**	**Level***	**Interpretation of Risk**
≤120	≤80	≤3	RM0	The concentrations are acceptable. Risks from long-term recreational exposures are negligible. No action necessary.
>120 to ≤610	>80 to ≤410	>3 to ≤30	RM1	The concentrations exceed conservative benchmarks. The potential for unacceptable exposures and associated risks cannot be discounted but are unlikely. Possible risk management measures include communication or signage.
>610	>410	>30	RM2	Risks from long-term recreational exposures are considered to be elevated. Measures to block exposure pathways may be warranted.

[#] The B[a]P guidelines are for total PAHs summed as B[a]P-equivalents.

* RM0, RM1, and RM2 refer to the level of *risk management* recommendations.

Note that the sensitivity analysis (Appendix B) indicates that oral exposures on land (during shoreline play, walking, or recreational fishing) and dermal exposures have significant impacts on the calculated sediment guidelines, whereas oral exposures via wading and swimming have a negligible impact.

Also note that the nearshore area of the Cornwall waterfront along the St. Lawrence River is made up of rock, gravel, or macrophyte beds. **Since there are no sediments within the first few metres of shore, it means the exposure calculations are over-assumptions, making these sediment guidelines conservative (cautious) when applied to recreational use of the Cornwall waterfront.** This should be taken into consideration when interpreting sediment sampling data.

To ensure the guidelines are applied properly, the following process is recommended:

1) Sediment concentrations of polycyclic aromatic hydrocarbons (PAHs) should be summed as benzo[a]pyrene-equivalents (B[a]P_{eq}) using *human health* toxic equivalency factors (TEFs) provided in Table B below. While these recommended TEFs from Kalberlah *et al*., 1995 (as cited in WHO EHC, 1998) are used in the ministry's brownfields program, it is acceptable to use the TEFs used by Health Canada or by the Canadian Council of Ministers of the Environment (CCME) and the sources from which those TEFs were obtained.

Table B: Human Health Toxic Equivalency Factors (TEFs) for PAHs

PAH	TEF
acenaphthene	0.001
acenaphthylene	0.01
anthracene	0.01
benz[a]anthracene	0.1
benzo[a]pyrene	1

PAH	TEF
benzo[b]fluoranthene	0.1
benzo[g,h,i]perylene	0.01
benzo[k]fluoranthene	0.1
chrysene	0.01

PAH	TEF
dibenz[a,h]anthracene	1
fluoranthene	0.01
indeno[1,1,3-cd]pyrene	0.1
pyrene	0.001

2) An upper estimate of the mean concentration (the 95% upper confidence limit of the mean; UCLM) or a conservative estimate (e.g., 95th percentile) should be calculated for lead, mercury, and B[a]P in sediment and compared to the sediment guidelines in Table A, though other statistical values may be considered.

Technical Summary

Cornwall is located in eastern Ontario along the north shore of the St. Lawrence River. Concerns have been expressed regarding risks from exposures to contaminants in St. Lawrence River sediment at Cornwall during recreational activities.

The Technology Assessment and Standards Development Branch (TASDB) of the Ontario Ministry of the Environment, Conservation and Parks (MECP) **developed Cornwall-specific recreational sediment guidelines to inform and guide risk management of the Cornwall waterfront in support of the Cornwall Sediment Strategy**. These guidelines are based on human health risks associated with potential exposures to sediment contaminants during recreational activities, including swimming and wading.

Caveats regarding these recreational sediment guidelines:

- They are not standards and they are non-regulatory.
- They are based on exposures from *recreation* along the Cornwall waterfront and are therefore **not appropriate for application to other exposure scenarios.**
- **Consumption of St. Lawrence River fish was not a part of this assessment**. For guidance on consuming fish from the St. Lawrence River, see the ministry's Guide to Eating Ontario Sport Fish, available online at www.ontario.ca/page/eating-ontario-fish-2017-18.
- **Since they have been developed specifically for the Cornwall waterfront, these guidelines may not be appropriate for other sites in Ontario.**
- The conclusions and recommendations presented here are based on current science and on the site description at the time of preparation of this report. New sediment data may indicate contaminant concentrations higher than those used in this assessment or the presence of contaminants not evaluated at all in this assessment. In either of these cases, re-assessment may be warranted. Furthermore, to keep up-to-date with current science, revisiting the current approach and input parameter values is recommended for the future.

Contaminant concentrations in Cornwall waterfront sediments were screened against health-based criteria and background concentrations which identified lead, mercury, and benzo[a]pyrene (B[a]P) as the contaminants of concern (COCs). (B[a]P used as an indicator of polycyclic aromatic hydrocarbons (PAHs)). These are the contaminants for which guidelines were developed. Though some other contaminants were above the screening criteria, their maximum concentrations were not excessive, so risks from exposure to those contaminants are likely to be minimal.

Site-specific sediment guidelines for the COCs were developed using (i) recreational exposure scenarios for the Cornwall waterfront, (ii) toxicity reference values (TRVs), and (iii) target risk levels: hazard quotients (HQs) and incremental lifetime cancer risk levels (ILCRs).

The Cornwall waterfront sediment guidelines were derived assuming a toddler is exposed 5 days/week during the summer and 2 days/week during spring and fall. Since a toddler (1 – 3 years old) has a lower body weight and higher incidental sediment ingestion rate than a child (4 – 11 years old), a toddler exposed 1 hour/day is roughly equivalent to a child exposed 3 hours/day.

The resultant recreational guidelines for lead, mercury, and B[a]P in sediment along the Cornwall waterfront are provided in Table A. To inform risk management, Table B provides guidance regarding risk levels and risk management.

Table A: Recreational Sediment Guidelines for the Cornwall Waterfront

Sediment Guidelines (ppm)			**Risk Management (RM)**	
Lead	**Mercury**	**B[a]P[#]**	**Level***	**Interpretation of Risk**
≤120	≤80	≤3	RM0	The concentrations are acceptable. Risks from long-term recreational exposures are negligible. No action necessary.
>120 to ≤610	>80 to ≤410	>3 to ≤30	RM1	The concentrations exceed conservative benchmarks. The potential for unacceptable exposures and associated risks cannot be discounted but are unlikely. Possible risk management measures include communication or signage.
>610	>410	>30	RM2	Risks from long-term recreational exposures are considered to be elevated. Measures to block exposure pathways may be warranted.

[#] The B[a]P guidelines are for total PAHs summed as B[a]P-equivalents.

* RM0, RM1, and RM2 refer to the level of *risk management* recommendations.

Note that the sensitivity analysis (Appendix B) indicates that oral exposures on land (during shoreline play, walking, or recreational fishing) and dermal exposures have significant impacts on the calculated sediment guidelines, whereas oral exposures via wading and swimming have a negligible impact.

Also note that the nearshore area of the Cornwall waterfront along the St. Lawrence River is made up of rock, gravel, or macrophyte beds. **Since there are no sediments within the first few metres of shore, it means the exposure calculations are over-assumptions, making these sediment guidelines conservative when applied to recreational use of the Cornwall waterfront.** This should be taken into consideration when interpreting sediment sampling data.

To ensure the guidelines are applied properly, the following process is recommended:

1) Sediment concentrations of polycyclic aromatic hydrocarbons (PAHs) should be summed as benzo[a]pyrene-equivalents (B[a]P_{eq}) using *human health* toxic equivalency factors (TEFs) provided in Table A below. While these recommended TEFs from Kalberlah *et al*., 1995 (as cited in WHO EHC, 1998) are used in the ministry's brownfields program, it is acceptable to use the TEFs used by Health Canada or by the Canadian Council of Ministers of the Environment (CCME) and the sources from which those TEFs were obtained.

Table B: Human Health Toxic Equivalency Factors (TEFs) for PAHs

PAH	TEF	PAH	TEF	PAH	TEF
acenaphthene	0.001	benzo[b]fluoranthene	0.1	dibenz[a,h]anthracene	1
acenaphthylene	0.01	benzo[g,h,i]perylene	0.01	fluoranthene	0.01
anthracene	0.01	benzo[k]fluoranthene	0.1	indeno[1,1,3-cd]pyrene	0.1
benz[a]anthracene	0.1	chrysene	0.01	pyrene	0.001
benzo[a]pyrene	1				

2) The 95% UCLM (95% upper confidence limit of the mean) or a conservative estimate (e.g., a 95^{th} percentile) should be calculated for lead, mercury, and B[a]P in sediment and compared to the sediment guidelines shown in Table A, though other statistical values may be considered.

Table of Contents

1.0 Introduction

Cornwall is located in eastern Ontario along the north shore of the St. Lawrence River. At the turn of the 20th century, industries began operating along the Cornwall waterfront releasing mercury and other contaminants; by the 1990s the major direct sources of local contamination had either ceased operations or had installed wastewater treatment to control releases; this area of the St. Lawrence has also received contaminants from urban and rural surface runoff and other sources upstream of Cornwall (deBarros & Anderson, 2010).

Concerns have been expressed regarding risks associated with potential exposures to contaminants in St. Lawrence River sediment at Cornwall for people recreating along the waterfront. This report discusses the assessment conducted by the Human Toxicology and Air Standards Section, Technology Assessment and Standards Development Branch (TASDB), Ontario Ministry of the Environment, Conservation and Parks (MECP), to develop **site-specific sediment guidelines for recreational use of the Cornwall waterfront**.

These sediment guidelines are intended to inform and guide risk management of the Cornwall waterfront in support of the Cornwall Sediment Strategy. In the context of this assessment, recreational activities along the Cornwall waterfront include swimming and wading, shoreline play, walking, and recreational fishing. However, they do not include fish consumption.

1.1 Description and Approach

Sediment guidelines for the Cornwall waterfront were developed using an approach similar to a human health risk assessment. The following process was used:

- Contaminants to be considered were identified based on existing data (Sections 2.1 & 2.2);
- Recreational areas were described (Section 2.3);
- Recreational exposure scenarios and exposure pathways (how people may be exposed) were identified (Sections 2.4 & 2.5);
- Target risk levels were selected (Section 3.1);
- Parameter values were selected (Section 3.2);
- Toxicological values were identified (Section 4.0 & Appendix A);
- Potential exposures to recreational users were expressed in the form of equations with the selection of various exposure parameters (Section 5.0);
- A model (in MS Excel) was built to calculate the sediment guidelines;
- The sediment guidelines were determined and presented (Section 6.1);
- Guidance on the application of the guidelines was presented (Section 6.2);
- A sensitivity analysis was conducted (Appendix B);
- Uncertainties and limitations were identified (Appendix C).

1.2 Scope of the Sediment Guidelines Presented in this Document

These recreational sediment guidelines only consider risks associated with exposures to contaminants in sediments of the Cornwall waterfront during recreational activities.

- The guidelines do not consider exposures to these contaminants from other sources.
- They are not standards and they are non-regulatory.
- They are based on exposures from *recreation* along the Cornwall waterfront and are therefore **not appropriate for application to other exposure scenarios** and do not replace the Brownfields generic standards.
- **Consumption of St. Lawrence River fish was not a part of this assessment**. For guidance on consuming fish from the St. Lawrence River, see the ministry's Guide to Eating Ontario Sport Fish, available online at www.ontario.ca/page/eating-ontario-fish-2017-18.
- **Since they have been developed specifically for the Cornwall waterfront, these guidelines may not be appropriate for other sites in Ontario.**
- The conclusions and recommendations presented here are based on current science and on the site description at the time of preparation of this report. New sediment data may indicate contaminant concentrations higher than those used in this assessment or the presence of contaminants not evaluated at all in this assessment. In either of these cases, re-assessment may be warranted. Furthermore, to keep up-to-date with current science, revisiting the current approach and input parameter values is recommended for the future.

2.0 Problem Formulation

This section describes the contaminated area, the available contaminant data, the people potentially exposed, and the ways people could be exposed.

2.1 Data Selection

The selection of contaminants for which sediment guidelines would be developed was based on existing sediment data which were collected as part of the Cornwall Sediment Strategy. Surface sediment contaminant data for the Cornwall waterfront were obtained from the St. Lawrence River Institute: Richman, 1996; Pelletier, 2010; Milani and Grapentine, 2015; Razavi *et al*., 2015; Windle and Ridal, 2016.

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

The purpose of this screening step was not to identify the contaminants which are important, but rather, to identify the *most* important contaminants for the development of sediment guidelines for Cornwall. Contaminants of concern (COCs) were selected by screening Cornwall sediment contaminant data using soil criteria developed for residential exposures. The process to identify COCs is summarized as follows:

i. The maximum sediment concentration of each contaminant was identified from the available data. [For polycyclic aromatic hydrocarbons (PAHs), the maximum concentration of each PAH was converted to benzo[a]pyrene equivalents (B[a]P_{eq}) using toxic equivalency factors (TEFs) for PAHs (as described below in Section 6.2) and these were summed.]

ii. Contaminants naturally found in soil at high levels (not associated with anthropogenic sources) were screened out due to their generally low human toxicity at environmental concentrations. Government agencies consider them innocuous (TCEQ, 2007; HC CSD, 2010 Part V). These include calcium, iron, magnesium, nitrogen, phosphorus, potassium, sodium, and sulphur.

iii. For the remaining contaminants, maximum sediment concentrations were compared to human health-based residential soil criteria protective against adverse health effects. (Health-based *soil* criteria were used because reliable and complete sets of health-based *sediment* criteria are lacking. It is common risk assessment practice to use soil as a surrogate for sediment if necessary. The MECP residential/parkland soil criteria for brownfields are sufficiently conservative to be suitable for screening purposes here.) MECP's human health component values (HHCVs) for the S1 pathway (direct soil contact – oral and dermal routes of exposure) were used (MOE, 2011).

    HHCVs are generally 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, 2017). 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 the ministry's HHCVs, US EPA's RBCs were adjusted to 20% of the tolerable daily intake.

iv. Contaminants not eliminated after screening against human health-based soil criteria were then screened against Ontario Typical Range (OTR) background soil concentrations. (Since Ontario background sediment concentrations are lacking for most of the contaminants for which there are Cornwall sediment concentrations, background *soil* values were used instead.)

    MECP's Table 1 background soil concentrations for residential/parkland land use were used (MOE, 2011); for contaminants for which no Table 1 background soil value is reported, background soil values were obtained from MOE (2011) Table 8.2 (OTR_{98} Old Urban Parks).

Where MOE (2011) background soil concentrations were lacking, values were obtained from a USGS (2005) survey of non-impacted soils across Canada and the US; from USGS data provided in Table 2 (0–5 cm soil depth), 98th percentiles were calculated as they represent a similar statistic used to obtain MOE (2011) background OTR concentrations.

Table 2-1 shows the screening values and maximum contaminant concentrations reported in Cornwall waterfront sediment. Yellow highlighting indicates exceedances of the criteria.

Note that the screening criteria used here are based on residential exposures to soil and on background concentrations of contaminants in soil, whereas the recreational sediment guidelines derived here are based on recreational exposure scenarios where sediment is the medium of exposure.

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

Contaminant	Max Sediment Conc (ppm) [sample size]	Naturally at High Conc	Human Health Soil Criteria[1] (ppm)	Background Soil[2] (ppm)	Ratio of Max to Screening Criterion
aluminum	18,000 [6]		(15,400)[1]	26,000	0.69
antimony	1.4 [6]		7.5	1.3	0.19
arsenic	30 [47]		0.95	18	1.7
barium	140 [6]		3800	180	0.037
beryllium	0.67 [6]		38	2.5	0.018
bismuth	0.3 [6]		nv[3]	(0.40)[2]	0.75
cadmium	1.5 [47]		0.69	1.2	1.3
calcium	73,000 [6]	√			
chromium	190 [47]		28,000	70	0.0068
cobalt	8.1 [6]		22	21	0.37
copper	662 [47]		600	92	1.1
gallium	5.6 [6]		nv	(18)	0.31
iron	130,000 [47]	√			
lanthanum	21 [6]		nv	(50)	0.42
lead	5200 [47]		200	120	26
lithium	15 [6]		(32)	nv	0.47
magnesium	14,000 [6]	√			
manganese	499 [47]		(360)	1400	0.36
mercury (total)	37 [166]		9.8	0.27	3.8
methylmercury	0.0032 [8]		2	nv	0.0016
molybdenum	2.1 [6]		110	2	0.019
nickel	290 [47]		330	82	0.88
phosphorus	1100 [6]	√			
potassium	4800 [6]	√			
rubidium	59 [6]		nv	(140)	0.42
selenium	3 [6]		110	1.5	0.027
sodium	590 [6]	√			
strontium	130 [6]		(9400)	77	0.014
thallium	0.47 [6]		0.29	1	0.47
uranium	1.3 [6]		23	2.5	0.057
vanadium	41 [6]		39	86	0.48
zinc	5000 [47]		5600	290	0.89
B[a]P[4]	19 [24]		0.078	0.3	63
dioxins & furans	26 pg/g [6]		48 pg/g	7 pg/g	0.54
PCBs (total)	0.57 [21]		0.35	0.3	1.6
petroleum hydrocarbons F1	<10 [75]		6900	25	0.0014
petroleum hydrocarbons F2	153 [75]		3100	10	0.049
petroleum hydrocarbons F3	2660 [75]		5800	240	0.46
petroleum hydrocarbons F4	4100 [75]		6100	120	0.67
benzene	<0.031 [51]		9.3	0.02	0.0033
ethylbenzene	<0.081 [51]		2100	0.05	0.000038
toluene	<0.36 [51]		1700	0.2	0.00021
xylenes	<0.16 [51]		4200	0.05	0.000038

1 Human health criteria in parentheses are adapted from US EPA Region III Risk-Based Concentration, set to a HQ of 0.2
2 Background values in parentheses are 98th percentile background values from USGS (2005)
3 *nv* means that no value is available from MOE (2011) or from US EPA / USGS
4 B[a]P sediment concentration was calculated as sum of PAHs in B[a]P-equivalents; background B[a]P is for B[a]P alone.

Maximum sediment concentrations of arsenic, cadmium, copper, lead, mercury, B[a]P, and polychlorinated biphenyls (PCBs) exceeded both the health-based and background criteria. **Lead, mercury, and B[a]P were the contaminants with the greatest exceedances and therefore are identified as the contaminants requiring the most attention.** The maximum concentrations of arsenic, cadmium, copper, and PCBs were less than two times their respective criteria indicating that risks from exposures to these contaminants are minimal. Furthermore, at least in part, risks from exposures to these contaminants may be resolved from risk management decisions made for lead, mercury, and B[a]P.

2.3 Description of Recreational Areas

Based on discussions with the Eastern Regional Office (MECP) and Environment and Climate Change Canada and an examination of photographs of the area of interest, the Cornwall waterfront can be described as follows:

- parks
- commercial docks
- residences with private docks and waters with macrophytes
- swim clubs
- rip-rap shoreline
- gravelly soil/sediment
- steep slope right down to water
- areas covered with grass and/or large rocks
- areas of very little exposed sediment
- areas of deep-water swimming or scuba diving

Along the waterfront, there is a lack of sand beaches and areas conducive to intense interaction with the sediment as would be more likely on a sand beach.

2.4 Description of Receptors and Exposure Pathways

Receptors and exposure pathways are dependent on the site and were determined using guidance from various agencies and professional judgement. Life stages considered were toddler, child, teen, and adult. The follow exposure pathways were considered:

- Sediment ingestion during shoreline play, walking, or recreational fishing
- Sediment ingestion during wading or swimming
- Dermal exposure to sediment during shoreline play, walking, recreational fishing, wading, or swimming

2.5 Conceptual Site Model

An exposure pathway is complete when contaminants from a site reach the receptor. If the source, release/transport mechanism, medium, exposure route, or the receptor itself is missing from the exposure pathway, then it is incomplete and exposure does not occur. 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 assessment is shown in Figure 2-1; ingestion of sediment and dermal exposure to sediment are assessed quantitatively.

Figure 2-1: Conceptual Site Model

PRIMARY SOURCE	RELEASE MECHANISM	EXPOSURE MEDIA	EXPOSURE ROUTE	Shoreline play, walking, recreational fishing	Wading, swimming
historic & current industrial activities, other urban & rural activities	discharges, runoff, deposition from upstream	sediment	ingestion	√	√
			dermal	√	√
			inhalation	□	□

Notes: √ = complete exposure pathway
□ = complete pathway but not assessed. See below.

Sediment ingestion and dermal exposure to sediment were quantitatively assessed for *shoreline play, walking, and recreational fishing* and for *wading and swimming*.

Inhalation of sediment during any of the above activities is considered a complete exposure pathway but was not quantitatively assessed. This exposure route is expected to be minor relative to the ingestion and dermal exposure pathways.

The contaminants identified in Cornwall sediments tend to occur in water at low concentrations. Therefore, the impact of water on overall exposures is minimal. However, exposure to contaminants in sediments that are suspended in water has been included.

3.0 Exposure Assessment

This section describes how exposures to the COCs were estimated for the Cornwall waterfront. Normally in a risk assessment, the contaminant concentration is an input parameter and the risk level [hazard quotient (HQ) for non-cancer or incremental lifetime cancer risk (ILCR) for cancer] is an output parameter, but the reverse is the case here.

3.1 Target Hazard Quotient (HQ) and Incremental Lifetime Cancer Risk Level (ILCR)

To develop the recreational site-specific sediment guidelines for the COCs identified in Section 2.2, target HQ and ILCR values were selected. Because of the inherent uncertainty in assessing risks from exposures to sediments, two target values were selected which resulted in three risk ranges. The target values and risk ranges are shown in Table 3-1.

Table 3-1: Target Hazard Quotient & Incremental Lifetime Cancer Risk Level

Value Selected for Target HQ (non-cancer) or ILCR (cancer)	Interpretation of Risk Levels for this Assessment
HQ < 0.2	The concentrations are acceptable. Risks from long-term recreational exposures are negligible.
ILCR < 10^{-6}	
HQ from 0.2 to 1	The concentrations exceed conservative benchmarks. The potential for unacceptable exposures and associated risks cannot be discounted but are unlikely.
ILCR from 10^{-6} to 10^{-5}	
HQ > 1	Risks from long-term recreational exposures are considered to be elevated.
ILCR > 10^{-5}	

A HQ of 0.2 and an ILCR level of 1-in-a-million (10^{-6}) are considered negligible because they allow for the potential significant co-exposures to the same contaminant from non-sediment sources (*i.e*, background exposures). A HQ of 1 and an ILCR level of 1-in-100,000 (10^{-5}) are considered a higher risk level because they don't account for potential co-exposures to the same contaminant from other (non-sediment) sources.

3.2 Receptor Characterization – Assumptions and Parameters Used

The receptors assessed here are recreators using the Cornwall waterfront. The selected assumptions and parameters were based on potential activities.

Note that the nearshore area of the Cornwall waterfront along the St. Lawrence River is made up of rock, gravel, or macrophyte beds. **There are no sediments within the first few metres of shore; therefore, the exposure calculations are over-assumptions, making these sediment guidelines conservative when applied to recreational use of the Cornwall waterfront.**

For non-cancer risk calculations, the receptor age category is the toddler because toddler exposure rates are higher than other age categories due to their behaviour and physiological characteristics. For cancer risk calculations, the receptor is assumed to be recreating at the waterfront from 1 year of age for the remainder of the lifespan.

All receptor parameters are summarized in Table 3-2; the details and rationales pertaining to the values selected for each parameter are described in Sections 3.2.1 to 3.2.7. The age ranges for the age categories were based on those presented in Richardson and Stantec (2013) as they more accurately correspond to the behaviours associated with each category.

Some important assumptions in this assessment are as follows:

- People visit the Cornwall waterfront 5 days/week during summer and 2 days/week during spring and fall.
- Each visit involves ingestion of sediment and getting sediment on all exposed body parts (body parts not covered with clothing).

Note that the sensitivity analysis (Appendix B) indicates that oral exposures on land (during shoreline play, walking, or recreational fishing) and dermal exposures have significant impacts on the calculated sediment guidelines, whereas oral exposures via wading and swimming have a negligible impact.

Table 3-2: Receptor Parameters

Parameter (unit)	Symbol	Exposure Pathway	Toddler (1–3 y)	Child (4–11 y)	Teen (12–19 y)	Adult (20+ y)	Source
Body Weight (kg)	BW	All	15.3	35.2	65.2	76.5	Richardson & Stantec, 2013
Sediment Ingestion Rate (mg/h)	SedIR	Oral: playing, walking, fishing	33	30	10	10	Low-exposure activity areas have gravelly soil/sediment & land is mostly grass-covered. US EPA EFH (2017) central tendency for soil only (soil & outdoor dust): 40 mg/d (1 - <2 y.o.), 30 mg/d (2 - <6 y.o., 6 - <12 y.o.), 10 mg/d (12 y.o. +)
	SedIR	Oral: wading, swimming	7.7				Recommended by HC (2017) for suspended sediment ingestion during in-water activities.
Skin Surface Area (cm^2)	SA head	Dermal: playing, walking, fishing, wading, swimming	663	660	740	1250	Vales are from US EPA EFH (2011), Table 7-2, *Mean Surface Area by Body Part*. For the toddler, values are average of 1 year olds, 2 y.o., and 3 to <6 y.o. For the child, 6 to <11 y.o. are used. For the teen, averages of 11 to <16 y.o. and 16 to <21 y.o. are used. For the adult, averages of adult male and adult female are used.
	SA hands		317	510	775	980	
	SA arms		877	1510	2480	2755	
	SA legs		1570	3110	5130	6400	
	SA feet		400	730	1085	1295	
	SA torso		2503	4280	6945	7405	
	SA Total		6330	10,800	17,155	20,085	
Sediment Loading Rate ($mg/cm^2/d$)	SL		0.04	0.04	0.02	0.02	0.04 is geometric mean of daycare children (1 - 6.5 y.o.) playing indoors and outdoors; 0.02 is geometric mean of adult groundskeepers (US EPA, 2004)
Exposure Frequency (h/d)	$EF_{h/d}$	Oral: playing, walking, fishing	1				US EPA EFH (2011) table 16-1: averages 30–83 min/d (doers only) for time spent playing on sand/gravel or playing on dirt, ages up to <21 y.o.
Exposure Frequency (d/wk)	$EF_{d/wk}$	Oral: playing, walking, fishing & Dermal: all	summer: 5 spring/autumn: 2 winter: 0				Professional judgement.
Exposure Frequency (h/mo)	$EF_{h/mo}$	Oral: wading, swimming	22				Professional judgement: (1 h/d) x (5 d/wk) x (52 wks/y) ÷ (12 mo/y) ≈ 22 d/mo
Exposure Frequency (mo/y)	$EF_{mo/y}$		3				Professional judgement. It is warm enough for outdoor swimming approximately 3 mo/y.
Exposure Duration (y)	ED	All	3	8	8	61	Exposure durations from Richardson & Stantec (2013); total averaging time for lifetime is 81 y including one year of infancy.
Averaging Time (y)	AT		3	8	8	61	

3.2.1 Body Weight (BW)

The body weight (BW) parameter values selected for this assessment are averages from Richardson and Stantec (2013) and are specific to the Canadian population: 15.3, 35.2, 65.2, and 76.5 kg for the toddler, child, teen, and adult, respectively. Richardson and Stantec (2013) explains that changes from previous values are due to increasing mean BW for most age categories except toddlers; toddler BW was mistakenly assumed in the Richardson (1997) compendium to have increased but has since been corrected.

3.2.2 Sediment Ingestion Rate (SedIR)

Estimated rates of incidental 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.

The types of substrate along the Cornwall waterfront (described in Section 2.3) are less conducive to behaviours leading to intense contact with sediments expected along sandy beaches. The 2017 update for Chapter 5 (Soil and Dust Ingestion) of US EPA's Exposure Factors Handbook recommends central tendency ingestion rates for soil only (which includes soil and outdoor settled dust) of 40 mg/d for 1 to <2 years old, 30 mg/d for 2 to <6 y.o. & 6 to <12 y.o, & 10 mg/d for 12 y.o. and older (US EPA EFH, 2017). Since toddlers in this assessment are 1 to <4 years old (based on Richardson & Stantec, 2013), a rate of 33 mg/d (average of 40 mg/d + 30 mg/d + 30 mg/d) was calculated and used for on-land activities: playing, walking, and recreational fishing.

For incidental sediment ingestion during wading and swimming, the Health Canada recommendation of 7.7 mg/h (all age categories) for in-water near-shore activities was used (HC, 2017). [*For comparison, sediment ingestion while swimming can be estimated. Based on a report by the St. Lawrence River Institute (Windle and Ridal, 2016), the maximum total suspended sediment (TSS) for St. Lawrence water at Cornwall is approximately 65 mg/L. For children, the recommended values for water ingestion while swimming (US EPA EFH, 2011) are 49 and 120 mL/h (mean and upper percentile, respectively). Put together, estimated rates of sediment ingestion while swimming would be 3.2 mg/h or a very conservative estimate of 7.8 mg/h*.]

3.2.3 Skin Surface Area (SA)

Skin surface area (SA) values for the head, arms, hands, legs, feet, and trunk (torso) were obtained from US EPA's Exposure Factors Handbook, Table 7-2, *Mean Surface Area by Body Part* (US EPA EFH, 2011). To obtain values for the toddler, means of 1 to <2 years, 2 to <3 years, and 3 to <6 years were averaged; for the child, values for 6 to <11 years were used; for the teen, means of 11 to <16 years and 16 to <21 years were averaged; for the adult, means of the adult male and the adult female were used.

The selection of unclothed body parts available for dermal exposure was based on anticipated clothing considerations and behaviour according to the season. The activity with the highest anticipated sediment-exposed skin drives the calculation. See Table 3-3.

Table 3-3: Assumptions of Body Parts Available for Dermal Exposure

Summer	Spring/Fall	Winter
head, hands, arms, legs, feet, torso	head & hands	no dermal exposure
Assumption of bathing trunks worn for swimming. (For scuba diving, this assumption is conservative.)	Limited exposures during these seasons	No exposures during winter months due to unavailability of sediment (from snow or frost cover) and/or temperatures that are unsuitable for bare skin. (See Section 3.2.5 on exposure frequency.)

For dermal exposures to sediment, this assessment is considered conservative for the following reasons:

- It is based on the assumption that all uncovered body parts are in contact with sediment on each day the person is present at the Cornwall waterfront.
- It is based on the assumption that sediment remains adhered to all exposed skin for 24 hours on each day of activity at the Cornwall waterfront.
- The activity with the highest sediment-exposed skin drives the calculations.

3.2.4 Sediment Loading Rate (SL)

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 adherence or sediment loading (SL).

In standard human health risk assessment practice, some assumptions for estimating dermal exposure may be too conservative, e.g., that all potentially exposed body parts are covered with a layer of soil or sediment during every exposure day or event. To prevent compounding conservatism, reasonable values for sediment loading were selected and used.

SL values were obtained from US EPA (2004); as the activities expected on the Cornwall waterfront generally involve lesser contact with sediment, the SL values selected were 0.04 $mg/cm^2/day$ for toddlers and children, based on the geometric mean of daycare children (1 - 6.5 years old) playing indoors and outdoors, and 0.02 $mg/cm^2/day$ for teens and adults, based on the geometric mean of adult groundskeepers.

3.2.5 Exposure Frequency (EF)

In calculating these guidelines, exposure frequency (EF) refers to how often a person may be exposed to sediment along the Cornwall waterfront. Various values for EF were used for the oral and dermal exposure calculations:

3.2.5.1 Oral Exposure During Wading and Swimming:

***$EF_{h/mo}$*:** For oral exposures during wading and swimming, an EF of 22 hours/month is selected based on 1 hour/day and 5 days/week (converted to hours/month: 1 hour/day x 5 days/week x 52 weeks/year ÷ 12 months/year). This value is based on professional judgement – an upper estimate of how often swimming could occur – rather than how often it actually occurs at Cornwall. For comparison, the US EPA Exposure Factors Handbook, Table 16-1 *Recommended Values for Activity Patterns*, recommends an upper estimate of 3 hours/month (181 minutes/month) which is the 95th percentile for swimming (doers only). ("Doers only" is a term used by US EPA indicating the proportion of the population who partakes in the activity. In this case, the EF is 3 h/mo among swimmers only, excluding those who do not partake in swimming.)

***$EF_{mo/y}$*:** Also for oral exposures during wading and swimming, an EF of 3 months/year is selected based on professional judgement. It is assumed that temperatures and weather are suitable for swimming only during the summer months.

3.2.5.2 Oral Exposure During Playing, Walking, and Recreational Fishing:

***$EF_{h/d}$*:** For oral exposures during playing, walking, and recreational fishing, an EF of 1 hour/day was selected for the sediment ingestion exposure pathway for all age categories; US EPA EFH (2011) table 16-1 shows averages of 30 – 83 minutes/day (doers only) for time spent playing on sand/gravel and averages of 30 – 63 minutes/day (doers only) for or playing on dirt, in both cases for ages up to <21 years old; for ages 18 to <65 years old and ≥65 years old, averages were not available but the medians are 0 minutes/day.

***EF$_{d/wk}$*:** For places where children play (including playgrounds and playing fields), RIVM (2007) recommends EFs of 125 days/year for children and 50 days/year for adults; for natural areas, city parks, beaches, and sports fields, RIVM (2007) recommends 25 days/year (children) and 10 days/year (adults). Based on field observations and site-specific information, Weston Solutions Inc., 2005 (on contract for the U.S. Army Corps and US EPA) recommended 30 days/year (central tendency estimate) and 90 days/year (reasonable maximum estimate) for all ages in high-use recreational areas.

According to 2018 climate statistics for Cornwall, Ontario (www.theweathernetwork.com), in June to September it rains an average of 11.5 days/month; therefore, there are 76 *no-rain* days during those months, or 4.36 days/week. Thus, an EF of 5 days/week for summer is selected for all ages as a rate of visitation to the Cornwall waterfront in in this assessment. Based on professional judgement, an EF of 2 days/week is selected for spring and autumn. An EF of 0 days/week is selected for winter based the temperatures making recreation less desirable, the unavailability of sediment during freezing temperatures, and snow cover in the winter season. This assumption is based on an analysis of climate in southern Ontario, as presented in Ontario's rationale for development of soil and groundwater standards (MOE, 2011). The overall frequency of exposure to sediment on the waterfront thus corresponds to 117 days/year.

3.2.5.3 Dermal Exposure During Playing, Walking, Recreational Fishing, Wading, & Swimming:

The EFs of 5 day/week for summer, 2 days/week for spring and fall, and 0 days/week for winter (as described above in section 3.2.5.2) were also used for dermal exposures.

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

Exposures are considered to occur from the toddler stage throughout the receptor's average lifetime of 81 years (Richardson and Stantec, 2013). This assessment uses the age categories, exposure durations, and averaging times that are presented in the 2013 Canadian Exposure Factors Handbook (Richardson and Stantec, 2013).

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. To estimate cancer risks, the calculation of the lifetime average daily dose (LADD) requires the use of an AT for the entire lifespan. A value of 81 years is selected, based on Richardson and Stantec (2013).

3.2.7 Duration of Sediment Adherence to Skin

According to standard risk assessment practice, this assessment 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 activities on the Cornwall waterfront. In some cases, lodged sediment or sediment with strong adherence to the skin may not be eliminated until after a thorough wash.

4.0 Toxicity Assessment – TRVs and RAFs

For each contaminant of concern (COC), toxicity reference values (TRVs) for non-cancer effects and for cancer effects (if available) 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 generally used 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 this assessment, the proportion absorbed into the body upon exposure to the contaminant in sediment. The following sections discuss the TRVs and RAFs selected for each of the COCs considered in this assessment.

4.1 Selection of Toxicity Reference Values (TRVs)

A summary of the TRVs selected is provided in Table 4-1. Appendix A provides discussions on the TRVs selected for each COC.

Table 4-1: Summary of TRVs Selected

COC	Oral Chronic Non-Cancer TRV		Oral Cancer TRV	
	Value (mg/kg/d)	Reference	Value* (per mg/kg/d)	Reference
lead	5×10^{-4}	EFSA, 2010	n/a	
mercury	3×10^{-4}	US EPA IRIS, 1995; HC CSD, 2010 (Part II)	n/a	
B[a]P	3×10^{-4}	US EPA IRIS, 2017	1	US EPA IRIS, 2017

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

4.2 Selection of Relative Absorption Factors (RAFs)

Absorption refers to the proportion of a contaminant that penetrates into the body of the receptor exposed. Absorption may change with the receptor (human or test animal), the contaminated medium, the route of exposure, and in some cases the form of the contaminant. Relative absorption factors (RAFs) are used in a risk assessment to make adjustments for the differences in the efficiency of contaminant absorption in the exposure scenario being assessed as compared to absorption in the toxicity study that forms the basis of the selected TRV. In human health risk assessment (HHRA), *relative absorption* is often used interchangeably with the term *relative bioavailability* (RBA).

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. 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.

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.

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. RAFs for mercury and B[a]P were obtained from the ministry's rationale for the development of soil standards (MOE, 2011). Those for lead were determined as follows:

- *Oral RAF for lead*: The oral lead TRV used in this assessment is from EFSA (2010) for lead in food. The uptake of lead is impeded when it is administered with food rather than a fasting state, and can vary with the type of food (Deshommes *et al*., 2012); thus, lead in soil or sediment could be as bioavailable as lead in food (or even more bioavailable). Therefore, an oral RAF of 1 is selected for lead in sediment in this assessment.

- *Dermal RAF for lead*: Various studies have found dermal absorption of lead to be below 1% (ATSDR, 2007). Since the dermal absorption is approximately 2 orders of magnitude (or more) lower than oral absorption, a dermal RAF of 1% is selected for lead in sediment.

The RAFs selected for use in this assessment are shown in Table 4-2.

Table 4-2: Summary of RAFs Selected

COC	RAF_O (oral relative absorption factor)		RAF_D (dermal relative absorption factor)	
	Value	**Notes / Reference**	**Value**	**Reference**
lead	1	See discussion above	0.01	See discussion above
mercury	0.5	MOE (2011)	0.1	MOE (2011)
benzo[a]pyrene	1	MOE (2011)	0.13	MOE (2011)

5.0 Calculation of Recreational Sediment Guidelines

The risk characterization stage of a typical risk assessment determines whether the estimated COC exposures exceed the identified TRVs. In this assessment, recreational exposure assessment information specific to the Cornwall waterfront and toxicity information specific to each COC are combined with a target hazard quotient (HQ, used with non-cancer TRVs) and an incremental lifetime cancer risk (ILCR, used with cancer TRVs) to determine sediment guidelines for each COC. The derivation approach taken roughly follows that of the MECP brownfields soil standards where background sediment concentrations and estimated daily intakes are not incorporated into the calculations.

The calculations were sub-divided into four parts based on route of exposure (oral and dermal) and endpoint (cancer and non-cancer health effects), described in sections 5.1 and 5.2 below. Parameter abbreviations used in these equations are described in Section 5.3.

The following exposure pathways were included:

- O-PWF: ingestion of sediment from playing, walking, or recreational fishing
- O-SW: ingestion of sediment from swimming or wading
- D-A: dermal exposure to sediment from all/any recreational activities

5.1 Non-Cancer Calculations

For non-cancer, the exposure pathways were represented by Equations 2 – 4. Equations 1 – 4 were combined and rearranged to solve for the concentration of contaminant in sediment ($conc_{sed}$). Target HQ values of 0.2 or 1 and contaminant-specific values for TRV, RAF_D, and RAF_O were applied for lead, mercury, and B[a]P. Season-specific $EF_{d/wk}$ values were used for O-PWF and D-A, and season-specific SA values were used for D-A; thus an EF parameter for weeks/year was not necessary for Equations 2 and 4.

$$HQ \times TRV = exp_{O-PWF} + exp_{O-WS} + exp_{D-A}$$

(Equation 1)

$$exp_{O-PWF} = conc_{sed} \frac{SedIR \times RAF_O \times EF_{h/d} \times EF_{d/wk} \times ED}{BW \times AT \times CF_1 \times CF_2}$$

(Equation 2)

$$exp_{O-WS} = conc_{sed} \frac{SedIR \times RAF_O \times EF_{h/mo} \times EF_{mo/y} \times ED}{BW \times AT \times CF_1 \times CF_3}$$

(Equation 3)

$$exp_{D-A} = conc_{sed} \frac{SA \times SL \times RAF_D \times EF_{d/wk} \times ED}{BW \times AT \times CF_1 \times CF_2}$$

(Equation 4)

5.2 Cancer Calculations

For cancer, the exposure pathways were represented by Equations 6 – 8. Equations 5 – 8 were combined and rearranged to solve for $conc_{sed}$. Incremental Lifetime Cancer Risk (ILCR) values of 10^{-6} and 10^{-5} and contaminant-specific values for CSF, RAF_D, and RAF_O were applied for B[a]P. For each exposure calculation (Equations 6 – 8), age-specific parameters were used to calculate weighted average exposures. Season-specific $EF_{d/wk}$ values were used for O-PWF and D-A, and season-specific SA values were used for D-A; thus an EF parameter for weeks/year was not necessary for Equations 6 and 8.

$$\frac{ILCR}{CSF} = exp_{O-PWF} + exp_{O-WS} + exp_{D-A}$$

(Equation 5)

$$exp_{O-SWF} = conc_{sed} \frac{RAF_O}{AT \times CF_1 \times CF_2}\left[\sum\left(\frac{SedIR \times EF_{h/d} \times EF_{d/wk} \times ED}{BW}\right)_i\right]$$

(Equation 6)

$$exp_{O-WS} = conc_{sed} \frac{RAF_O}{AT \times CF_1 \times CF_3}\left[\sum\left(\frac{SedIR \times EF_{h/mo} \times EF_{mo/y} \times ED}{BW}\right)_i\right]$$

(Equation 7)

$$exp_{D-A} = conc_{sed} \frac{RAF_D}{AT \times CF_1 \times CF_2}\left[\sum\left(\frac{SA \times SL \times EF_{d/wk} \times ED}{BW}\right)_i\right]$$

(Equation 8)

5.3 Parameter Abbreviations for all Calculations

The following abbreviations are used in Equations 1 to 8 (in sections 5.1 and 5.2):

AT = averaging time (years)
BW = body weight (kg_{BW})
CF_1 = unit conversion factor (1,000,000 mg_{sed}/kg_{sed})

CF_2 = unit conversion factor (7 days/week)
CF_3 = unit conversion factor (365 days/year)
$conc_{sed}$ = concentration of contaminant in sediment (mg_{chem}/kg_{sed})
CSF = oral cancer slope factor (or oral cancer toxicity reference value) (per $mg_{chem}/kg_{BW}/d$)
ED = exposure duration (years)
$EF_{d/wk}$ = exposure frequency in days/week (d/wk)
$EF_{h/d}$ = exposure frequency in hours/day (h/d)
$EF_{h/mo}$ = exposure frequency in hours/month (h/mo)
$EF_{mo/y}$ = exposure frequency in months/year (mo/y)
$exp_{O\text{-}PWF}$ = oral exposure from playing, walking, or recreational fishing ($mg_{chem}/kg_{BW}/d$)
$exp_{O\text{-}SW}$ = oral exposure from swimming or wading ($mg_{chem}/kg_{BW}/d$)
$exp_{D\text{-}A}$ = dermal exposure from all/any recreational activities: playing, walking, recreational fishing, swimming, or wading ($mg_{chem}/kg_{BW}/d$)
HQ = hazard quotient (unitless)
ILCR = incremental lifetime cancer risk level (unitless)
RAF_D = dermal relative absorption factor for contaminant in sediment (unitless)
RAF_O = oral relative absorption factor for contaminant in sediment (unitless)
SA = surface area of exposed body parts (cm^2)
SedIR = sediment ingestion rate (mg_{sed}/h)
SL_i = sediment loading rate on skin ($mg_{sed}/cm^2/d$)
TRV = non-cancer toxicity reference value ($mg_{chem}/kg_{BW}/d$)

6.0 Site-Specific Sediment Guidelines and their Application

6.1 Recreational Site-Specific Sediment Guidelines for Cornwall

Table 6-1 shows the results of the non-cancer and cancer calculations. Note that since reliable cancer TRVs do not exist for lead and mercury, cancer calculations were not conducted for these contaminants.

Table 6-1: Results of Calculations

Contaminant	Calculated Sediment Concentrations (ppm)			
	Non-Cancer		Cancer	
	HQ=0.2	HQ=1	ILCR=10^{-6}	ILCR=10^{-5}
lead	123	613	nc	nc
mercury	83	415	nc	nc
B[a]P	49	247	3	28

* nc = not calculated

Results from Table 6-1 were rounded and applied accordingly to determine the recreational guidelines for lead, mercury, and B[a]P in sediment along the Cornwall waterfront. For B[a]P, there are both cancer and non-cancer calculations, so the more stringent of the two calculations is used. The sediment guidelines are presented in Table 6-2. It is assumed that since exceedances of other contaminants were not excessive, risks from exposures to these contaminants would be resolved through risk management decisions made for lead, mercury, and B[a]P. Guidance on interpreting the sediment guidelines and recommended risk management actions are also presented in Table 6-2.

Table 6-2: Recreational Sediment Guidelines for the Cornwall Waterfront

Sediment Guidelines (ppm)			**Risk Management (RM)**	
Lead	**Mercury**	**B[a]P#**	**Level***	**Interpretation of Risk**
≤120	≤80	≤3	RM0	The concentrations are acceptable. Risks from long-term recreational exposures are negligible. No action necessary.
>120 to ≤610	>80 to ≤410	>3 to ≤30	RM1	The concentrations exceed conservative benchmarks. The potential for unacceptable exposures and associated risks cannot be discounted but are unlikely. Possible risk management measures include communication or signage.
>610	>410	>30	RM2	Risks from long-term recreational exposures are considered to be elevated. Measures to block exposure pathways may be warranted.

The B[a]P guidelines are for total PAHs summed as B[a]P-equivalents.

* RM0, RM1, and RM2 refer to the level of *risk management* recommendations.

For comparison, note that the human health-based *soil* criteria for ingestion and dermal contact with *residential soil* at brownfield sites (used in calculating the ministry's soil standards) are 120 ppm for lead, 9.8 ppm for mercury, and 0.078 ppm for B[a]P (MOE, 2011); the Ontario Typical Range background *soil* concentrations for these contaminants are 120 ppm for lead, 0.27 ppm for mercury, and 0.3 ppm for B[a]P (MOE, 2011).

Please note that the current sediment guidelines are specific to risks associated with *recreational* use of the Cornwall waterfront and are not reflective of any other risks from living in the Cornwall area. Furthermore, the purpose of these guidelines is to inform risk management of the recreational areas along the Cornwall waterfront; therefore the guidelines do not account fish consumption.

The sensitivity analysis in Appendix B identifies the parameters which are the most significant at driving the final values of the sediment guidelines. Appendix C discusses the uncertainties and limitations associated with the assumptions used in this assessment.

The conclusions and recommendations presented here are based on current science and on the site description at the time of preparation of this report. New sediment data may indicate contaminant concentrations higher than those used in this assessment or the presence of contaminants not evaluated at all in this assessment. In either of these cases, re-assessment may be warranted. Furthermore, in order to keep up-to-date with current science, revisiting the current approach and input parameter values is recommended for the future.

Note that nearshore area of the Cornwall waterfront along the St. Lawrence River is made up of rock, gravel, or macrophyte beds. **Since there are no sediments within the first few metres of shore, it means the exposure calculations are over-assumptions, making these sediment guidelines conservative when applied to recreational use of the Cornwall waterfront.** This should be taken into consideration when interpreting sediment sampling data.

6.2 Application of Cornwall Recreational Sediment Guidelines

To ensure that the guidelines are applied properly, the following process is recommended:

1) For B[a]P and other PAHs, PAH concentrations should be summed as B[a]P-equivalents ($B[a]P_{eq}$) and compared to the sediment guidelines for B[a]P. Expressing the concentration of PAHs in $B[a]P_{eq}$ requires toxic equivalency factors (TEFs) for *human health*. Table 6-3 displays TEFs from Kalberlah *et al*., 1995 (as cited in WHO EHC, 1998) which are used in the ministry's Brownfields program. However, it is acceptable to use any TEF scheme that is generally accepted in human health risk assessment practice, including the TEFs used by Health Canada or by the Canadian Council of Ministers of the Environment (CCME) and the sources from which those TEFs were obtained.

Table 6-3: Toxic Equivalency Factors (TEFs) Relevant for Human Health

PAH	TEF	PAH	TEF	PAH	TEF
acenaphthene	0.001	benzo[b]fluoranthene	0.1	dibenz[a,h]anthracene	1
acenaphthylene	0.01	benzo[g,h,i]perylene	0.01	fluoranthene	0.01
anthracene	0.01	benzo[k]fluoranthene	0.1	indeno[1,1,3-cd]pyrene	0.1
benz[a]anthracene	0.1	chrysene	0.01	pyrene	0.001
benzo[a]pyrene	1				

2) As with the application of any guidelines or standards, an understanding of proper methodologies for sampling and analyses is required. Based on data quality, it is preferential

to use **95% UCLM** (95% upper confidence limit of the mean) sediment concentrations to compare to the sediment guidelines shown in Table 6-2. However, other statistical values may be considered. In addition, sampling should be reflective of the exposure unit (the areas throughout which a receptor moves and encounters the sediment for the duration of the exposure) and hotspots should be addressed appropriately.

7.0 References

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.

deBarros C, Anderson J. 2010. The 2005 Cornwall Sediment Strategy. Ontario Ministry of the Environment and Environment Canada. 123 pp.

EFSA. 2010. Scientific Opinion on Lead in Food. EFSA Panel on Contaminants in the Food Chain (CONTAM), European Food Safety Authority. Parma, Italy. EFSA Journal 8:1570 (147 pp.).

HC. 2017. Supplemental Guidance on Human Health Risk Assessment of Contaminated Sediments: Direct Contact Pathway. Contaminated Sites Division, Health Canada. ISBN 978-0-660-07989-9. March 2017.

HC CSD. 2010, Part II. 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 CSD. 2010, Part V. Federal Contaminated Site Risk Assessment in Canada, Part II: Guidance on Human Health Detailed Quantitative Risk Assessment for Chemicals ($DQRA_{CHEM}$). Prepared by: Contaminated Sites Division, Safe Environments Directorate. Health Canada. September 2010.

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.

Milani D, Grapentine L. 2015. Benthic Conditions in the St. Lawrence River at Cornwall Area of Concern 2012 and Trends from 1997 to 2012. Environment Canada, Water Science and Technology Directorate. Burlington, ON, Canada. June 3, 2015.

MOE. 2011. 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.

Pelletier, M. 2010. Sediment Quality Monitoring in Lake Saint-François. Scientific and Technical Report. Environment Canada – Science and Technology Branch. Quebec Water Quality Monitoring and Surveillance. December 2010. 66 pages.

Razavi R, Windle M, Savard L, Ridal J. 2015. Spatial Analysis of Mercury Concentrations in Shoreline Sediments of the St. Lawrence River (Cornwall) Area of Concern. Report prepared for the Ontario Ministry of the Environment and Climate Change, Kingston, ON, Canada.

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

Richardson GM and Stantec Consulting Ltd. 2013. 2013 Canadian Exposure Factors Handbook. Toxicology Centre, University of Saskatchewan, Saskatoon, SK, Canada.

Richman LA. 1996. St. Lawrence River Remedial Action Plan Technical Report No. 7: St. Lawrence River Sediment Assessment, 1994. Cornwall, ON, Canada. St. Lawrence River RAP. February 1996.

RIVM. 2007. CSOIL 2000: An Exposure Model for Human Risk Assessment of Soil Contamination – A Model Description. E Brand, PF Otte, JPA Lijzen. RIVM Report No. 711701054.

TCEQ. 2007. Compounds for which Calculation of a Human Health PCL is Not Required. Texas Commission on Environmental Quality. March 30, 2007.

US EPA. 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Office of Superfund Remediation and Technology Innovation, U.S. Environmental Protection Agency. Washington, DC, USA. EPA/540/R/99/005. July 2004.

US EPA. 2017. Regional Screening Level (RSL) Residential Soil Table (TR = 1E-6, HQ=0.1). U.S. Environmental Protection Agency, Region III. June 2017.

US EPA EFH. 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 EFH. 2017. Update for Chapter 5 of the Exposure Factors Handbook – Soil and Dust Ingestion. National Center for Environmental Assessment, U.S. Environmental Protection Agency. Washington, DC, USA. EPA/600/R-17/38F. September 2017.

US EPA IRIS. 1995. Mercuric Chloride (CASRN 7487-94-7). Integrated Risk Information System. U.S. Environmental Protection Agency. Washington, DC, USA. May 1995.

US EPA IRIS. 2017. Toxicological Review of Benzo[a]pyrene (CASRN 50-32-8). Integrated Risk Information System (IRIS). U.S. Environmental Protection Agency. Washington, DC. EPA/635/R-17/003Fa. January 2017.

USGS. 2005. Major- and Trace-Element Concentrations in Soils from Two Continental-Scale Transects of the United States and Canada. U.S. Geological Survey. Reston, Virginia, USA. Report # 2005-1253.

Weston Solutions Inc. 2005. Human Health Risk Assessment – General Electric / Housatonic River Site, Pittsfield, Massachusetts – Rest of River. Volume IIIA: Appendix B: Phase 2 Direct Contact Risk Assessment. Prepared for: U.S. Army Corps of Engineers, New England District, Concord, MA and U.S. Environmental Protection Agency, New England Region, Boston, MA. Prepared by: Weston Solutions, Inc., West Chester, PA. Feb. 2005.

WHO EHC. 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.

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.

Windle M, Ridal J. 2016. Spatial Analysis of Mercury Concentrations and Other Contaminants in Nearshore Sediments of the Cornwall Area of Concern (AOC) in 2015 - Draft Report for Environment and Climate Change Canada and the Ontario Ministry of the Environment and Climate Change in Support of the St. Lawrence River (Cornwall) Remedial Action Plan. St. Lawrence Institute of Environmental Sciences. Cornwall, ON, Canada. Aug. 26, 2016.

Appendix A: Selection of Toxicity Reference Values (TRVs)

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	Comp	
Cal EPA DW 2010	1.7E-03	5	LOAEL	10	10	10	10			3000	• Knuckles *et al.*, 2001 • Fisher 344 rats, 5/sex/dose • Doses: 5, 50, 100 mg/kg/d BaP 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

Agency	TRV (mg/kg/d)	Point of Departure		Uncertainty Factors							Notes
		Dose (mg/kg/d)	Basis	A	H	L	S	D	X	Comp	
MDH 2012	2E-04	0.02	NOAEL	10	10					100	• Chen *et al*., 2012 • Neonatal Sprague-Dawley rats, 10/sex/dose, in peanut oil solution • Doses: 0.02, 0.2, 2 mg/kg/d BaP; by gavage on post-natal days 5-11 • Effects were dose-dependent & persisted into adolescence & adulthood • Some effects were noted even at lowest dose 0.02 mg/kg/d MDH (2012): • Critical effect: neurotoxicity (significant alterations in performance in tests designed to evaluate learning & locomotor abilities)
HC DW 2016	6.7E-05	0.02	NOAEL	10	10			3		300	HC DW (2016): • Critical effect: neurodevelopmental toxicity • UF_D of 3: database deficiencies for non-cancer effects given studies on neurological & neurodevelopmental effects; data on low-dose effects is limited; limited data to support any non-cancer MOAs
US EPA IRIS 2017	3E-04	0.092	$BMDL_{1sd}$	10	10			3		300	US EPA IRIS (2017): • Critical effect: Neurobehavioural changes (altered responses in 3 behavioural tests) • UF_D of 3: lack of a standard multigenerational study or extended 1-generation study that includes exposure from premating through lactation • Although 2 other TRVs were derived (reproductive & immunological critical effects), developmental toxicity was chosen as the basis for the overall TRV.

Selection: 3 x 10^{-4} mg/kg/d: US EPA IRIS (2017)

Rationale: 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 large magnitude of uncertainty. The 3 remaining TRVs were based on the same study with a more sensitive endpoint than the study used by Cal EPA. Of these, US EPA IRIS (2017) used BMD modelling which is considered to be a more robust method of selecting a POD.

Oral Slope Factor

Agency	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Extrapolation Method	Notes
RIVM 2001b	0.2	Linear non-threshold	• RIVM, 2001a • Wistar rats, 52/sex/dose, 5 d/wk, for 2 y • Doses: 3, 10, 30 $mg/kg_{BW}/d$ BaP 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	11.5	Linearized multistage procedure	• Neal and Rigdon, 1967 • CFW-Swiss Mice; 17–180 d old; 9–73 mice/dose + 289 controls (unspecified gender combinations) • Doses: 1, 10, 20, 30, 40, 45, 50, 100, 250 ppm BaP in diet; treatment time from 1–197 d • Critical effect: gastric tract tumours (papillomas & squamous cell carcinomas) in M & F mice • Calculated CSF of 11.5 per mg/kg/d rounded to 12 per mg/kg/d
Cal EPA DW 2010	2.9	Multi-stage Weibull-in-time model	• 5 slopes calculated: 1 from mouse data (Culp *et al*., 1998) & 4 from rat data (RIVM, 2001a) Culp *et al.*, 1998 • Female B6C3F1 mice, 48/dose, for 2 y • Authors used only females because of their low spontaneous liver tumour incidence. (Also, female mice are more sensitive than males because of lower ability to conjugate BaP reactive metabolites.) • Doses: 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} RIVM, 2001a (same study as RIVM, 2001b) • Critical effect: increases in liver tumours & combined tumours of oral cavity & forestomach • Slopes 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 • The most health-protective slope 1.7 $(mg/kg/d)^{-1}$ from mouse data was selected; multiplied by Age Sensitivity Factor of 1.7 to account for higher sensitivity of children to carcinogens = 2.9 $(mg/kg/d)^{-1}$
HC CSD 2010	2.3	Linear extrapolation & surface area correction	• Neal and Rigdon, 1967 (same study as Cal EPA ATH, 2009) • Critical effect: gastric tumours (mostly squamous cell papillomas, with a few carcinomas)

Agency	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Extrapolation Method	Notes
NZ MfE 2011	2.08	various	• 4 datasets: Neal & Rigdon ,1967 (same as Cal EPA ATH, 2009); Brune *et al*., 1981; Culp *et al*., 1998 (same as Cal EPA DW, 2010); RIVM, 2001a (same as RIVM, 2001b) Brune *et al*., 1981: • Sprague-Dawley rats (male & female; 32/sex/dose) • Doses: 0.016, 0.049, 0.107 mg/kg/d via diet or gavage; for 2 y • Treated until moribund or dead • Geometric mean of several $BMDL_{10}$ estimates from 4 datasets, with allometric cross-species scaling • Critical effects: forestomach tumours in mice and various tumours in rats
HC DW 2016	1.3	Log logistic	• Culp *et al*., 1998; Wester *et al*., 2012; Moffat *et al*., 2015 (review article) • 2 studies selected based on oral exposure, chronic duration, multiple doses, & sufficient number of animals per group • In both studies, forestomach was determined to be most sensitive tissue. (Forestomach data is relevant because MOA is genotoxic, induction of tumours at multiple sites, in various species, & both sexes.) Culp *et al*., 1998 (same study as Cal EPA DW, 2010) • Critical effect: forestomach tumours in female mice • $BMDL_{10}$ = 0.5389 mg/kg/d Wester *et al*., 2012 • Male & female Wistar rats • Doses: 3, 10, 30 mg/kg/d by gavage, 5 d/wk, for 104 weeks • Critical effect: forestomach tumours in male and female rats • $BMDL_{10}$ = 0.8 mg/kg/d • 0.5389 mg/kg/d (Culp *et al*., 1998; Moffat *et al*., 2015) is lowest $BMDL_{10}$ identified for forestomach tumours • mouse POD of 0.5389 mg/kg/d (Moffat *et al*., 2015) adjusted with allometric scaling $(0.03\ kg \div 70\ kg)^{1/4}$ to obtain human equivalent dose: 0.07758 mg/kg/d • CSF = 0.1 ÷ 0.07758 mg/kg/d (where 0.1 is the 10% benchmark response)

Agency	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Extrapolation Method	Notes
US EPA IRIS 2017	1	Linear extrapolation from $BMDL_{10}$ using multistage Weibull model	• 3 CSFs calculated: 1 from mouse data (Beland & Culp, 1998), 2 from rat data (RIVM, 2001a) Beland & Culp, 1998 (same study as Culp *et al*., 1998 used by Cal EPA DW, 2010 but reported differently) • 5, 25, 100 ppm BaP in diet ≈ 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 $(mg/kg/d)^{-1}$ RIVM, 2001a (same study as RIVM, 2001b) • Critical effect: combined tumours of forestomach, oral cavity, liver, jejunum/duodenum, kidney, skin, & mammary glands; human equivalent CSF = 0.3 $(mg/kg/d)^{-1}$ • Selected most health protective CSF derived: 1 $(mg/kg/d)^{-1}$ from mouse data (Beland & Culp *et al*., 1998)

Selection: 1 $(mg/kg/d)^{-1}$: US EPA IRIS (2017)

Rationale: The studies by 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, 2017). 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 (Cal EPA ATH, 2009; HC CSD, 2010; NZ MfE, 2011) were not considered further.

The CSFs which are derived from several studies are preferable to those relying on a single study. The CSF derivations by HC DW (2016) and US EPA IRIS (2017) are roughly equal in robustness and in their final values. Of the two, the CSF by US EPA IRIS (2017) is selected.

References

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, Schmähl 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. Sacramento, California, USA. 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.

HC DW. 2016. Guidelines for Canadian Drinking Water Quality – Guideline Technical Document – Benzo[a]pyrene. Prepared by the Federal-Provincial-Territorial Committee on Drinking Water. Health Canada. Ottawa, Ontario, Canada. January 2016.

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.

MDH. 2012. Toxicological Summary for Benzo[a]pyrene CAS: 50-32-8. 2012 Health Based Guidance for Groundwater. Health Risk Assessment Unit, Environmental Health Division, Minnesota Department of Health. March 2012.

Moffat I, Chepelev NL, Labib S, Bourdon-Lacombe J, Kuo B, Buick JK, Lemieux F, Williams A, Halappanavar S, Malik AI, Luijten M, Aubrecht J, Hyduke DR, Fornace AJ Jr, Swartz CD, Recio L, Yauk CL. 2015. Comparison of toxicogenomics and traditional approaches to inform mode of action and points of departure in human health risk assessment of benzo[a]pyrene in drinking water. Crit Rev Toxicol 45:1–43.

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

NZ MfE. 2011. Toxicological Intake Values for Priority Contaminants in Soil. New Zealand Ministry for the Environment. Wellington, New Zealand. ISBN: 978-0-478-37238-0.

RIVM. 2001a. 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). Kroese ED, Muller JJA, Mohn GR, Dortant PM, Wester PW. Bilthoven, The Netherlands. RIVM Report No. 6658603 010. (Draft 1999, Final 2001.)

RIVM. 2001b. 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.

US EPA IRIS. 2017. Toxicological Review of Benzo[a]pyrene (CASRN 50-32-8). Integrated Risk Information System, U.S. Environmental Protection Agency. Washington, DC, USA. EPA/635/R-17/003Fa. August 2013.

Wester PW, Muller JJA, Slob W, Mohn GR, Dortant PM, Kroese ED. 2012. Carcinogenic activity of benzo[a]pyrene in a 2 year oral study in Wistar rats. Food Chem Toxicol 50:927–935.

Lead (Pb)
CAS # 7439-92-1

Oral Chronic Non-Cancer

Agency or Group (medium)	TRV (mg/kg/d) = A÷[BxC]	A) Point of Departure	B) Relationship between IQ & BLL	C) Relationship betweenBLL & Intake Rate	Notes

Cal EPA DW 2009 (DW)	2.86 µg/d ≈ 1.9E-04	-1 IQ point	-1 IQ point per BLL increase of 1 µg/dL	0.35 µg/dL BLL per 1.0 µg/d intake from DW ≈ 5.9 µg/dL per µg/kg/d	• Lanphear *et al*., 2005
					• 1333 children aged 58 mo – 5 y, exposed via any/all media
					• Critical effect: Neurotoxicity (decrease in IQ) in children
					• Relationship between IQ (intelligence quotient) & BLL (blood lead level) is non-linear with greater relative change in IQ at lower BLLs
					• No LOAEL identified
					• POD of -1 IQ point based on CDC (1991) stating decrease of a few IQ points may be very significant on population level for increased need for remedial education, & based on Lanphear *et al*. (2005) and Carlisle & Dowling (2006) demonstrating decreased IQ can occur at BLLs much <10 µg/dL
					  ○ Carlisle & Dowling (2006) used 1 IQ point as indicator of “minimal adverse effects on cognitive function”
					• IQ-BLL slope determined with linear models described by Lanphear *et al*. (2005) & reanalysis by Hornung (2005; personal communication to Cal EPA): $UCL_{97.5}$ on slope = -0.9 IQ points per µg/dL (rounded to -1)
					  ○ Model estimates mean slope of -0.47 IQ points per µg/dL
					  ○ Used $UCL_{97.5}$ to account for variability & uncertainty in data, to be reasonably certain that result is not underestimate of true slope
					• BLL-intake slope determined using US EPA’s IEUBK (Integrated Exposure Uptake Biokinetic Model for Lead in Children) for children 12–24 mo. old
					  ○ Each 1.0 µg/d increment in DW intake leads to 0.35 µg/dL BLL increase
					• To express intake rates as “per kg_{BW}”, toddler BW of 15.3 kg (Richardson & Stantec, 2013) can be applied to Cal EPA’s values

Agency or Group (medium)	TRV (mg/kg/d) = A÷[BxC]	A) Point of Departure	B) Relationship between IQ & BLL	C) Relationship betweenBLL & Intake Rate	Notes
EFSA 2010 (diet)	5.0E-04	-1 IQ point	-1 IQ point per BLL increase of 1.2 µg/dL	1.2 µg/dL BLL per 0.5 µg/kg/d intake from diet ≈ 2.4 µg/dL per µg/kg/d	• Lanphear *et al*., 2005 (Same key study as Cal EPA DW, 2009) • Same critical effect as Cal EPA DW, 2009 • POD of -1 IQ point based on 1% change in full scale IQ score: ○ 1 IQ point shift in distribution would impact population's socioeconomic status & productivity: -1 IQ point α 4.5% increased risk of failing to graduate high school (Schwartz, 1994) and α 2% decrease in worker productivity (Grosse *et al*., 2002) • IQ-BLL slope determined with Lanphear *et al*. (2005) data using piecewise linear model since it provided more certainty in estimates of $BMDL_{01}$ than other models ○ $BMDL_{01}$ = 1.2 µg/dL for developmental neurotoxicity BMR of -1 IQ point • BLL-intake slope determined using IEUBK model for children 0–7 y old ○ Assuming negligible exposure from air, soil, & dust, 1.2 µg/dL corresponds to dietary Pb intake of 0.5 µg/kg/d (based on 20 kg child)
WHO JECFA 2011 (diet)	3.2E-04	-0.5 IQ points	-1 IQ point per BLL increase of 2.1 µg/dL	0.16 µg/dL BLL per 1.0 µg/d intake from diet ≈ 3.2 µg/dL per µg/kg/d	• Lanphear *et al*., 2005 (Same key study as Cal EPA DW, 2009) • Same critical effect as Cal EPA DW, 2009 • POD: A population decrease of 0.5 IQ points is considered negligible, whereas a population decrease of 3 IQ points is deemed to be a concern • IQ-BLL slope: Bilinear model provided best fit of Lanphear *et al*. (2005) data ○ BLL of 2.1 µg/dL is associated with a decrease in 1 IQ point • BLL-intake slope from US EPA (1986) analysis of Ryu *et al*. (1983) ○ 0.16 µg/dL BLL corresponds to dietary intake of 1.0 µg/d ○ To express intake rates as "per kg_{BW}", WHO applied toddler BW of 20 kg
WHO JECFA 2011 (diet); NZ MfE 2011 (soil)	1.9E-03	-3 IQ points			
Wilson & Richardson 2013 (soil)	6E-04	-1 IQ point	-1 IQ point per BLL increase of 2.1 µg/dL	0.16 µg/dL BLL per 1.0 µg/d intake from diet ≈ 3.2 µg/dL per µg/kg/d	• Lanphear *et al*., 2005 (Same key study as Cal EPA DW, 2009) • Same critical effect as Cal EPA DW, 2009 • POD: 1 IQ point decrement is identified as the "essentially negligible effect level" in infants, toddlers, & children. ○ Noted that Cal EPA (2009) selected -1 IQ point for risk assessment of Pb in soil & for risk management • IQ-BLL slope adopted from WHO JECFA (2011) • BLL-intake slope adopted from WHO JECFA (2011)

Selection: 5 x 10^{-4} mg/kg/d: EFSA (2010)

Rationale: All the recent oral chronic non-cancer TRVs for Pb are based on a formula where the *A*, the point of departure (a decrease in IQ), is divided by the product of *B*, the relationship between IQ and blood Pb level (BLL), and *C*, the relationship between the BLL and intake rate. Regarding A, the decrease in IQ for children exposed to Pb, most TRV derivations use a decrease in 1 IQ point as the point of departure. WHO JECFA (2011) proposed two separate TRV calculations where a decrease in 0.5 IQ points is considered negligible and a decrease in 3 IQ points is considered to be a concern. Because the point of departure is based on a change rather than on absence/presence of an effect, it is difficult to draw a firm line. However, a decrease in 1 IQ point is reasonable; although it might not be measurable or significant for an individual, on a population basis it may be.

Regarding B, the relationship between an increase in BLL and a decrease in IQ, in studies with children with BLLs < 10 µg/dL, a reasonable relationship is in the range of -1 IQ point per 1 µg/dL. Both Cal EPA DW (2009) and EFSA (2010) use values in this range.

Regarding C, the relationship between intake rate and BLL, various studies indicate a relationship between 0.93 and 3.1 µg/dL per µg/kg/d. EFSA (2010) uses a relationship of 2.4 µg/dL per µg/kg/d and WHO JECFA (2011) uses 3.2 µg/dL per µg/kg/d. Cal EPA uses a higher estimate that may be more appropriate for drinking water. Regarding bioavailability, soil is likely to be more similar to diet than to water. Therefore, EFSA and WHO have the more relevant values for the relationship between intake and BLL. Of the three parameters used to derive oral chronic TRVs, EFSA (2010) has incorporated values that are most appropriate and relevant for Ontario soils.

References

Cal EPA. 2009. Revised California Human Health Screening Levels for Lead. Integrated Risk Assessment Branch, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. September 2009.

Cal EPA DW. 2009. Public Health Goals for Chemicals in Drinking Water – Lead. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. April 2009.

Carlisle JC, Dowling K. 2006. Child-specific Health Guidance for Lead. Presented at the Annual Meeting of the Society of Toxicology, March 2006, San Diego, California, USA. Toxicologist Abstract 2191:449.

CDC. 1991. Preventing Lead Poisonings in Young Children – A Statement by the Centers for Disease Control and Prevention. Centers for Disease Control and Prevention, Public Health Service, U.S. Department of Health and Human Services. Atlanta, GA, USA. October 1991.

EFSA. 2010. Scientific Opinion – Scientific Opinion on Lead in Food. EFSA Panel on Contaminants in the Food Chain (CONTAM), European Food Safety Authority. Parma, Italy. EFSA Journal 8:1570 (147 pp).

Grosse SD, Matte TD, Schwartz J, Jackson RJ. 2002. Economic Gains Resulting from the Reduction in Children's Exposure to Lead in the United States. Environ Health Perspect 110:563-569.

Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, Canfield RL, Dietrich KN, Bornschein R, Greene T, Rothenberg SJ, Needleman HL, Schnaas L, Wasserman G, Graziano J, Roberts R. 2005. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. Environ Health Perspect 113:894-899.

NZ MfE. 2011. Toxicological Intake Values for Priority Contaminants in Soil. New Zealand Ministry for the Environment. Wellington, New Zealand.

Richardson GM and Stantec Consulting Ltd. 2013. 2013 Canadian Exposure Factors Handbook. Toxicology Centre, University of Saskatchewan, Saskatoon, SK, Canada. Available at: www.usask.ca/toxicology.

Ryu JE, Ziegler EE, Nelson SE, Fomon SJ. 1983. Dietary Intake of Lead and Blood Lead Concentration in Early Infancy. Am J Dis Child 137:886-891.

Schwartz J. 1994. Societal Benefits of Reducing Lead Exposure. Environ Res 66:105-124.

US EPA. 1986. Air Quality Criteria for Lead. Environmental Criteria and Assessment Office, United States Environmental Protection Agency. Washington, DC, USA. EPA600883028F.

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

WHO JECFA. 2011. Safety Evaluation of Certain Food Additives and Contaminants. WHO Food Additives Series: 64. Prepared by the Seventy-Third Meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). Food and Agriculture Organization of the United Nations (FAO) and World Health Organization (WHO). Geneva, Switzerland. ISBN 978 924 166064 8.

Mercury (Hg)
CAS # (various)

Oral Chronic Non-Cancer

<table>
<tr><th rowspan="2">Agency</th><th rowspan="2">TRV (mg/kg/d)</th><th colspan="2">Point of Departure</th><th colspan="7">Uncertainty Factors</th><th rowspan="2">Notes</th></tr>
<tr><th>Dose (mg/kg/d)</th><th>Basis</th><th>A</th><th>H</th><th>L</th><th>S</th><th>D</th><th>X</th><th>Comp</th></tr>
<tr><td>US EPA IRIS 1995;
HC CSD 2010</td><td>3E-04</td><td>0.226;
0.317;
0.633</td><td>LOAEL</td><td colspan="2">10</td><td>10</td><td>10</td><td></td><td></td><td>1000</td><td>Druet et al., 1978
• Rats, 7-9 weeks old, subcutaneous injections of $HgCl_2$ 3x/wk for up to 8 wks
• Doses: 50, 100, 250, 500, 1000, 2000 μg/kg_{BW}/wk
• Critical effect: Fixation of IgG in kidneys at ≥50 μg/kg/wk ≈ 0.226 mg_{Hg}/kg/d
Bernaudin et al.,1981
• Rats fed $HgCl_2$ at 3 mg/kg/wk (≈ 2.22 mg/kg_{Hg}/d) by gavage, for 60 d
• Critical effects: Deposition of IgG in renal glomeruli after 15 d in 80% of exposed rats (After 60 d, 100% of rats had IgG deposition in renal glomeruli & arteries); LOAEL = 0.317 mg/kg/d
Andres, 1984
• Rats fed $HgCl_2$ 3 mg/kg (≈ 2.22 mg_{Hg}/kg/d) by gavage 2x/wk, for 60 d
• Critical effects: IgG deposits in renal glomeruli; morphological lesions of ileum & colon with abnormal IgA & IgG deposits, hair- & weight-loss after 2-3 wks, mortality in 2 rats after 30-40 d; LOAEL = 0.633 mg/kg/d</td></tr>
<tr><td>Cal EPA DW 1999</td><td>1.6E-04</td><td>0.16</td><td>$NOAEL_{ADJ}$</td><td>10</td><td>10</td><td></td><td>10</td><td></td><td></td><td>1000</td><td rowspan="3">• NTP, 1993
• Rats, $HgCl_2$ by gavage for 6 mo.
• Doses: 0.23, 0.462. 0.739, 1.847, 3.694 mg_{Hg}/kg/d
• Critical effects: increased kidney weight [also decreased BW gains for Cal EPA DW] & at ≥0.462 mg_{Hg}/kg/d
• [$NOAEL_{ADJ}$ = NOAEL x 5/7 (d/wk, adjusted to continuous exposure)]</td></tr>
<tr><td>RIVM 2001</td><td>2E-03</td><td>0.23</td><td>NOAEL</td><td>10</td><td>10</td><td></td><td></td><td></td><td></td><td>100</td></tr>
<tr><td>ATSDR 1999 (intermediate);
WHO CICAD 2003;
UK EA 2009;
WHO DW 2011</td><td>2E-03</td><td>0.16</td><td>$NOAEL_{ADJ}$</td><td>10</td><td>10</td><td></td><td></td><td></td><td></td><td>100</td></tr>
</table>

Selection: 3E-04 mg/kg/d: US EPA IRIS (1995) and HC CSD (2010)

Rationale: The key studies used by US EPA IRIS (1995) and HC CSD (2010) identified autoimmune glomerulonephritis as a critical and highly specific Hg-induced endpoint in the Brown Norway rat, which is an appropriately sensitive animal model to test kidney toxicity of Hg. The critical endpoint of autoimmune glomerulonephritis in these studies is considered to be a more sensitive marker of toxicity than increments in body weight and kidney weight from the NTP (1993) study used in the other TRVs derivations. For these reasons, the TRV derived by US EPA IRIS (1995) was selected.

References

Andres P. 1984. Brief Communications: IgA-IgG disease in the Intestine of Brown-Norway Rats Ingesting Mercuric Chloride. *Clin Immunol Immunopathol* 30:488-494.

ATSDR. 1999. Toxicological Profile for Mercury. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. March 1999.

Bernaudin JF, Druet E, Druet P, Masse R. 1981. Inhalation or Ingestion of Organic or Inorganic Mercurials Produces Auto-Immune Disease in Rats. *Clin Immunol Immunopathol* 20:129-135.

Cal EPA DW. 1999. Public Health Goal for Inorganic Mercury in Drinking Water. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. February 1999.

Druet P, Druet E, Potdvin F, Sapin C. 1978. Immune Type Glomerular Nephritis Induced by $HgCl_2$ in the Brown Norway Rat. *Ann Immunol (Paris)* 129c:777-792.

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.

NTP. 1993. Toxicology and Carcinogenesis Studies of Mercuric Chloride (CAS no. 7487-94) in F344/N Rats and B6C3F1 Mice (Gavage Studies). Research Triangle Park, NC: U.S. Department of Health and Human Services, National Institutes of Health; NIH publication no. 91-3139. National Toxicology Program technical report no. 408. NTIS number: PB94-10649/XAB.

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.

UK EA. 2009. Contaminants in Soil: Updated Collation of Toxicological Data and Intake Values for Humans – Mercury. Better Regulation Science Program. United Kingdom Environment Agency. Science Report: SC050021. Bristol, United Kingdom. March 2009.

US EPA IRIS 1995. Mercuric Chloride (CASRN 7487-94-7). Integrated Risk Information System. U.S. Environmental Protection Agency. Washington, DC, USA. May 1995.

WHO CICAD. 2003. Elemental Mercury and Inorganic Mercury Compounds: Human Health Aspects - Concise International Chemical Assessment Document 50. World Health Organization. Geneva, Switzerland.

WHO DW. 2011. Guidelines for Drinking-Water Quality, Fourth Edition. World Health Organization. Geneva, Switzerland. ISBN 978 92 4 154815 1.

Appendix B: Sensitivity Analysis

Several parameters are used in the model calculations to develop the recreational site-specific guidelines for the Cornwall waterfront. A sensitivity analysis can determine which parameters are the most significant at driving the final values of the guidelines. Table B-1 shows the results of the sensitivity analysis for the mercury RM0 sediment guideline.

A 20% change to input parameters involved in all three exposure pathways evaluated leads to a 20% change in the calculated sediment concentration; these are the toxicity reference value and body weight. A 20% change to input parameters involved only in oral exposure via on-land activities (shoreline play, walking, or recreational fishing) results in an 8% change in the calculated sediment concentration. A 20% change to input parameters involved only in dermal exposures also creates an 8% change. A 20% change in EF (days/week), which is involved in both the dermal calculations and the oral on-land calculations, results in a 17% change. Conversely, a 20% change to input parameters only involved only in oral exposure via in-water activities (wading/swimming) leads to a 1% change in the calculated sediment concentration. **The most sensitive parameters are the TRV, body weight, and exposure frequency (days/week) because they are used in calculating exposures for both the on-land and in-water activities. All parameters involved in on-land activities are sensitive, whereas parameters involved in only in-water activities are not sensitive.**

This analysis indicates that for mercury, parameters affecting oral exposures on land (during shoreline play, walking, or recreational fishing) and dermal exposures have roughly equal impacts on the calculated sediment concentration. In contrast, **the oral pathway of exposure via wading and swimming has a negligible impact on the final sediment concentration**.

Table B-1: Sensitivity of Mercury Sediment Guideline (HQ 0.2) to Changes in Parameter Value

<table>
<tr><th colspan="2" rowspan="2">Parameter*</th><th rowspan="2">Parameter Initial Value</th><th colspan="3">Exposure Pathway</th><th colspan="2">Effect of 20% Parameter Change on Mercury Sediment Concentration of 83 ppm</th></tr>
<tr><th>Oral via on-land Activities</th><th>Oral via in-water Activities</th><th>Dermal via on-land & in-water Activities</th><th>New Value (ppm)</th><th>% Change</th></tr>
<tr><td colspan="2">Body Weight (kg)</td><td>15.3</td><td>✓</td><td>✓</td><td>✓</td><td>66</td><td>20%</td></tr>
<tr><td colspan="2">TRV (mg/kg/d)</td><td>3×10^{-4}</td><td>✓</td><td>✓</td><td>✓</td><td>66</td><td>20%</td></tr>
<tr><td rowspan="3">Exposure Frequency (d/wk)</td><td>summer</td><td>5</td><td rowspan="3">✓</td><td rowspan="3"></td><td rowspan="3">✓</td><td rowspan="3">70</td><td rowspan="3">17%</td></tr>
<tr><td>spring/fall</td><td>2</td></tr>
<tr><td>winter</td><td>0</td></tr>
<tr><td colspan="2">Oral RAF (unitless)</td><td>0.5</td><td>✓</td><td>✓</td><td></td><td>75</td><td>10%</td></tr>
<tr><td rowspan="6">Skin Surface Area (cm^2)</td><td>head</td><td>663</td><td rowspan="6"></td><td rowspan="6"></td><td rowspan="6">✓</td><td rowspan="6">76</td><td rowspan="6">8%</td></tr>
<tr><td>hands</td><td>317</td></tr>
<tr><td>arms</td><td>877</td></tr>
<tr><td>legs</td><td>1570</td></tr>
<tr><td>feet</td><td>400</td></tr>
<tr><td>torso</td><td>2503</td></tr>
<tr><td colspan="2">Sediment Loading Rate ($mg/cm^2/d$)</td><td>0.04</td><td></td><td></td><td>✓</td><td>76</td><td>8%</td></tr>
<tr><td colspan="2">Dermal RAF (unitless)</td><td>0.1</td><td></td><td></td><td>✓</td><td>76</td><td>8%</td></tr>
<tr><td colspan="2">Sediment Ingestion Rate (mg/h)
- oral exposure, shoreline play</td><td>33</td><td>✓</td><td></td><td></td><td>76</td><td>8%</td></tr>
<tr><td colspan="2">Exposure Frequency (h/d)
- oral exposure, shoreline play</td><td>1</td><td>✓</td><td></td><td></td><td>76</td><td>8%</td></tr>
<tr><td colspan="2">Sediment Ingestion Rate (mg/h)
- oral exposure, wading/swimming</td><td>7.7</td><td></td><td>✓</td><td></td><td>82</td><td>1%</td></tr>
<tr><td colspan="2">Exposure Frequency (mo/y)
- oral exposure, wading/swimming</td><td>3</td><td></td><td>✓</td><td></td><td>82</td><td>1%</td></tr>
<tr><td colspan="2">Exposure Frequency (h/mo)
- oral exposure, wading/swimming</td><td>22</td><td></td><td>✓</td><td></td><td>82</td><td>1%</td></tr>
</table>

* The toddler is the age category used in the non-cancer calculations.

Appendix C: Uncertainties and Limitations

To develop the recreational site-specific sediment guidelines for the Cornwall waterfront, conservative assumptions have been used. Uncertainties and limitations associated with these assumptions are briefly discussed below.

- The nearshore area of the Cornwall waterfront along the St. Lawrence River is made up of rock, gravel, or macrophyte beds. **Since there are no sediments within the first few metres of shore, it means the exposure calculations are over-assumptions, making these sediment guidelines conservative** when applied to recreational use of the Cornwall waterfront. This should be taken into consideration when interpreting sediment sampling data.

- It is important to note that **the scope of this assessment was limited to exposures only from *recreational use* of the Cornwall waterfront**. Potential exposures to COCs from other pathways such as fish consumption were not evaluated. Therefore, the sediment guidelines developed in this assessment are not directly applicable to other uses of the Cornwall waterfront (such as the consumption of fish or wildlife) or to any other potential exposures from living in the Cornwall area.

- Over time, contaminants in sediment may move or may get buried by depositing sediments. Therefore, contaminant exposures are not likely to be constant during long-term activities at any particular location along the Cornwall waterfront. A person at a fixed location would not be exposed to the same concentrations of contaminants over the long term.

- Sediment guidelines were developed for only three contaminants (lead, mercury, and B[a]P). Though some contaminants exceeding the screening criteria were not included as contaminants for development of sediment guidelines, adverse health effects from these contaminants are unlikely because (1) the exceedances are minimal, (2) recreational exposures are presumed to be lower than residential exposures which form the basis of the screening criteria, and (3) risk management decisions for these contaminants may also capture exposures to other contaminants to some degree.

- Analyses of sediment samples collected in the future may indicate higher contaminant concentrations than those used for the screening step (Section 2.2 above) or the presence of contaminants not evaluated at all in this assessment. In either of these cases, re-assessment may be warranted.

- The sediment guidelines are based on recent science. However, since scientific research perpetually moves forward, advancements in scientific knowledge may render parts of the approach used here and/or the values of some of the input parameters out of date. At some point in the future it may be worth revisiting the approach and input parameters used.

- 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.

- Risks to infants (up to 1 year old) were not assessed in this assessment since they would not be unsupervised, are expected to have a low overall frequency of exposure at the Cornwall waterfront, and would have a very low sediment ingestion rate compared to other age categories. Calculations based on the high exposure frequency and sediment ingestion rate of the toddler would be protective of the infant.

- Additional pathways of exposure from recreation were not assessed in the current assessment but are theoretically possible, such as ingestion of contaminants in water and inhalation of contaminants in sediment or water. However, these pathways are expected to be negligible compared to oral and dermal exposures to contaminants in sediment.

- Site-specific bioavailability 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. In the future, site-specific bioavailability or bioaccessibility data could contribute to developing more accurate sediment guidelines.