LeBreton Flats Infrastructure and Remediation Project

Ecological & Human Health Risk Assessment
Common & Riverfront Park Areas

Comments received from Senes

Reviewer/ Date of Review	No	Comment from Senes	Action from DSI
Senes Consultants February 2003	**1**	Section 2/Risk Assessment Methodologies: However, it is noted that a discussion of Level 1 and Level 2 risk management (as termed in the MOE risk assessment framework) has not been discussed.	Page 2-1, Section 2.1, After the Last Paragraph: Add «The MOE (1996; 1997) as divided risk management decisions in two (2) categories: Level 1 and Level 2 Risk Management. The current risk assessment is a Level 2 Risk Management. However, many items from the Level 1 Risk Management have been incorporated, such as the lifetime cancer risk acceptable level and the apportionment of the Reference Dose. No generic criteria were developed. At the end of the process, decisions on remediation measures minimizing risks will be proposed.»
	2	Section 4.1 Soil Characterization: However in keeping with the MOE checklist, no justification was provided that the site is not a sensitive site.	Page 4-2, Section 4.2, After the Last Paragraph: Add «The site is not considered a sensitive site based on the following elements : 1. The site does not include or have a potential to have an adverse effect on sites designated in the MOE guidelines (MOE, 1996; 1997); 2. The site has more than two (2) metres of overburden on approximately 75 % of its surface area; 3. The site has a pH usually between 5 and 9.»
	3	Section 4.1 Soil Characterization: DSI have used the more restrictive of the MOE Table B values and CCME residential/parkland numbers but it should be noted that the CCME numbers include consideration of potable groundwater, which is not the case at the site.	Comment: To ensure continuity, the same criteria as those selected for the characterization studies were used in the risk assessment.
	4	Section 4.1 Soil Characterization: Although it is appropriate to use the UCL of the mean for risk characterization, it may be prudent to conduct the screening with the upper 95th percentile of all the data rather than of the mean.	Comment: The use of the 95th percentile of all the data would lead almost to the same conclusion for the selection of COCs. In fact, if we used the 95th percentile, all the substances, except barium, would have been selected for the HHRA and ERA. The 95th percentile will be used in the next HHRA (i.e, Ottawa Municipal Park).
	5	Section 4.1 Soil Characterization: However, it is unsure where some data are included in the breakdown between Tables 5 and 6. For example for zinc, in Table 4 (all depths) it is recorded that 193 samples were included. In Table 5 (<1 m depth), 56 samples are included and in Table 6 (>1 m depth), 83 samples are included for a total of 139 samples, leaving 54 samples unaccounted for.	Comment: Some samples overlapped the two (2) layers (e.g., 0-5 m) and have thus been excluded after the breakdown (i.e., <1 m depth, >1 m depth). This situation did not affect the COC selection since it was based on all the data (see Table 4).
	6	Section 4.1 Soil Characterization: Although it is appropriate to evaluate these 10 COCs for both an ecological and human health perspective it should be noted that the MOE allows further screening based on the breakdown of the guideline into these two components.	Comment: For this assessment, we used the most conservative between CCME and MOE criteria for COC selection for both risk assessment, which retain COC that could have been excluded otherwise.
	7	Section 4.1 Soil Characterization: The description for the elimination of TPH as a contaminant of concern is somewhat confusing. From a check of the data collected using the CCME methodology, the F2 fraction concentration of TPH at SS-5 fails the CCME criterion value for ecological concerns (1500 mg/kg vs. 150 mg/kg). This would imply that the F2 fraction should be carried through the ecological risk assessment.	Comment: The sounding showing an excess in the F2 fraction is located in area to be remediated, as indicated in the legend of the table inserted in Appendix 1. Add in the Table 1 footnote «100: Concentration of the TPHCWG carbon chains that will remediated by an option yet to be selected on the basis of pump tests».

8	Section 5.1 Problem Formulation: It is noted that the organic carbon fraction values are based on in-situ measurements. However, data for this could not be located in the Phase II reports (DSI 2002a, b).	Comment: Page 4-1, Section 4.1, 4th Paragraph, Last Sentence: There is a reference to these data «Some of the results from this characterization study are incorporated in this study (see Appendix 1 - Table 1).» An example of a certificate of analysis was provided to Senes.
9	Section 5.1 Problem Formulation: It is unclear what source term was used in the assessment. On page 5-4 it states that the soil concentration used for modeling corresponds to the arithmetic mean, however on page 5-7 it states that the 95% UCL was used. From the detailed calculations it appears both are used – this should be clarified in the text.	Comment: The average concentration has been used to predict the fate and environmental media concentrations of COCs for the Problem Formulation phase only. This calculation aims to compare predicted values to measured values. UCL has been used for the exposure assessment and the risk characterization phases. Page 5-4, Section 5.1.5, Last Paragraph: Replace by «The soil concentration used for the environmental fate modeling of each COC corresponds to the arithmetic mean of all data measured in the study areas (i.e., Common, Riverfront Park) for each soil layer considered. It must be noted that the exposure scenario of ecological receptors w/ill be based on the UCL of the mean (section 5.1.7.4).»
10	Section 5.1 Problem Formulation: The Phase II report (DSI 2002b) states that the management of the groundwater after the construction work should be included in the risk assessment. There is no mention of such an assessment in the DSI report. In addition, Figure 5 shows that there are two wet ponds on the site. There is no discussion on the impact of groundwater or surface runoff to these ponds and potential receptors.	Comment : The statement about the management of groundwater after the construction was made before the update of the report. The update results showed that the groundwater was slightly contaminated, with no VOCs and metals. It is believed that no further impacts on the groundwater quality will occur after site remediation is completed. Also, the two (2) pounds are not currently present on the site.
11	Section 5.1 Problem Formulation: No discussion was found that explicitly addresses the potential groundwater issues.	Page 5-2, Add a bullet between Bullet 5 and Bullet 6 «6. The groundwater is slightly contaminated, with no VOCs and metals. It is believed that no further impacts on the groundwater quality will occur after site remediation is completed.»
12	Section 5.1 Problem Formulation: Note that Figure 9 does not correspond to the assessment endpoints provided on page 5-6.	Has been modified.
13	Section 5.2 Assessment Method: It is unclear what level of assessment is intended. Section 2.1 of the DSI report states that the ERA corresponds to a Tier 2 assessment. Section 5.2.2 of the DSI report states that the Tier 2 ERA will follow if needed.	Page 5-8, Section 5.2.2, First Paragraph, Last Sentence : Replace by «At this step of the process, the inverse decision, i.e., to conclude that there is a risk when there is none, is less worrisome because the quantitative or site specific ERA (Tier 3) will evaluate the risk in detail, if needed (see section 2.1).»
14	Section 5.3 Hazard Assessment: the following differences should be noted: • The MOE has toxicity benchmarks for PAHs of 40 mg/kg in soil. • The toxicity benchmark for arsenic for birds was based on copper acetoarsenite. This species is unlikely to be found in the soil, it may be more appropriate to use the benchmark for sodium arsenite in mallard ducks of 5.1 mg/kg/d. However, the benchmark used in the assessment is more cautious. • No benchmarks are reported for chromium species in birds. While it is acknowledged that the hexavalent species of chromium is more toxic, a benchmark is available for trivalent chromium, which could be used to assess the potential impacts in birds exposed to chromium from the site. • For lead exposure in birds, the toxicity benchmark is based on metallic lead, which is not bioavailable. Another benchmark exists for lead acetate, which is more bioavailable, and should be used in the assessment. This value is 1.13 mg/kg/d and is based on a study on Japanese Quail carried out by Edens et al. 1976.	Comments: The use of toxicity benchmark for arsenic based on sodium arsenite exposure of mallard duck (5.1 mg/kg/d) instead of the copper acetoarsenite (2.46 mg/kg/d) as increase proportionally the risk for bird by a factor of about 2. Even with this latest calculation, the Hazard Ratio (HR) is still below one (0.003). The same conclusion was reached the toxicity benchmark for lead was used with lead acetate (1.13 mg/kg/d) instead of metallic lead (3.85 mg/kg/d). The risk for gull has been increased by a factor of about 3 and is still below the unity (HR = 0.43). For chromium, the calculation based on the trivalent form (1 mg/kg/d) give a HR for gull of 0.007. These results do not modify the overall conclusion. The text, tables and figures will be adjusted in consequence.
15	Section 5.4 Exposure Dose Assessment: Note that the same equation (Eing = Ef + Es) appears twice on pages 5-10 and 5-11.	Has been modified.

16	Section 5.4 Exposure Dose Assessment: It would be beneficial to provide more detail on the calculation of concentration in small mammals and earthworms as only a reference was provided. Spot checks of the estimated concentrations in small mammals and earthworms were completed to verify the values. Are the concentrations provided in Appendix 4 (Tables 4 and 5) the results of the product of 2 distributions?	Comment: A footnote in Table 4 and Table 5 of Appendix 4 will describe the calculation for these receptors.
17	Section 5.5 Risk Characterization: Based on the results of the ecological risk assessment it was suggested that risk management measures be implemented to reduce the risk to, earthworms and terrestrial plants. However, Section 7 of the report indicates that risk management measures are not needed since the screening index values are only slightly above 1 for plants and earthworms.	Page 5-15, Section 5.5.7, Second to Fourth Paragraph: Replace by «Based on the assessment endpoints (hypotheses) and the quotient method, the Hazard Ratio (HR) calculated for earthworms and plants would lead to a weak risk for specific COCs. On this basis, we could then conclude that, for individual organism: 1. There is an inhibition of the growth or the activities of the soil microorganisms; 2. There is a reduction of the survival or the growth of earthworms; 3. There is an inhibition of the growth and reproduction of terrestrial plants. However, if we consider the uncertainty related to the exposure concentration (i.e., soil) and to the reference values (ENEV), the HR could overestimate by up to one order of magnitude. In addition, there is no direct field evidence how this potential risk could affect these receptors on a population, community or ecosystem level. Consequently, the calculated risk can be considered has weak and non significant.»
18	Section 5.5 Risk Characterization: From a review of the data it would appear that the UCL 95% is below the MOE ecological toxicity data for chromium and zinc and although MOE does not provide a value for lead, the UCL is below the CCME ecological value. A comparison to these criteria would provide a broader perspective to the analysis and strengthen the rationale for no mitigation measures.	Page 7-1, Section 7.1.2, After Bullet 4: Add Bullet 5 «Finally, it must be point out that the all the COCs that show a HR higher than one (lead, zinc, chromium) have their soil UCL 95 % lower than the MOE or CCME ecological toxicity limit.»
19	Section 6.1 Hazard Identification: As DSI has stated that the contamination is heterogeneous at the site. Is there any particular infrastructure planned at the site that would make part of it more attractive and thus weight the exposure time accordingly? The source characterization may not be appropriate if it is likely that more time would be spent in a specific area of the site.	Comment: To our knowledge, no specific infrastructure planned at the site would make part of it more attractive.
20	Section 6.2 Toxicity Assessment: Some additional discussion is warranted regarding the selection of toxicity data used to assess exposure from PAHs. For example, the MOE has a dose-response document that should be discussed (MOEE 1997). As well, there is no discussion regarding the selection of data. For example, IRIS has an oral slope factor for benzo(a)pyrene yet the information from CalEPA has been used in this assessment with no rationale provided.	Comment: Additional information is presented in the new Section 6.2.2.1 Polycyclic Aromatic Hydrocarbons Slope Factors and in the new Section 6.2.2.2 Polycyclic Aromatic Hydrocarbons Reference Doses (see text below). For this study, no discussion on the MOE document will Comment: Additional information is presented in the new Section 6.2.2.1 Polycyclic Aromatic Hydrocarbons Slope Factors and in the new Section 6.2.2.2 Polycyclic Aromatic Hydrocarbons Reference Doses (see text below). For this study, no discussion on the MOE document will be provided. However, this information will be integrated in the next HHRA (i.e., Ottawa Municipal Park). Page 6-2, Section 6.2.1, Add a new Section 6.2.2.1 Polycyclic Aromatic Hydrocarbons Slope Factors: «Many authors also proposed equivalence factors to define the carcinogenic strength of other PAHs, based on benzo(a)pyrene (Nisbet & Lagoy, 1992; CalEPA, 1999; Krewski et al., 1989; Malcolm & Dobson, 1994; McClure, 1996; Meek et al., 1994, Muller et al., 1997; U.S. EPA, 1993). For example, CalEPA (1999) uses identical potency equivalency factors to those proposed by Nisbet & Lagoy (1992), with the exception of dibenzo(a,h)anthracene. Therefore, the equivalence factor for a PAH was established by attributing a relative toxic weight to the composite comparative to benzo(a)pyrene (this molecule has a factor equal to 1). Then, by multiplying the measured PAH concentrations taken individually by the equivalence factor, it is possible to obtain the benzo(a)pyrene substance equivalent.

The slope factor for the oral route for benzo(a)pyrene was established by way of the median value of the slopes obtained in the Neal & Rigdon (1967) study of the mouse and in Brune et al. (1981) study of the rat Sprague Dawley. A significant increase in tumours was observed in the small intestine (Neal & Rigdon, 1967; Brune et al., 1981), in the oesophagus and the larynx (Brune et al., 1981) after repeated ingestion of benzo(a)pyrene. The value proposed by the U.S. EPA (IRIS, 2002) is established at 7.3 (mg/kg/d)-1 and is based on the geometric medium of peaks obtained from four different extrapolation methods of low doses. The California EPA (1999) prefers linear multistage extrapolation estimates obtained from the study by Neal & Rigdon (1967) and proposes a coefficient of 11.7 (mg/kg/d)-1. This latter value being more conservative, it has been used for the HHRA. This aspect will be discussed in the uncertainty section.

According to the U.S. EPA, there are no epidemiological or toxicological studies that can establish the specific relationship between PAH exposures by inhalation and the increase of tumours in the bronchial tree. Therefore, no slope factor was developed by this agency for this route. Nonetheless, the CalEPA (1999) proposes a slope factor for this pathway by using the Thyssen et al. (1981) study results. These authors noted a significant increase in tumours in the nasal cavity, the pharynx, the larynx and the trachea in Syrian hamsters exposed to benzo(a)pyrene for 109 weeks. Based these results, the authors suggest a LOAEL of 9.5 mg/m3. Finally, numerous studies were conducted in animals, specifically the mouse (ATSDR, 1995), for dermal pathways. No governing agency or regulatory body has proposed a reference value for this pathway.

Add a new Section 6.2.2.2 Polycyclic Aromatic Hydrocarbons Reference Doses after the new Section 6.2.2.1: «U.S. EPA (2002) has defined a reference dose for naphthalene for ingestion and inhalation routes. The reference dose is based on a NOAEL of 71 mg/kg/d obtained with a rat study (BCL, 1980). An uncertainty factor of 3,000 has been used (10 to extrapolate from rats to humans, 10 to protect sensitive humans, 10 to extrapolate from subchronic to chronic exposure, and 3 for database deficiencies including the lack of chronic oral exposure studies and 2-generation reproductive toxicity studies) to derive a chronic reference dose for naphthalene of 0.02 mg/kg-day. For the respiratory route, the reference concentration is based on a rat study (NTP,1992). The adjusted LOAEL of 9.3 mg/m3 for nasal effects was divided also by an uncertainty factor of 3,000 to arrive at a chronic reference concentration C for naphthalene of 0.003 mg/m3. There are no known reference doses to date for the dermal pathways.»

Add a new Section 6.2.2.3 Metals and Inorganics Slope Factors: «U.S. EPA proposes slope factors for arsenic (ingestion, inhalation) and chromium VI (inhalation only) based on several human studies in general population (Tseng et al 1968; Tseng, 1977) and occupational milieu (Brown and Chu, 1983a, 1983b,1983c; Lee-Feldstein, 1983; Higgins et al, 1982; Enterline and Marsh, 1982). For lead, CalEPA (1999) gives a slope factor for ingestion and inhalation based on a toxicological study on rats (Azar et al.. 1973).»

And a new Section 6.2.2.4 Metals and Inorganics Reference Doses: «For non carcinogenic COCs, U.S. EPA (IRIS) gives reference dose for arsenic (Tseng et al 1968; Tseng, 1977), barium (Wones et al, 1990; Brenniman and Levy, 1984; NTP, 1994) and chromium VI (Mckenzie et al, 1958). These RfD are based on a NOAEL for which uncertainty factors varying from 3 to 300 has been applied. For copper, Health Canada (CCME, 1999a) established safe exposure doses in order to avoid any deficiency or excess of this essential element (Adult: 0.03 mg/kg/d; Child 3-11 years old: 0.05 to 0.1 mg/kg/d).

		Recently, Health Canada (see Rationale in appendix 8) revised its dose limit or Total Daily Intake based on the results of the Food and Nutrition Board of the Institute of Medecine (IOM, 2002). This organism state that the maximum acceptable dose would be 10 mg/person. Considering that the normal daily dose is about 5 mg/person (IOM, 2002, IPCS, 1998), Health Canada proposes to add the maximum dose of the IOM with this daily dose to obtain a value of 15 mg/person. In assuming a body weight of 70 kg, the TDI would be then 0.021 mg/kg/d.»
21	Section 6.2 Toxicity Assessment: Additionally, it should be noted that the MOE has a toxicity value for lead (an Intake of Concern of 1.85 µg/kg/d) and the Guidance on Site Specific Risk Assessment for Use at Contaminated Sites in Ontario (MOEE 1996) states that this value should be used in lieu of values from other jurisdictions.	Comment: The calculation and the text have been be adjusted to take into account the MOE toxicity value for lead (1.85 ug/kg/d). The Hazard Ratio (HR) of each scenario has increased by a factor of about 2. Nevertheless, the HR is still well below 1 (< 0.1).
22	Section 6.2 Toxicity Assessment: A review of the data contained in Table 15 revealed other items: • CalEPA has inhalation toxicity data for arsenic and copper, which were not included in the study. • The value of 1.0E-04 for inhalation of chromium VI is an RfC not an RfD. • There is a number of 1.3 mg/L (or 0.037 mg/kg/d) for copper in HEAST (USEPA, 1997). • There is a typographical error in the chromium VI unit risk for inhalation (should read 0.012 (µg/m3)-1), the slope factor shown is correct.	Comment: The calculation and the text have been adjusted to take into account the available toxicity values for inhalation (i.e., arsenic, copper and chromium VI). For copper, the most recent value proposed by Health Canada (2002) will be used (0,03 mg/kg/d) see new section 6.2.2.4. Toxicological fax sheet for this substance will be presented in an appendix. Since the exposure by inhalation is not a significant pathway, the inclusion of the reference dose has not change the risk index. For copper, the new reference dose is less conservative than the former and consequently reduces the risk. For that substance also, the risk remain negligible.
23	Section 6.3 Exposure Assessment: The selection of the local population in the future scenario is appropriate although it is uncertain why the exposure time for the local population after redevelopment was reduced to 0 (Table 17).	Comment: The local population, after redevelopment, is exposed via inhalation of particle or gas emitted all year long, but there will be no direct contact to soil (n of days = 0). This population corresponds only to the local resident that will not use the site. The local population that will use the site and visitors (i.e., user group) will be exposed to clean soil trough ingestion, inhalation and direct contact for a maximum of 5 days per year. A footnote will be added in the table 17 for additional explanation.
24	Section 6.3 Exposure Assessment: Thus only pathways for volatile contaminants (e.g. naphthalene in soil) would need to be considered.	Comment: Based on an average soil concentration for naphthalene (e.g., the most volatile substance on the site), air concentration predicted by CalTOX would be non detectable before the redevelopment (see table 10).
25	Section 6.3 Exposure Assessment: The DSI report states that the assumed exposure for 5 days a year corresponds to 20 site visits of 6 hours each. This is not true as soil ingestion, although expressed on a daily basis, does not occur evenly throughout a 24-hour period and thus cannot be directly scaled on an hourly basis.	Comment: On a yearly basis over a lifetime, this assumption is valid. However, with CalTOX, the exposure is express in term of duration (i.e., nb. of years) and frequency (i.e., nb. days/year). After redevelopment, an average exposure of 5 days is assumed for the user group, which could be interpreted as 20 visits of 6 hours or any other way.
26	Section 6.3 Exposure Assessment: One parameter that was difficult to trace was skin surface area. We could not locate the values in the Richardson 1997 report. If there was some adjustment for portion of the body exposed this should be specified in the table.	Comment: Based on the Richardson study (1997), only the hands and arms exposure by dermal contact were used. This will be mentioned in Table 17.
27	Section 6.3 Exposure Assessment: Some of the exposure times are unusual (e.g. it is unlikely that an infant 0-6 months of age would spend 10 hours at rest and 11.2 hours active each day). However, the assumptions regarding active and resting exposure time are conservative and unlikely to have a significant impact on the result.	Comment: In CalTOX, the contact rate for the inhalation route take into account the resting and active time indoor, and also the time spent indoor and outdoor. This breakdown is not defined explicitly in the literature (e.g., U.S. EPA). The time spent indoor resting has been fixed to 10 hours for all target groups and the active time (outdoor and indoor) has been adjusted to consider the values proposed by Richardson (1997) for each target group (outdoor). The active time indoor corresponds to the difference of these values and a total of 24 hour. For children or baby, these assumptions are conservative.

28	Section 6.3 Exposure Assessment: One point of note is that with the use of an exposure model it is beneficial to provide more detailed output (such as predicted air concentrations) to allow the verification of results.	Comment: An appendix will include an example of intermediate calculation (pages 1, 9, 10 and 11 of CalTOX outputs) for two (2) substances: naphthalene and lead. The first page will show the timed average concentration for each environmental media (e.g., air, soil), whereas the other pages will give intermediate calculation for the exposure dose. It must be mentioned that all the output (18 pages) for these substance was provided to SENES for peer review. These data will allow a comparison of method for dose calculation (e.g., dermal exposure).
29	Section 6.3 Exposure Assessment: In general, the results of the spot checks had good agreement however, a few issues were identified: • Dermal exposure rates calculated following the methodology outlined by USEPA RAGS, also shown as equation 78 in the CalTOX Technical Manual (CalEPA, 1993), with an absorption fraction of 0.03 for arsenic produced much higher dose estimates than those given in tables .• Naphthalene exposures calculated were higher than those produced by CalTOX. • It is not obvious where the difference between the exposure estimates for the Common area and Riverfront areas are since the same source term was used for each and no indication was found regarding different exposure scenarios.	See response to comment #34. A table has been added to the Appendix 3 that shows the initial concentration used for modeling and corresponding to scenario presented in Table 18.
30	Section 6.4 Risk Characterization: The use of a HR of 1 for comparison is not appropriate as the Guidance on Site Specific Risk Assessment for Use at Contaminated Sites in Ontario (MOEE 1996) states that to deviate from the apportionment of 20% a multi-media exposure assessment must be completed along with an assessment of exposures from other sources not associated with the site (i.e. background levels). The results provided by DSI show, however, that HRs are generally less than 0.01 and would not be considered an issue.	Comment: This aspect will not be considered in the actual version of the HHRA. Since all the Hazard Ratio (HR) are lower than 0.1, it will not modify the interpretation of the results, nor the conclusion. We must mention however that this will be included in the next HHRA (i.e., Ottawa Municipal Park).
31	Section 6.4 Risk Characterization: The calculation of an absorbed dose for dermal exposure does not preclude its inclusion in the assessment of a hazard ratio or risk. Appendix D of the MOEE (1996) provides guidance on the use of absorbed versus administered doses.	Comment: As proposed by MOE and U.S. EPA (RAGS), the exposure dose can be converted to an internal dose. In our case, since the dermal route is insignificant (e.g., COC not easily absorbed by skin), the internal dose calculation was not considered.
32	Section 6.4 Risk Characterization: The MOE guidance requires the assessment of mixtures of contaminants that may act on the same target organ. Thus, an acknowledgment of the total risk from PAHs exposure should be incorporated.	Comment: In this version of the HHRA, we will not calculate the total risk by target organ. In the present case this calculation will not change the overall conclusion of the study. This calculation will be made in the next study (i.e., Ottawa Municipal Park). However, for the sake of the discussion, we added a new section on the general aspect of interaction between PAHs. Page 6-8, Add new Section 6.2.3 Interactions between Polycyclic Aromatic hydrocarbons: «Humans are usually exposed to a complex mixture of PAHs. The majority of the toxicological studies only assess individual PAH effects. When humans are exposed to these complex mixtures, there are a variety of toxic responses that could be additives (where the combined effect is equal to the sum of effects from each substance calculated individually (1+1=2)), synergic (if the effect of the mixture is higher than the sum of the individual effects (1+1=5)), the potentialization (if there is an increase in a substance's toxicity with another non-toxic substance (2+0=10) or antagonistic (if the effect of 2 substances is less than predicted (2+2=1)).

Therefore, interactions between different PAHs need to be considered, when possible. For example, non carcinogenic PAHs could become carcinogenic in the presence of other PAHs. Also, the simultaneous introduction of a non carcinogenic PAHs or a minute carcinogen, such as benzo(g,h,i)perylene, fluoranthene or pyrene could increase benzo(a)pyrene tumour incidences. On the contrary, some studies have equally demonstrated that PAHs mixtures could be less toxic with certain PAHs. For example, some authors suggest that while benzo(a)anthracene is administered with benzo(a)pyrene, the benzo(a)anthracene has an anti carcinogenic effect in reducing the mutagenic activity of benzo(a)pyrene on embryonic cells in hamsters (ATSDR, 1995).

These interactions existing between the many PAHs are too numerous and complex to understand. Aside from the interactions between different PAHs, all substances with a capacity of inducing the enzymes implicated in the detoxification of PAHs would influence the toxicity of the PAHs (increase the reactive metabolites). Nicotine in the smoke of a cigarette would modify PAH toxicity (Foth et al., 1988, ATSDR, 1995).

Despite the importance of this phenomenon and the fact that little information/literature is available regarding the interactions between the numerous PAHs and the complexity of this problem, this aspect was not considered in this assessment.»

33 Section 6.5 Sensitivity And Uncertainty Analysis: There is a lack of information regarding the range of uncertainty applied to each of the input parameters that was used in the simulation however the results do seem appropriate. The uncertainty analysis focussed on the uncertainty associated with the source term. This is certainly a key area of uncertainty, although other areas that contribute to uncertainty, such as toxicity data and exposure parameters, should be discussed.

Page 6-15, Section 6.5.2, Replace the Section by a new one: «Section 6.5.2 Uncertainty Analysis: The evaluation of the uncertainty in the risk estimations can yield useful information to the risk assessor and may help to focus on key factors and parameters that affect significantly the risk levels. This evaluation must take into account several factors such as the representativeness and precision of the soil data, the input parameters, the exposure scenarios, the toxicological information, etc. For discussion, all these sources of uncertainty as been regrouped into three main contributors: the source term, the exposure parameters and the toxicological reference values.

6.5.2.1 Uncertainty Related to the Source Term: In the present case, the soil characterization was based on the chemical analysis of 10 COCs out of more than 40 samples for PAHs and 180 samples for inorganic substances in the study areas (see Table 4). These soil samples were taken at different depths and sampling points throughout the site. Even if there was no specific sampling strategy (i.e., random or stratified sampling), the importance of this sampling effort may have compensated for the statistical representativeness of the sample. On the other hand, the sample size affects directly the precision of the estimation, giving a narrower confidence interval relatively to the mean. Based on the coefficient of variation, the variability surrounding the COCs can vary from 50 to 300 %.

6.5.2.2 Uncertainty Related to the Exposure Dose: To illustrate the variability surrounding the input parameters, an uncertainty analysis has been done with CalTOX. As an example, the cancer risk level related to the exposure of the local population to Benzo(a)pyrene in the Common area before development (current state) was calculated using Monte-Carlo simulations (see Table 21).

The calculations were done using the average and standard deviation of Benzo(a)pyrene in upper and deep soils instead of the UCL95% used for risk calculation for all the scenarios. The results indicate that the cancer risk level calculated with CalTOX (point estimate) fell between the 95th percentile and the maximum value obtained using Monte-Carlo simulations using the average concentration (see Table 31). Considering the uncertainty related to all the parameters used for the simulations (see Appendix x), the CalTOX estimation would seem to be an appropriate reasonable maximum. On the other hand, the global variability around the risk estimate, based on physico-chemical, landscape and exposure parameters is less than an order of magnitude with a factor of about 2.8 (CV=276%).

		6.5.2.3 Uncertainty Related to the Reference Values: The slope factors used for the oral and respiratory route were provided essentially by the U.S. EPA (IRIS) and CalEPA. For PAHs, we used the CalEPA coefficient (11.7 [mg/kg/d]-1) for benzo(a)pyrene and its potency factors for the other congeners. If we compare this latter value to the one proposed by the U.S. EPA (7.3 [mg/kg/d]-1), the risk index would increased by about 38%. However, the incidence on the calculated risk would be non significant. On the other hand, despite the limited data for respiratory pathways, the study by Thyssen et al. (1981) is judged to be sufficient by CalEPA to define a carcinogenic coefficient for inhalation. When considering all the COCs (PAHs, metals and metalloids), we can mention that the conservatism of the carcinogenic coefficients used for the oral and respiratory pathways provides an adequate assessment of potential risks associated with soils contaminated with these COCs. For the non carcinogenic substances, the reference doses were defined by U.S. EPA (As, Ba, Cr VI), MOEE (Pb) and Health Canada (Cu). The uncertainty around these values is usually taken into account in using uncertainty or modifying factors. In our case, these factors can vary from 3 (e.g., As, Ba) to 3,000 (i.e., naphthalene). 6.5.2.4 Risk Evaluation: When considering the global variation of all input parameters, including the source terms, the uncertainty may reach at most one order of magnitude. However, the conservatism of the hypotheses, the level of protection considered in the toxicological values (i.e., RfD, RfC and slope factor) give a good margin of safety and a good confidence in the estimated risk. The risk level after the redevelopment can be then judged as non significant.»
34	Section 6.5 Sensitivity And Uncertainty Analysis: Again there is a lack of information to assess the appropriateness of the Monte Carlo simulation (e.g. was only the source concentration varied, what type of distribution was used, how many runs were completed). Considering the results of the overall analysis, the results of the Monte Carlo simulation are likely not necessary.	See response to Comment 33.
35	Section 7.0 Ecological and Human Health Risk Management: A groundwater monitoring component should be incorporated into the risk management plan due to the presence of PAHs on the site.	Page 7-5, Add at the end of the section: «Furthermore, a limited groundwater monitoring program should be implemented».
36	Overall Opinion: On a minor note, the references in the document should be checked to ensure that all cited documents are present in the reference list. For example, Concannon et al. 1997 and Prescott and Richard 1996 are not present in the list of references.	Has been modified.