APPENDIX J

Response to Peer Review Comments

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June 8, 2007

Christopher Ollson
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Jacques Whitford Limited
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[via email: COllson@jacqueswhitford.com]

Re: Peer Review of the Regions of York and Durham Energy-from-Waste Generic Risk Assessment

Dear Chris:

Intrinsik Environmental Sciences Inc. completed a peer review of the human health and ecological risk assessment components of the Jacques Whitford report entitled "Draft Report, Energy-From-Waste Generic Risk Assessment Feasibility Study". The review was completed by Chris Bacigalupo, Ruth Hull, Shannon McDowell and myself.

Specific review comments are provided in an Attachment to this letter. Overall, it is our opinion that the Human Health Risk Assessment (HHRA) and Ecological Risk Assessment (ERA) are well written and presented in a clear, logical and concise manner. In general, the approach seems to err on the side of conservatism (i.e., may substantially overestimate risks in some cases) and as such it is unlikely that risks are underestimated. It is our opinion that the HHRA and ERA will be acceptable once the issues outlined below are addressed.

We thank you for the opportunity to review this important work. If you require any additional information or clarification of our comments, please do not hesitate to contact me at your convenience at 905-814-7800 (ext. 222) or esigal@intrinsikscience.com.

Sincerely,
INTRINSIK ENVIRONMENTAL SCIENCES INC.

Elliot A. Sigal, Executive Vice President
Senior Scientist

ATTACHMENT: PEER REVIEW COMMENTS

1.0 Human Health Risk Assessment

Overall, the HHRA is well written and presented in a clear, logical and concise manner. All of the major components of a typical HHRA (including problem formulation, exposure assessment, toxicity assessment, risk characterization and uncertainties) have been presented. The general selection and assessment of human receptors, exposure pathways and locations are appropriate. In general, the approach seems to err on the side of conservatism (i.e., may substantially overestimate risks in some cases); however, this is expected for such a report.

The only substantial improvements might be to either modify or remove Section 3.0 from the report. This section seems awkward and confusing at times and does not appear to fit into the overall risk assessment process. A discussion of chemical screening provided in Section 3.0 appears to contradict the potency screening discussion in Section 2.0.

In a number of specific situations, we were unable to re-create the exposure and risk calculations given the data provided. This does not automatically indicate that the modelling techniques are incorrect or erroneous but rather, a number of the final model values could not be recreated with the information provided. The report would likely benefit from a final QA/QC review of the input parameters, assumptions and calculation intended for use in the assessment.

Some of the specific comments presented below may result in changes to the quantitative estimates of risk for some chemicals. The specific comments have been provided in the order of perceived importance to the overall quantitative results.

HHRA Specific Comments

1. Table C.1 (of Appendix C) indicates that a mixing zone of 10 cm was used to derive soil concentrations under both tilled and untilled soil conditions. The rationale provided states that a mixing zone of 10 cm is "*supported by studies which indicated that the highest soil concentrations from surficial deposition may be 10 to 15 cm below surface*". No references or studies have been provided to support this claim. Secondly, the rationale field indicates that "*recommended values range from 1 cm (unfurrowed) to 20 cm (furrowed); however, since some leaching of deposited particulates will occur, a value of 10 is being used*."

   A review of the U.S. EPA, 2005 document does not indicate a recommended range of soil depths. It mentions that tilled soil depths are approximately 10 to 20 cm, depending on the local conditions; however, it does not recommended a range of soil mixing zones. This parameter/assumption is very important and can have a significant influence on the overall outcome of the assessment. The U.S. EPA 2005 recommends a mixing zone for untilled soils of 2 cm and 20 cm for tilled soils. Untilled soil concentrations are typically used to assess direct soil contact pathways (e.g., soil ingestion and dermal contact). By using a mixing zone of 10 cm (instead of 2 cm), the assessment has potentially under predicted direct soil contact exposures by 5 fold while potentially over predicting root-uptake related pathways by 2 fold. If this assumption is to be used in the risk assessment, it warrants a significant

discussion in the main report to justify why the U.S. EPA 2005 recommended mixing zones should not be used.

2. Figure (Table) 2-2 – Missing B(a)P, B(a)F and B(a)A from the B(A)P group; if only Cr(III) is considered, then the exclusion of Cr(VI) and/or Cr(total) should be explained.

3. Figure (Table) 3-2 – MPOI locations require some explanation: 1-hour – vapour and particulate are in same location, Scenario 1 and 3 are the same while Scenario 2 is different; 24-hour - all in same location; Annual - Scenario 1 and 2 are the same, Scenario 3 is different. This information is not intuitive and should be explained.

4. Page 30. Section 5.1.1 indicates that the receptor age classes (*i.e.,* infant, toddler, child, teen, and adults) and associated physical characteristics (*e.g.,* body weight, inhalation rates, etc) recommended by Health Canada (2004) were used, in part, to facilitate the exposure assessment. Appendix A reports that produce intake rates from the U.S. EPA Exposure Factors Handbook (EFH) were used. A number of significant comments follow:

    i. Although the EFH table number is provided, we were not able to find any indication as to which age class, percentile, region or season was used to develop age-specific vegetable intakes. It is noted that the receptor age classes reported by the U.S. EPA EFH differ from those of Health Canada.

    ii. The U.S. EPA EFH reports per capita intakes of exposed vegetables (e.g., Table 9-9) as g/kg-day. It appears as though the unit conversion between g/kg-day and kg/day (as reported in Appendix A.2 for example) may be incorrect or calculations are occurring that have not been reported.

    iii. As the U.S. EPA EFH points out, intake rates are reported as g/kg/day and correcting for body weight to derive an intake on a g/day or kg/day basis using a single body weight (such as 16.5 kg for a toddler as reported by Health Canada) is not appropriate. The intake rates reported by the U.S. EPA EFH were indexed over the body weights of the actual survey respondents.

    iv. The use of data presented in Table 13-71 is considered appropriate. It should be noted, however, that the fractions selected for use in the assessment are consumer only fractions (i.e., those who did not report gardening were excluded from the dataset). Although this is conservative, it was considered appropriate in the derivation of a reasonable maximum exposed individual.

    v. The EFH cautions that consumption rate data are based on short-term observations (i.e., 7 days) and therefore are not appropriate for use in long-term exposure assessments. This is particularly true for home produced vegetables and fruits since consumption rates would be highly correlated to season (*i.e.,* spring, summer, fall and winter). As

a result, the U.S. EPA EFH attempted to derive a long-term distribution of the average daily intake rates of home produced foods from the short-term data available for major food groups (vegetables, fruits and meats). The seasonally adjusted distributions for a given region (e.g., the north eastern region) were derived by averaging the intake rates for each of the four seasons (spring, summer, winter and fall). The assessment may consider comparing the home garden vegetable consumption rates used in the assessment with the seasonally adjusted intakes reported by the EFH.

5. Section 5.22 provides a table of "relative bioavailability" values. No discussion or definition of the term "relative" bioavailability could be found in the text. Secondly, the relative oral bioavailability values reported in Table (or Figure) 5-7 are not consistent with those reported in several of the appendices. It is unclear which values were actually used in the exposure/risk calculations.

   The following inconsistencies were noted:

   - Arsenic oral 0.35 vs. 1.0
   - Cadmium oral 0.75 vs. 1.0
   - Cobalt oral 0.29 vs. 1.0
   - lead oral 0.6 vs. 1.0
   - nickel oral 0.19 vs. 1.0
   - PCBs dermal 0.14 vs. 1.0
   - PCDD dermal 0.03 vs. 1.0

6. Section 5.4.2 provides the general equation of a Hazard Quotient (HQ); however, the equation used in Appendix D (i.e., the actual methods) differs from this. Appendix D (Section D.3.2) indicates the use of a "reference dose absorption factor" on the bottom of the HQ. We were unable to easily re-create the HQ for lead and nickel as a result of soil/dust ingestion (Table F.2). It appears that there may be some confusion surrounding the application of RAF values in the derivation of the final HQ value. From the information provided, it is unclear if the assessment intended to consider chemical, media and route-specific RAF values.

7. Page 9. Figure (or Table) 3-3. Section 3.1.1 indicates that the "*Guideline A-7 emission concentrations limits were used as default exhaust stack air emission estimates for eight pollutants contained in the guideline to evaluate the potential risk to the surrounding environment.*" If the A-7 emission limits were used, why do the maximum COPC concentrations presented in Table 3-3 change from one scenario to the next (i.e., 4000,000 t/y; 266,666 t/y and 133,33 t/y) for these eight COPCs? Should the concentrations not be the same under all scenarios for these eight compounds since you've used the A-7 limits, which are maximum emission limits for a facility of this nature?

8. Page 24 provides a discussion of typical Ontario background concentrations of COPC in soil and the general approach used to predict facility related COPC concentrations in soil. The assessment notes that typical Ontario soil concentrations are not "site specific background" and that local background soils should be collected during a site-specific assessment so that cumulative effects can be evaluated.

Section 5.6.2.3 does indicate that background concentrations were not incorporated into the current study, however, it should be clearly stated (perhaps within another location of the report) that the current study does not consider cumulative health risks but rather facility related risks only. The same type of exercise (i.e., a comparison of modelled versus ambient) could be conducted with air data collected from within Ontario.

9. Section 3.1.4 provides a very limited dataset of background air quality; data from other locations might be useful to supplement this dataset (e.g., Dann).

10. Figure (Table) 3-11 – Combustion gases: total pollutant concentrations for Scenario 1 vs. 2 vs. 3 seems incorrect; Scenario 1 columns do not add up (we believe the Scenario 1 Total Pollutant Concs are incorrect and that the Total Pollutant plus Bkg columns are correct).

11. Figure (Table) 5-3 – B(a)P group missing phenanthrene; missing DEHP; this table/figure should be consistent with Figure (Table) 2-2.

12. Section 5.2.4 – Additivity of Risks – not conservative to exclude the potential for this to occur; compounds acting on similar systems *via* similar toxicological mechanisms should be considered additive (i.e., we suggest grouping the chlorinated monocyclic aromatics together and consider additive).

13. Page 24. Indicates that soil loadings were modelled over a 35 year life expectancy of the facility. Appendix D (Equation D1.2.2) indicates 28 years.

14. No references were provided with respect to the derivation of house-hold dust ingestion rates for infants, toddlers and children. The assessment has appropriately used the recommended soil/dust ingestion rates provided by Health Canada (2004); however, it appears to have derived an indoor dust ingestion intake (24.7 mg/d for toddler) based on the surface area of ½ a finger and “finger mouthing events” per hour. No citation for such an approach has been provided. Intrinsik is of the opinion that the soil/dust ingestion rate of 80 mg/day for a toddler (as recommended by Health Canada) inherently includes both outdoor soil and indoor dust.

15. Using the information provided in Appendix D, C and the main report, we were unable to recreate the soil concentration for lead under scenario 1. Appendix E (table E.1) reports a concentration of lead in soil of 9.73E-03. Although our estimate of 7.1E-03 is very close, we cannot explain the difference. It should be noted that Appendix D does not indicate that the U.S. EPA, 2005 recommends the soil loss constant due to erosion be set to zero. It is unclear if this loss constant has been evaluated in the current assessment and may be the reason for the disparity in values.

16. Using the concentration of lead in soil (as reported in Table E.1), the equation in Appendix D (Section D.2.3.5), and the data provided in Table A.2, we were unable to recreate the chronic daily intake of lead for the residential Toddler (Soil/Dust – Summer – Outdoor) as reported in Table F.2. We were unable to recreate the lead concentration in wild game as reported in Table E.1. It is noted that we were able to

recreate the concentration of lead in aboveground and belowground produce due to deposition and root uptake, given the reported concentration of lead in soil.

17. Appendix D makes reference to unitized yearly deposition rates and chemical-specific emission rates during the derivation of the deposition term. It is noted that actual unitized deposition rates (s/m^2-yr) were not found in either the risk assessment or the air modeling document. The risk assessment already reports annual average chemical-specific deposition rates (g/m^2) and, therefore, there would be no need for the emission rate parameter Q (g/s).

18. Exposure and risk calculations were confirmed for an infant's exposure to dioxins via the consumption of breast-milk. It is noted, however, that the RA appears to reference the 1998 draft U.S. EPA HHRA Protocol rather then the final 2005 Protocol. A comparison of the risk assessment methods from 1998 versus the final 2005 protocol identified a number of potential minor deviations. The breast milk consumption rate recommended in the 2005 protocol differs slightly from what has been used.

19. The discussion on page 51 speaks to the conservatism of selected fish consumption rates recommended for use by Health Canada (2004). These data are cited as coming from the Canadian Compendium of Exposure Factors (Richardson, 1997). The Richardson (1997) consumption rates for non-native populations include a wide variety of fish products (including canned salmon, tuna, shell fish, seafood, etc.). The assessment should point this out and discuss the applicability of these intake rates to the exposure scenario being developed.

20. Appendix B (TRVs)

    - In many cases, only the selected value is provided; however when an IRIS value is available but not used, it should still be discussed. A good example is nickel, IRIS is used for oral; however, IRIS numbers are not even addressed for inhalation.
    - Dichloromethane – should include discussion of EPA slope factors/RfDs
    - Formaldehyde – should include discussion of EPA inhalation unit risk
    - Nickel – inhalation risk - we do not agree with using the nickel refinery dust or nickel sub-sulphide slope factor for incinerator emissions; nickel sub-sulphide will not be present in incinerator emissions
    - Nickel – oral – as JW is well aware, MOE feels that there are updated/new TRVs for nickel that should be used; we disagree, however this should be addressed
    - Pentachlorophenol – need to explain the basis for doing route-to-route extrapolation for non-carcinogenic endpoints but not for carcinogenic endpoints
    - PCDD/F – a more comprehensive toxicological profile is suggested, especially given the issues related to carcinogenicity and mechanism of action. The latest HC number is 2.3 pg/kg/day; not 2.0 pg/kg/day. The following text is taken from our Gagetown assessment and might be useful:

      "In the Priority Substance List assessment, Health Canada concluded that there is no adequate demonstration that human populations exposed to dioxins and furans have suffered excess cancer. However, based on the results of studies in animals, it was assumed that chlorinated dioxins and furans are non-genotoxic carcinogens and reproductive toxicants with a

threshold, and therefore a tolerable daily intake for human exposure was derived (CEPA, 1990). Based on JECFA (2001); Health Canada (2004b; 2005) has most recently adopted a tolerable level of 70 picograms per kilogram body weight per month or approximately 2.3 picograms per kilogram of body weight per day. The Health Canada tolerable daily intake or TRV of 2.3 pg TEQ/kg body weight/day has been adopted for the current assessment." [Health Canada. 2005. Factsheet: It's Your Health: Dioxins and Furans. Available at http://www.hc-sc.gc.ca/iyh-vsv/environ/dioxin_e.html. Accessed on April 25, 2006.]

- PCBs – we do not like the handling of PCBs; an explanation as to why dioxin-like congeners were not considered should be provided. If PCDD/F is not evaluated using a slope factor then PCBs should not be evaluated in that way. Why was the HC total PCB TRV not given any consideration?

2.0 Ecological Risk Assessment

Overall, the Ecological Risk Assessment (ERA) is well written and concise. All of the major components of a typical ERA (including problem formulation, exposure assessment, toxicity assessment, risk characterization and uncertainty analysis) have been presented.

In a number of situations, we were unable to re-create the exposure calculations given the data and information provided. This does not automatically indicate that the modelling techniques are incorrect, but rather a number of the exposure point concentrations could not be recreated with the information provided. The report would benefit from a final QA/QC review of the input parameters, assumptions and calculations, and a review of the accompanying text.

The scope of the peer review of the ERA included an overall review of the report, with particular focus on: review of the chemical of potential concern (COPC) selection process; review of the valued ecosystem components (VECs) selected for study and spot-checking of parameter values within the exposure model for selected VECs; review of exposure pathways; spot-checking of uptake models used in the exposure model; review of the TRV selection and development methods; and review of whether the conclusions are supported by the ERA results. Comments are subdivided according to these topics.

2.1 Chemicals of Potential Concern

The approach identified in Section 2.0 and Section 6.3.1 for the identification of chemicals of potential concern (COPC) does not account for issues specific to ecological receptors. Screening of COPCs for human health will capture many of the most ecologically-relevant COPCs, but possibly not all of them. A similar screening could be conducted for ecological receptors, using ecotoxicity data.

Section 6.3.1. Sulphur dioxide could be evaluated for impacts on terrestrial plants.

2.2 Valued Ecosystem Components

The Valued Ecosystem Components (VECs) selected for assessment are good representatives of various trophic levels and food chains. The evaluation of fish, terrestrial plants, soil invertebrates and benthic invertebrates at the community level is sufficient. The selection of shrew and vole (herbivorous and insectivorous small mammals), robin (omnivorous bird), muskrat (herbivorous aquatic mammal), and fox and hawk (carnivorous mammal and bird) covers most food chains. However, the aquatic food chains are not well represented. The muskrat was selected to cover ingestion of aquatic plants, benthic invertebrates and fish. However, 80% of its diet is aquatic plants (so it is a good representative of this pathway) while only 2.5% of its diet is fish and 2.5% of its diet is benthic invertebrates. Therefore, these latter two food-chain pathways are not adequately assessed. ERAs typically evaluate a benthivorous bird (e.g., a breeding female mallard) and a piscivorous bird and mammal (e.g., great blue heron or kingfisher, and mink). Alternate VECs could be selected as appropriate to the region to address these pathways.

It is noted that predicted incremental risks via the ingestion of benthic invertebrates and fish would not approach an HQ=1 even if a diet of 100% benthic invertebrates or 100% fish was assumed for the muskrat (or a similar small mammal). However, mink are known to be more sensitive to some contaminants (e.g., Hg, PCBs) than other small mammals, and the assessment of risks to small mammals does not address potential risks to birds.

Selected VEC parameter values were reviewed:

- the meadow vole dry soil ingestion rate could not be verified;
- the muskrat diet assumptions are acceptable. However, a reference should be provided, or it should be stated that the breakdown of food items is an assumption;
- red fox food intake rate could not be verified, and appears higher than those provided in the cited references.
- for all species, references should be provided for food, water and soil intake rates as well as diet composition. Some parameter values appear to come from Sample and Suter, 1994 (and if so, this should be stated).

2.3 Exposure Pathways

The ERA has assessed relevant pathways, and excluded pathways that are insignificant, with one exception. The rationale for exclusion of inhalation as a pathway is not well supported. There is insufficient toxicity data to assess risks to birds from this pathway. However, data exist for small mammals for some chemicals. It would be sufficient to assess only a small mammal (with a small home range), placed at the MPOI. Relevant inhalation toxicity data should be used (e.g., reproductive effects or survival, not cancer or lung irritation) and oral data should not be used to assess inhalation exposures. Also, oral and inhalation exposures should not be summed, but rather assessed separately. In addition, oral and inhalation HQs should not be summed. The oral and inhalation risk estimates should be kept separate using relevant exposures and TRVs for each.

2.4 Uptake Models

The main report (Section 6.4.2) would benefit from additional discussion of the uptake models used, specifically, discussion of the use of regression models in addition to uptake factors. The current text only mentions uptake factors. This raised questions until reviewing

Appendix H where the regression models are described. We recommend that the main text of the report include discussion of regression models, because there is greater scientific support for using these models, where available.

Appendix H, Page 6, Section H.2.1. Biotic Uptake Factors. Ensure that the models used to estimate COPC uptake into plants and fish (from the HHRA) are relevant for the ERA (e.g., fish fillet vs. whole body fish, relevant plant parts that have not been washed). The plant type and part (e.g., root vs. shoot) should be specified particularly for metals where the models are not the same.

A brief review of the model spreadsheet provided by Jacques Whitford revealed that a number of factors (e.g., bioavailability and metabolic factors, Koc) were incorporated into the exposure point concentration calculations. These factors are mentioned in the Appendix H text. However, the values selected for these factors are not given for each COPC, which may explain why we were unable to reproduce the exposure point concentrations based on the information provided in the report. All parameter values should be listed within the report, so that exposure and risk calculations can be verified.

Values for uptake factors should be provided. For example, for values taken from Table 11 of Sample et al., 1998, it should be noted whether the mean, maximum, median or some other value was used (as was done for chromium and nickel).

2.5 TRV Selection

Secton 6.5.2. A comment is made on page 65 that “It is recognized that not all ecological risk assessors scale for body weight, however, it was determined that this was appropriate for this generic ERA.” The point is not that not everybody does body weight scaling. The point is that there is no scientific support for using body weight scaling for developing chronic TRVs. Dr. Brad Sample, who developed the first set of wildlife TRVs in 1994, recommends against using dose scaling specifically for development of chronic TRVs. However, it is acknowledged that dose scaling has been used in the past, and generally is still supported by regulatory agencies (i.e., an ERA using dose scaling likely still will be approved by federal and provincial regulators in Canada). However, the proponent is encouraged to use alternate methods to derive chronic TRVs in future.

In several instances, mammalian data were used to derive avian TRVs. There is no scientific support for this extrapolation. Physiology is very different between these classes. Assessing birds using mammalian data is implying we know about the potential risks to birds, when in fact we are simply assessing another generic mammal. It is not necessary to do this, since the exposure pathways for birds are already assessed using mammals. That is, the ERA already is assessing herbivorous, insectivorous and carnivorous mammals (vole, shrew, fox). Why assess the robin and hawk (omnivorous and carnivorous birds), which consider the same exposure pathways, if you are using the same TRV? You are gaining no new knowledge, and are implying we know more than we do. Rather, simply do not evaluate risks to birds from these chemicals, and acknowledge this as an uncertainty. If your HQs are more than 3 orders of magnitude lower than 1, then an argument could be made that birds would not be expected to be 1000+ times more sensitive than the mammals. In addition, there are avian data for some chemicals for which mammalian data were used (e.g., cobalt). A search should be conducted to determine whether avian data are available for use in TRV development.

Various uncertainty factors were used to extrapolate from acute to chronic data, and NOAELs to LOAELs. Acute data should only be used to assess acute exposures. The mechanism of toxicity for acute doses often is quite different from that for chronic doses. Therefore, unless information is available on acute-chronic ratios, this extrapolation is not supported. Having said that, this extrapolation has been done in ERAs in the past, and likely will be accepted by regulatory agencies at this time. However, the proponent is encouraged to use alternate methods to derive chronic TRVs in future.

Another common approach used in the past, which generally is considered conservative, is to extrapolate from LOAELs to NOAELs by applying an uncertainty factor of 10. In this assessment, an uncertainty factor of 5 was used to extrapolate from a NOAEL to a LOAEL. This is not necessarily conservative, because in many cases, there is only a two-fold difference between a NOAEL and a LOAEL. Thus, this reverse extrapolation is not conservative. To avoid this uncertainty and potential underestimate in risk, a preferred approach is to use the NOAEL and acknowledge the conservative nature of the TRV.

TRVs for mammals for several of the PAHs were based on a carcinogenicity endpoint. Cancer is not evaluated in ERA. The relevance of this endpoint to the assessment endpoints should be discussed, or alternate TRVs selected for use in the ERA.

Several TRVs were spot-checked to see if they were calculated or selected correctly. The arsenic TRV for vole has been calculated correctly using the methods described in the ERA. It is noted that the generic scaling factor was used (0.94) from Sample and Arenal (1999), although an arsenic trioxide scaling factor of 0.874 is available from this paper. This would not have any significant influence on the resulting TRV, or the results or conclusions of the ERA.

Table H.15 Benchmark Values for Phytotoxicity Assessment. The Chromium (total) value was incorrectly used for Chromium III even though there is a value for Chromium III in MOE 1996.

Section 6.6.1. It is agreed that HQs should not be summed unless the chemicals have the same target organ and mode of action. The ERA summed the HQs for PAHs. However, the endpoint selected for the TRVs for various PAHs were different (cancer, mortality, reproduction). The relevance of summing HQs derived from these studies should be considered. Having said that, summing the HQs does result in a conservative Hazard Index, as is discussed in this section.

2.6 Support for Conclusions

The assumptions, models and calculations used for the ERA were spot-checked by Intrinsik and found generally to be correct. Once the issues identified in this review are addressed, the risk assessment should be acceptable. The conclusions are well supported by the analysis. We agree with the recommendation that several chemical/VEC combinations will require particular attention in any site-specific risk assessment (e.g., dioxin exposure to aquatic VECs).

Minor Comments

- Page 17, last sentence, do HQ values really indicate a probability of occurrence?
- Page 20, Section 3.2.5. A HQ value of 1.03 would necessarily be considered to exceed a value of 1.0.
- Figures 3-7 and 3-8. It is unclear why SO_2 is not addressed in these tables.
- Section 6.3 Problem Formulation. This section would benefit from mention of plants, invertebrates and aquatic VECs. It currently reads as though the ERA is assessing only terrestrial wildlife.
- References. The reference section (pg 49) is incomplete and not in alphabetical order.
- Appendix H, page 10, third last line. This sentence refers to “Table 3.4”. However, this table is not within the appendix.

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1. Table C.1 (of Appendix C) indicates that a mixing zone of 10 cm was used to derive soil concentrations under both tilled and untilled soil conditions. The rationale provided states that a mixing zone of 10 cm is "supported by studies which indicated that the highest soil concentrations from surficial deposition may be 10 to 15 cm below surface". No references or studies have been provided to support this claim. Secondly, the rationale field indicates that "recommended values range from 1 cm (unfurrowed) to 20 cm (furrowed); however, since some leaching of deposited particulates will occur, a value of 10 is being used." A review of the U.S. EPA, 2005 document does not indicate a recommended range of soil depths. It mentions that tilled soil depths are approximately 10 to 20 cm, depending on the local conditions; however, it does not recommended a range of soil mixing zones. This parameter/assumption is very important and can have a significant influence on the overall outcome of the assessment. The U.S. EPA 2005 recommends a mixing zone for untilled soils of 2 cm and 20 cm for tilled soils. Untilled soil concentrations are typically used to assess direct soil contact pathways (e.g., soil ingestion and dermal contact). By using a mixing zone of 10 cm (instead of 2 cm), the assessment has potentially under predicted direct soil contact exposures by 5 fold while potentially over predicting root-uptake related pathways by 2 fold. If this assumption is to be used in the risk assessment, it warrants a significant discussion in the main report to justify why the U.S. EPA 2005 recommended mixing zones	Jacques Whitford has adopted a mixing zone of 10 cm for use in this risk assessment. The US EPA recommends a value of 1 cm for exposures including direct ingestion of soil and surface water runoff in non-agricultural (i.e., untilled) areas. They recommend a value of 20 cm for exposures including plant uptake and surface water runoff in agricultural areas. Jacques Whitford feels it appropriate to use a value of 10 cm because we would expect some downward migration of the CoPCs into the soil over the 35-year operating lifetime of the facility. A value of 10 cm is more consistent with the root uptake zone of the modeled vegetation, which tends to be a more important pathway in the overall risk to both human and ecological receptors than the ingestion of soil. The majority of the uptake of the CoPC and subsequent exposure is from environmental receptors that would be exposed to tilled soil, such as garden produce and crops.

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should not be used.	
2. Figure (Table) 2-2 – Missing B(a)P, B(a)F and B(a)A from the B(A)P group; if only Cr(III) is considered, then the exclusion of Cr(VI) and/or Cr(total) should be explained.	Table 2-2 is now consistent with Table 5-3, which shows the benzo(a)pyrene group, containing all eight carcinogenic PAHs, as defined by the WHO. In addition, the Chromium modeled is Total Chromium. All tables have been updated to rectify these inconsistencies. Justification as the selection of TRVs is provided in Appendix B, Toxicity Profiles.
3. Figure (Table) 3-2 – MPOI locations require some explanation: 1-hour – vapour and particulate are in same location, Scenario 1 and 3 are the same while Scenario 2 is different; 24-hour - all in same location; Annual - Scenario 1 and 2 are the same, Scenario 3 is different. This information is not intuitive and should be explained.	The locations provided in the original air modelling tables were incorrect and have been corrected in the final report.
4. Page 30. Section 5.1.1 indicates that the receptor age classes (i.e., infant, toddler, child, teen, and adults) and associated physical characteristics (e.g., body weight, inhalation rates, etc) recommended by Health Canada (2004) were used, in part, to facilitate the exposure assessment. Appendix A reports that produce intake rates from the U.S. EPA Exposure Factors Handbook (EFH) were used. A number of significant comments follow:	In general, text updates have been made to Section 5.1 to clarify the source of the intake/ingestion/consumption rates.
i. Although the EFH table number is provided, we were not able to find any indication as to which age class, percentile, region or season was used to develop age-specific vegetable intakes. It is noted that the receptor age classes reported by the U.S. EPA EFH differ from those of Health Canada.	As now indicated in Section 5.1.3.4, the mean values (for both sexes combined, if applicable) were selected from the Tables referenced in Appendix A for each Health Canada age class. Where age groupings in the EFH were different than those assumed in this risk assessment, the highest reported intake for any of the age groups reported in the EFH falling within our assumed age categories was used.

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ii. The U.S. EPA EFH reports per capita intakes of exposed vegetables (e.g., Table 9-9) as g/kg-day. It appears as though the unit conversion between g/kg-day and kg/day (as reported in Appendix A.2 for example) may be incorrect or calculations are occurring that have not been reported.	The human health model used needs to have the ingestion rates in either kg (dry weight) or kg (wet weight) (depending on the food type and the necessary input to the equation) per day. Therefore, the number as presented in Appendix A.2 may have been adjusted based on moisture content. In addition, the intake is multiplied by the receptor's body weight to obtain a kg (as consumed) per day unit. This body weight adjustment is then divided back out when the receptor's intake (chronic daily or lifetime average daily) is calculated, making the body weight adjustment mathematically null. Therefore, we have not technically 'corrected' for body weight and the EFH numbers are used as was their intention.
iii. As the U.S. EPA EFH points out, intake rates are reported as g/kg/day and correcting for body weight to derive an intake on a g/day or kg/day basis using a single body weight (such as 16.5 kg for a toddler as reported by Health Canada) is not appropriate. The intake rates reported by the U.S. EPA EFH were indexed over the body weights of the actual survey respondents.	
iv. The use of data presented in Table 13-71 is considered appropriate. It should be noted, however, that the fractions selected for use in the assessment are consumer only fractions (i.e., those who did not report gardening were excluded from the dataset). Although this is conservative, it was considered appropriate in the derivation of a reasonable maximum exposed individual.	Due to the conservative, generic nature of this risk assessment, we agree that the use of these values is appropriate.
v. The EFH cautions that consumption rate data are based on short-term observations (i.e., 7 days) and therefore are not appropriate for use in long-term exposure assessments. This is particularly true for home produced vegetables and fruits since consumption rates would be highly correlated to season (i.e., spring, summer, fall and winter). As a result, the U.S.	Using a short-term recall survey rate over the entire year likely overestimates a receptor's actual intake, adding to the conservatism of the model. A quick comparison of the total adult intake of vegetables (above and below-ground) in our model (2.866 g/kg-d) to the mean total intake of consumer only intake of homegrown vegetables for the Northeast region (Table 13-14 EFH) (1.78 g/kg-d)

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EPA EFH attempted to derive a long-term distribution of the average daily intake rates of home produced foods from the short-term data available for major food groups (vegetables, fruits and meats). The seasonally adjusted distributions for a given region (e.g., the north eastern region) were derived by averaging the intake rates for each of the four seasons (spring, summer, winter and fall). The assessment may consider comparing the home garden vegetable consumption rates used in the assessment with the seasonally adjusted intakes reported by the EFH.	illustrates this point. The seasonally averaged values were not appropriate for use in our assessment, as we evaluated food intake on an age-group basis, while these values are provided simply by region and season (or total).
5. Section 5.22 provides a table of "relative bioavailability" values. No discussion or definition of the term "relative" bioavailability could be found in the text. Secondly, the relative oral bioavailability values reported in Table (or Figure) 5-7 are not consistent with those reported in several of the appendices. It is unclear which values were actually used in the exposure/risk calculations.	The values present in the draft report for oral and inhalation exposures were actually absolute bioavailabilities, not relative. In addition, all bioavailability values (with the exception of dermal) have since been conservatively set to 1.0, assuming everything is 100% bioavailable. Relative dermal bioavailabilities were taken from Health Canada (2004a).
6. Section 5.4.2 provides the general equation of a Hazard Quotient (HQ); however, the equation used in Appendix D (i.e., the actual methods) differs from this. Appendix D (Section D.3.2) indicates the use of a "reference dose absorption factor" on the bottom of the HQ. We were unable to easily re-create the HQ for lead and nickel as a result of soil/dust ingestion (Table F.2). It appears that there may be some confusion surrounding the application of RAF values in the derivation of the final HQ value. From the information provided, it is unclear if the assessment intended to consider chemical, media and route-specific RAF values.	The equations presented in the main body of the report are meant to be general, while the detailed, method-specific equations are presented in Appendix D. Our human health model is set up to use absolute bioavailabilities so that a route-of-exposure bioavailability is used to adjust the intake of a specific CoPC, and a bioavailbility specific to the TRV selected is used to adjust the TRV accordingly. All oral and inhalation bioavailbilities (both intake and TRV) were assumed to be 1.0 for the purposes of this assessment. As detailed previously, the relative bioavailibilities were taken from Health Canada, 2004a.

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7. Page 9. Figure (or Table) 3-3. Section 3.1.1 indicates that the "Guideline A-7 emission concentrations limits were used as default exhaust stack air emission estimates for eight pollutants contained in the guideline to evaluate the potential risk to the surrounding environment." If the A-7 emission limits were used, why do the maximum COPC concentrations presented in Table 3-3 change from one scenario to the next (i.e., 4000,000 t/y; 266,666 t/y and 133,33 t/y) for these eight COPCs? Should the concentrations not be the same under all scenarios for these eight compounds since you've used the A-7 limits, which are maximum emission limits for a facility of this nature?	The Guideline A-7 emission concentration limits were used as default exhaust stack air emissions for eight pollutants contained within the guideline. The rate (t/y) at which the emissions are emitted is not the same as the concentration within the emissions plume. The emission concentrations are the same for all three scenarios, only the rate changes, depending on the operating scenario modeled.
8. Page 24 provides a discussion of typical Ontario background concentrations of COPC in soil and the general approach used to predict facility related COPC concentrations in soil. The assessment notes that typical Ontario soil concentrations are not "site specific background" and that local background soils should be collected during a site-specific assessment so that cumulative effects can be evaluated. Section 5.6.2.3 does indicate that background concentrations were not incorporated into the current study, however, it should be clearly stated (perhaps within another location of the report) that the current study does not consider cumulative health risks but rather facility related risks only. The same type of exercise (i.e., a comparison of modelled versus ambient) could be conducted with air data collected from within Ontario.	This has been updated in the text in a number of locations. Again to reiterate, background soil concentrations were not included in this evaluation at a generic risk assessment. Therefore, other than for inhalation pathway for some chemicals, this risk assessment does not consider cumulative impacts.
9. Section 3.1.4 provides a very limited dataset of background air quality; data from other locations might be useful to supplement this dataset (e.g., Dann).	Given project time constraints an exhaustive review of ambient air quality in the area could not be determined, rather representative stations and timings were determined. This is something that will be covered in more detail in the site specific risk assessment.

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10. Figure (Table) 3-11 – Combustion gases: total pollutant concentrations for Scenario 1 vs. 2 vs. 3 seems incorrect; Scenario 1 columns do not add up (we believe the Scenario 1 Total Pollutant Concs are incorrect and that the Total Pollutant plus Bkg columns are correct).	This was a calculation error in excel and has been rectified in the final report.
11. Figure (Table) 5-3 – B(a)P group missing phenanthrene; missing DEHP; this table/figure should be consistent with Figure (Table) 2-2.	Phenanthrene is not considered a carcinogenic PAH by the WHO. As stated in the response to comment 2, all tables of CoPCs within the report have been corrected to eliminate any inconsistencies.
12. Section 5.2.4 – Additivity of Risks – not conservative to exclude the potential for this to occur; compounds acting on similar systems via similar toxicological mechanisms should be considered additive (i.e., we suggest grouping the chlorinated monocyclic aromatics together and consider additive).	Section 5.6.3.1 now presents a discussion on the additivity of specific CoPCs. Where it could be shown that the CoPCs had the same toxicological endpoint on the same target organ, the risks were summed.
13. Page 24. Indicates that soil loadings were modelled over a 35 year life expectancy of the facility. Appendix D (Equation D1.2.2) indicates 28 years.	This was a typo and has been corrected. Soil loadings were modeled over a 35 year life expectancy of the facility.
14. No references were provided with respect to the derivation of house-hold dust ingestion rates for infants, toddlers and children. The assessment has appropriately used the recommended soil/dust ingestion rates provided by Health Canada (2004); however, it appears to have derived an indoor dust ingestion intake (24.7 mg/d for toddler) based on the surface area of ½ a finger and “finger mouthing events” per hour. No citation for such an approach has been provided. Intrinsik is of the opinion that the soil/dust ingestion rate of 80 mg/day for a toddler (as recommended by Health Canada) inherently includes both outdoor soil and indoor dust.	The methodology was adopted from Appendix A (Table A-1) of Appendix B.5 of the MOE Rationale Document. The values for dust ingestion were added to the soil ingestion values in order to be conservative.
15. Using the information provided in Appendix D, C and the main report, we were unable to recreate the soil concentration for lead	Jacques Whitford does not set the loss constant, kse, to zero. It is calculated using the recommended equation, as indicated in

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under scenario 1. Appendix E (table E.1) reports a concentration of lead in soil of 9.73E-03. Although our estimate of 7.1E-03 is very close, we cannot explain the difference. It should be noted that Appendix D does not indicate that the U.S. EPA, 2005 recommends the soil loss constant due to erosion be set to zero. It is unclear if this loss constant has been evaluated in the current assessment and may be the reason for the disparity in values.	Appendix D (from the HHRAP).
16. Using the concentration of lead in soil (as reported in Table E.1), the equation in Appendix D (Section D.2.3.5), and the data provided in Table A.2, we were unable to recreate the chronic daily intake of lead for the residential Toddler (Soil/Dust – Summer – Outdoor) as reported in Table F.2. We were unable to recreate the lead concentration in wild game as reported in Table E.1. It is noted that we were able to recreate the concentration of lead in aboveground and belowground produce due to deposition and root uptake, given the reported concentration of lead in soil.	During the peer review meeting it was agreed that although values could not be exactly replicated that overall conclusions in the report are considered valid. It is believed that now updates have been made to the modelling it is likely that these calculations could be replicated.
17. Appendix D makes reference to unitized yearly deposition rates and chemical-specific emission rates during the derivation of the deposition term. It is noted that actual unitized deposition rates (s/m2-yr) were not found in either the risk assessment or the air modeling document. The risk assessment already reports annual average chemical-specific deposition rates (g/m2) and, therefore, there would be no need for the emission rate parameter Q (g/s).	This was a typo and has been corrected. Deposition rates were not unitized and, as our model is set up for both unitized and non-unitized deposition rates, for the purposes of this assessment, the emission rate parameter (Q) was set to 1 as it has already been taken into account in the deposition and concentration rates presented by the air modelers.
18. Exposure and risk calculations were confirmed for an infant's exposure to dioxins via the consumption of breast-milk. It is noted, however, that the RA appears to reference the 1998 draft U.S. EPA HHRA Protocol rather then the final 2005 Protocol. A comparison of the risk assessment methods from 1998 versus the final 2005 protocol identified a number of potential minor	The references have been updated to the appropriate protocol (2005). The equations used are consistent with the protocol for dioxins and furans; however, there was a slight difference in the set up within the model to make the intake calculation consistent with the equation used for all the other organics. The only variation from the US EPA (2005) equations was the use of a density factor. The US

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deviations. The breast milk consumption rate recommended in the 2005 protocol differs slightly from what has been used.	EPA (2005) assume a density of breast milk equal to 1 g/mL (or 1 kg/L); however, Jacques Whitford has used a value of 1.03 (Environmental Health Directorate. 1998. Exposure factors for assessing total daily intake of Priority Substances by the general population of Canada. Priority Substances Section, Bureau of Chemical Hazards, Health Canada, Ottawa, Ontario. March 1998.). These slight variations have been noted in Appendix D. In addition, the breast milk consumption rate as provided by the US EPA (2005) assumes a 12-month weighted average. We have chosen to adopt a value of 0.742 mL/d, which is representative of an exclusively breast fed infant over the first 6 months of age, consistent with the Health Canada definition of an infant. US EPA (2005) also assume a 90% absorption rate (absorption factor of 0.9) of ingested dioxins and furans and PCBs. We have conservatively assumed an absorption factor of 1.0 for all CoPCs.
19. The discussion on page 51 speaks to the conservatism of selected fish consumption rates recommended for use by Health Canada (2004). These data are cited as coming from the Canadian Compendium of Exposure Factors (Richardson, 1997). The Richardson (1997) consumption rates for non-native populations include a wide variety of fish products (including canned salmon, tuna, shell fish, seafood, etc.). The assessment should point this out and discuss the applicability of these intake rates to the exposure scenario being developed.	Fish ingestion rates for the local residents and subsistence farmers were taken from the EFH (US EPA, 1997). Although Richardson (1997) (as cited in Health Canada, 2004a) provides fish consumption rates for non-native populations, as pointed out, these rates include a wide variety of fish products, including marine fish (cod, haddock), canned salmon, tuna, and sardines, freshwater fish, and shrimp (fresh, frozen, and canned). As the intention of this assessment is to evaluate the risks arising from the consumption of fish caught within the modeled watershed, these values were not considered appropriate. Mean fish intake rates for individuals who eat fish and reside in households with recreational fish consumption from the EFH were used. Fish ingestion rates for the First Nations and Métis receptors were taken from Health Canada (2004a), but it should be noted that these are not specific to Ontario First Nations.

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20. Appendix B (TRVs) • In many cases, only the selected value is provided; however when an IRIS value is available but not used, it should still be discussed. A good example is nickel, IRIS is used for oral; however, IRIS numbers are not even addressed for inhalation. • Dichloromethane – should include discussion of EPA slope factors/RfDs • Formaldehyde – should include discussion of EPA inhalation unit risk	The Toxicity Profiles (Appendix B) have been updated to include a discussion of all potentially relevant TRVs.
• Nickel – inhalation risk - we do not agree with using the nickel refinery dust or nickel sub-sulphide slope factor for incinerator emissions; nickel sub-sulphide will not be present in incinerator emissions	Specific to nickel, Jacques Whitford has decided not to use a nickel slope factor for incinerator emissions. Nickel is no longer assessed on a carcinogenic basis.
• Nickel – oral – as JW is well aware, MOE feels that there are updated/new TRVs for nickel that should be used; we disagree, however this should be addressed	Jacques Whitford agrees that the oral RfD for Ni should remain as 20 ug/kg-d and not any updated factor. Thus this remains the same in the report.
• Pentachlorophenol – need to explain the basis for doing route-to-route extrapolation for non-carcinogenic endpoints but not for carcinogenic endpoints	Agreed, for pentachlorophenol, Jacques Whitford has assumed an inhalation cancer slope factor equal to that of the oral cancer slope factor.
• PCDD/F – a more comprehensive toxicological profile is suggested, especially given the issues related to carcinogenicity and mechanism of action. The latest HC number is 2.3 pg/kg/day; not 2.0 pg/kg/day. The following text is taken from our Gagetown assessment and might be useful: "*In the Priority Substance List assessment, Health Canada concluded that there is no adequate demonstration that human populations exposed to dioxins and furans have suffered excess cancer. However, based on the results of*	The most recent Health Canada guideline (2.3 pg/kg-d) has been adopted for use in this assessment.

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studies in animals, it was assumed that chlorinated dioxins and furans are non-genotoxic carcinogens and reproductive toxicants with a threshold, and therefore a tolerable daily intake for human exposure was derived (CEPA, 1990). Based on JECFA (2001); Health Canada (2004b; 2005) has most recently adopted a tolerable level of 70 picograms per kilogram body weight per month or approximately 2.3 picograms per kilogram of body weight per day. The Health Canada tolerable daily intake or TRV of 2.3 pg TEQ/kg body weight/day has been adopted for the current assessment." [Health Canada. 2005. Factsheet: It's Your Health: Dioxins and Furans. Available at http://www.hc-sc.gc.ca/iyh-vsv/environ/dioxin_e.html. Accessed on April 25, 2006.] • PCBs – we do not like the handling of PCBs; an explanation as to why dioxin-like congeners were not considered should be provided. If PCDD/F is not evaluated using a slope factor then PCBs should not be evaluated in that way. Why was the HC total PCB TRV not given any consideration?	Further to this comment, Jacques Whitford has decided not to evaluate PCBs using a slope factor. In addition, the Health Canada Total PCB TRV was adopted for assessment of non-carcinogenic endpoints. It is this value the Food Protectorate Branch uses in evaluation of PCB risk to Canadians from food.
Chemicals of Potential Concern The approach identified in Section 2.0 and Section 6.3.1 for the identification of chemicals of potential concern (COPC) does not account for issues specific to ecological receptors. Screening of COPCs for human health will capture many of the most ecologically-relevant COPCs, but possibly not all of them. A similar screening could be conducted for ecological receptors, using ecotoxicity data.	The CoPCs evaluated in this ERA were selected based on recommendations and guidelines from the Ontario Ministry of the Environment (1999, 2004), as well as an emissions inventory from the KMS-Peel EFW facility. Based on these sources, the CoPCs evaluated in this ERA are believed to encompass the major toxicants which would be expected to effect ecological health from this facility. A future site-specifc risk assessment should involve a CoPC

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Section 6.3.1. Sulphur dioxide could be evaluated for impacts on terrestrial plants.	screening which is specific to the differences in sensitivity between humans and wildlife. JW agrees with the peer reviewers comment, and has incorporated Sulphur dioxide emission into the phytotoxicity assessment.
Valued Ecosystem Components The Valued Ecosystem Components (VECs) selected for assessment are good representatives of various trophic levels and food chains. The evaluation of fish, terrestrial plants, soil invertebrates and benthic invertebrates at the community level is sufficient. The selection of shrew and vole (herbivorous and insectivorous small mammals), robin (omnivorous bird), muskrat (herbivorous aquatic mammal), and fox and hawk (carnivorous mammal and bird) covers most food chains. However, the aquatic food chains are not well represented. The muskrat was selected to cover ingestion of aquatic plants, benthic invertebrates and fish. However, 80% of its diet is aquatic plants (so it is a good representative of this pathway) while only 2.5% of its diet is fish and 2.5% of its diet is benthic invertebrates. Therefore, these latter two food-chain pathways are not adequately assessed. ERAs typically evaluate a benthivorous bird (e.g., a breeding female mallard) and a piscivorous bird and mammal (e.g., great blue heron or kingfisher, and mink). Alternate VECs could be selected as appropriate to the region to address these pathways. It is noted that predicted incremental risks via the ingestion of benthic invertebrates and fish would not approach an HQ=1 even	JW agrees with the peer review comments. The aquatic food chain was not adequately represented by the VECs selected in this ERA. To remedy this shortcoming, three additional VECs are now being evaluated. The muskrat, which consumes a large proportion of aquatic vegetation; the Mallard, whose diet is largely comprised of aquatic invertebrates; and the Belted kingfisher, which consumes a highly piscivorous diet.

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if a diet of 100% benthic invertebrates or 100% fish was assumed for the muskrat (or a similar small mammal). However, mink are known to be more sensitive to some contaminants (e.g., Hg, PCBs) than other small mammals, and the assessment of risks to small mammals does not address potential risks to birds. Selected VEC parameter values were reviewed: • the meadow vole dry soil ingestion rate could not be verified; • the muskrat diet assumptions are acceptable. However, a reference should be provided, or it should be stated that the breakdown of food items is an assumption; • red fox food intake rate could not be verified, and appears higher than those provided in the cited references. • for all species, references should be provided for food, water and soil intake rates as well as diet composition. Some parameter values appear to come from Sample and Suter, 1994 (and if so, this should be stated).	Parameters such as food intake, water intake, etc. are derived using USEPA and/or CCME supported equations. Percentage estimates of dietary components are obtained using a weight of evidence approach which relies on multiple sources of information. This approach is required due to the extreme variability in diet which is seen in most wildlife species. The opening paragraph of the VEC descriptions section of Appendix H (section H.1.1) has been modified to clarify these issues.
Exposure Pathways The ERA has assessed relevant pathways, and excluded pathways that are insignificant, with one exception. The rationale for exclusion of inhalation as a pathway is not well supported. There is insufficient toxicity data to assess risks to birds from this pathway. However, data exist for small mammals for some chemicals. It would be sufficient to assess only a small mammal (with a small home range), placed at the MPOI. Relevant inhalation toxicity data should be used (e.g., reproductive effects or survival, not cancer or lung irritation) and oral data should not	The inhalation pathway is not explicitly evaluated in this ERA. JW acknowledges that given the nature of this facility, inhalation exposure is probable for many VECs. Given the state of knowledge regarding inhalation risks to ecological receptors, JW chose to rely on the conclusions of the inhalation toxicity aspect of the HHRA as a measure of potential inhalation risk to wildlife. Due to the conservatism of the approach used in the HHRA, the absence of unacceptable risk to human health, was taken to be an indication

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be used to assess inhalation exposures. Also, oral and inhalation exposures should not be summed, but rather assessed separately. In addition, oral and inhalation HQs should not be summed. The oral and inhalation risk estimates should be kept separate using relevant exposures and TRVs for each.	that ecological receptors would be adequately protected from this pathway also. The ERA has been modified to clarify the decision not to explicity assess inhalation risks in the ERA. Future site-specific risk assessments should strive to evaluate the inhalation risks to ecological receptors.
The main report (Section 6.4.2) would benefit from additional discussion of the uptake models used, specifically, discussion of the use of regression models in addition to uptake factors. The current text only mentions uptake factors. This raised questions until reviewing Appendix H where the regression models are described. We recommend that the main text of the report include discussion of regression models, because there is greater scientific support for using these models, where available.	The uptake factors and regression models used towards the derivation of EPCs are discussed in detail in the ERA Appendix (App. H). Given the complexity of the calculations and rationale for these models, the discussion of these aspects was felt more suitable for the Appendix. In the main report, the reader is directed to the appendix for detailed information on the calculations and methods followed in the exposure assessment.
Appendix H, Page 6, Section H.2.1. Biotic Uptake Factors. Ensure that the models used to estimate COPC uptake into plants and fish (from the HHRA) are relevant for the ERA (e.g., fish fillet vs. whole body fish, relevant plant parts that have not been washed). The plant type and part (e.g., root vs. shoot) should be specified particularly for metals where the models are not the same.	The uptake factors used for estimating CoPC uptake to plants and fish estimate the concentrations that are relevant to the ERA. Fish concentrations are whole body values, and are provided (and discussed) in Appendix D. Plant concentrations are conservatively based on the maximum concentration of either roots, shoots, or fruits (Appendix D).
A brief review of the model spreadsheet provided by Jacques Whitford revealed that a number of factors (e.g., bioavailability	JW agrees with the comments by the peer reviewer regarding the ability to reproduce numbers. Initial drafts of the report did not

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and metabolic factors, Koc) were incorporated into the exposure point concentration calculations. These factors are mentioned in the Appendix H text. However, the values selected for these factors are not given for each COPC, which may explain why we were unable to reproduce the exposure point concentrations based on the information provided in the report. All parameter values should be listed within the report, so that exposure and risk calculations can be verified. Values for uptake factors should be provided. For example, for values taken from Table 11 of Sample et al., 1998, it should be noted whether the mean, maximum, median or some other value was used (as was done for chromium and nickel).	contain adequate information to generate many of the EPCs. In response to these comments, the Exposure Assessment section of the appendix (App. H) has been modified. All relevant equations, metabolic factors, bioavailability factors, rationale, etc. have been added. Verification of the numbers used in the ERA is now possible. Values for all point estimates of uptake factors are now provided in Tables presented in the Appendix. The source of these values are presented in the accompanying text.
TRV Selection Secton 6.5.2. A comment is made on page 65 that "It is recognized that not all ecological risk assessors scale for body weight, however, it was determined that this was appropriate for this generic ERA." The point is not that not everybody does body weight scaling. The point is that there is no scientific support for using body weight scaling for developing chronic TRVs. Dr. Brad Sample, who developed the first set of wildlife TRVs in 1994, recommends against using dose scaling specifically for development of chronic TRVs. However, it is acknowledged that dose scaling has been used in the past, and generally is still supported by regulatory agencies (i.e., an ERA using dose scaling likely still will be approved by federal and provincial regulators in Canada). However, the proponent is encouraged to use alternate methods to derive chronic TRVs in future.	JW acknowledges the comments of the reviewer. Although the strategy of applying body weight scaling for the purpose of generating TRVs can be questioned, it remains widely supported by the regulatory agencies. Currently, a scientifically supported alternative to the Sample and Arenal (1999) equations does not exist.

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In several instances, mammalian data were used to derive avian TRVs. There is no scientific support for this extrapolation. Physiology is very different between these classes. Assessing birds using mammalian data is implying we know about the potential risks to birds, when in fact we are simply assessing another generic mammal. It is not necessary to do this, since the exposure pathways for birds are already assessed using mammals. That is, the ERA already is assessing herbivorous, insectivorous and carnivorous mammals (vole, shrew, fox). Why assess the robin and hawk (omnivorous and carnivorous birds), which consider the same exposure pathways, if you are using the same TRV? You are gaining no new knowledge, and are implying we know more than we do. Rather, simply do not evaluate risks to birds from these chemicals, and acknowledge this as an uncertainty. If your HQs are more than 3 orders of magnitude lower than 1, then an argument could be made that birds would not be expected to be 1000+ times more sensitive than the mammals. In addition, there are avian data for some chemicals for which mammalian data were used (e.g., cobalt). A search should be conducted to determine whether avian data are available for use in TRV development.	JW agrees with the comments of the peer reviewer. While the use of uncertainty factors to extrapolate mammalian toxicity to an equivalent avian value has been done in the past, there is little scientific evidence which supports this. For this ERA, in the absence of avian toxicity, TRVs will not be generated based on mammalian toxicity data. For those CoPCs which do not have avian toxicity available, the risks to avian species will no longer be evaluated. In response to the reviewers comments, a source of avian toxicity for Cobalt was identified (USEPA, 2005). A TRV was generated from this value and incorporated into the ERA.
Various uncertainty factors were used to extrapolate from acute to chronic data, and NOAELs to LOAELs. Acute data should only be used to assess acute exposures. The mechanism of toxicity for acute doses often is quite different from that for	There is only one CoPC, where acute toxicity is used to derive a TRV (avian TRV for Pentachlorophenol). JW agrees that chronic endpoints are preferred for the purpose of an ERA. However, in the absence of chronic toxicity, a UF of 30 is issued, and is considered

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chronic doses. Therefore, unless information is available on acute-chronic ratios, this extrapolation is not supported. Having said that, this extrapolation has been done in ERAs in the past, and likely will be accepted by regulatory agencies at this time. However, the proponent is encouraged to use alternate methods to derive chronic TRVs in future.	appropriate to conservatively assess toxicity.
Another common approach used in the past, which generally is considered conservative, is to extrapolate from LOAELs to NOAELs by applying an uncertainty factor of 10. In this assessment, an uncertainty factor of 5 was used to extrapolate from a NOAEL to a LOAEL. This is not necessarily conservative, because in many cases, there is only a two-fold difference between a NOAEL and a LOAEL. Thus, this reverse extrapolation is not conservative. To avoid this uncertainty and potential underestimate in risk, a preferred approach is to use the NOAEL and acknowledge the conservative nature of the TRV.	JW acknowledges the comments of the reviewer, and has modified the ERA accordingly. The preferred measure of toxicity for TRVs in this ERA is the chronic LOAEL. For certain CoPCs the only chronic endpoints available were NOAELs. In this situation, the NOAEL is used as the TRV (without the application of uncertainty factors). This method avoids overestimating the effects threshold (thus, underestimating potential risks). For mammalian VECs, NOAEL based TRVs were used for the following CoPCs: naphthalene, phenanthrene, 1,2-dichlorobenzene, barium, and inorganic mercury. For avian VECs, a NOAEL based TRV was used for vanadium.
TRVs for mammals for several of the PAHs were based on a carcinogenicity endpoint. Cancer is not evaluated in ERA. The relevance of this endpoint to the assessment endpoints should be discussed, or alternate TRVs selected for use in the ERA.	JW acknowledges the comments of the reviewer, and has modified the ERA accordingly. Inconsistencies were identified in the studies used to determine TRVs for some of the PAHs. It is agreed by JW that carcinogenicity in not a valid endpoint for basing TRVs for ecological health.
Several TRVs were spot-checked to see if they were calculated or selected correctly. The arsenic TRV for vole has been calculated correctly using the methods described in the ERA. It is	

DRAFT Regions of York and Durham Energy-from-Waste Generic Risk Assessment Prepared by Jacques Whitford Limited, dated May 25, 2007 Intrinsik Environmental Sciences Inc. Comments – June 8, 2007	
Comment	**Response**
noted that the generic scaling factor was used (0.94) from Sample and Arenal (1999), although an arsenic trioxide scaling factor of 0.874 is available from this paper. This would not have any significant influence on the resulting TRV, or the results or conclusions of the ERA.	
Table H.15 Benchmark Values for Phytotoxicity Assessment. The Chromium (total) value was incorrectly used for Chromium III even though there is a value for Chromium III in MOE 1996.	The value used for Chromium in the Phytotoxicity assessment was the proper value. The ERA evaluates the toxicity of total Chromium to ecological receptors (including plants). The phytotoxicity table referred to by the reviewer incorrectly listed trivalent Chromium as the CoPC. Chromium III has been replaced by total Chromium in the ERA.
Section 6.6.1. It is agreed that HQs should not be summed unless the chemicals have the same target organ and mode of action. The ERA summed the HQs for PAHs. However, the endpoint selected for the TRVs for various PAHs were different (cancer, mortality, reproduction). The relevance of summing HQs derived from these studies should be considered. Having said that, summing the HQs does result in a conservative Hazard Index, as is discussed in this section.	

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Environmental & Occupational Health +Plus

Health Impact Evaluation and Issues Management

June 10, 2007

To: Dr. Chris Ollson
Cc: Elliot Sigal
From: Lesbia F. Smith, MD

Re: Comments on Report: Energy from Waste Durham York
Generic Risk Assessment Feasibility Study Report # 1009497.02

Air dispersion modeling

This process requires inputs from three types of process data:

1. Actual functioning facility and actual stack measurements of contaminants; or
2. Actual functioning facility and actual atmospheric measurements of contaminants; or
3. proposed technology with phantom readings of contaminants as projected; or
4. Other hypothetical situation with a combination of real and phantom readings of contaminants; and
5. meteorological data

As a supporting document to the HHRA and ERA, it provides the inputs for calculating risks of exposure to each of the emissions pollutants at various levels of exposure (distance – up to 10 km here) and concentrations predicted. The risk will depend on selection of target organism (humans for example) and where they are most likely to be located (the grid).

The selection of pollutants was taken to be the same as the existing facility in another part of Ontario, while the meteorological data was applied to the new proposed new site. The resulting maximum concentrations (wet and dry deposition; 1 hour, 24 hour and yearly totals) are then used as input for the HHRA. We do not know how these assumptions actually reflect the real technology which is contemplated for use.

Since the consultants are working with a theoretical scenario, the assumptions made were reasonable and conservative, as applied to the new proposed facility, *provided that it perform similarly to the one which was mimicked in the modeling.*

HHRA

Appendix E provides the exposure point concentrations calculated for of contaminants of concern, all receptors, all locations, and all scenarios. (The appendix needs its own summary indicating those receptors, locations and scenarios that are or may be of concern. This can be picked up in the Summary Report and Executive Summary so that the curious reader can easily navigate the original calculations or results.

Appendix A describes the human receptor characteristics. It could highlight the situations or conditions that are or may be of concern. This section could also use its own summary for reasons stated above.

Appendix B *is* a straightforward toxicity assessment for all of the real and hypothetical CoPCs. It uses data from the literature with standard references. This does not have any surprises.

Draft report summarizes the scope and limitations of this report. Most importantly, this is a hypothetical situation on two grounds: first, there is no actual facility plan to evaluate; and two, the evaluation is based on actual performance of another facility and on hypothetical scenarios. This double estimation requires this risk assessment to provide caveats on final interpretation of results, regardless of their signaling a problem or not.

Because this draft report is what is likely to be read in total, it does well to provide the actual data for those concentrations, CoPCs and scenarios which may be of concern.

This information can be brought forward again in the Summary which should be short.

Critical comments:

Content and organization:
I found this document heavy on data (large volumes, 2 smaller volumes, and the actual report) and low on explanations. As it is a technical risk assessment, that is ok. However, I have stated above how the overall effort can be improved by adding a narrative to each set of data calculations repeating the assumptions, and scenarios calculated. These narratives can be picked up in the summary report, and key conclusions then picked up in the Executive Summary.

Executive Summary

The executive summary can bring forward material from each section and in 2-3 pages, provide the main points or conclusions. The executive summary provides the reader with an overview of the feasibility study and will help them see the entire picture before they read the details. We can expect that the public will only read the executive summary. Thus, the executive summary must hit all the highlights in a form that is easy to absorb and retain. A picture here might be useful (as well is might be in the Summary Report). The objectives of this report do not appear to be to give recommendations. I presume that these will come from stakeholders once they have worked though the meaning and implications of the findings of this Report.

Technical feasibility study

A techincal feasilibty study poses questions such as whether the technology needed for the system exists, how difficult it will be to build, and whether the firm has enough experience using that technology. None of these considerations is worked through in this study. Hence I ask if this should be referred to as a feasibility study, or a scenario proposal (see below).

"The aim of the internal methodological project is to build upon the existing expertise in scenario building and to attempt to create a model, which uses quantitative variables of the scenario process thus enabling to easily update already developed scenarios. The advantage of such a model would be that *once prepared* (italics mine) scenarios would not be completed for all times but would be open for updates when central variables change."[1]

Since we know nothing about the technology to be used, we can only use scenarios to forecast issues of concern. This capability of this study should be emphasized. The real answer cannot come without more detail about the actual proposal.

Because this study is going to be work-shopped at 7 public venues, it is important that everything about it is clear and that the way the results are obtained is absolutely clear.

[1] Dr. Bernd Beckett. June 10, 2007 http://www.isi.fraunhofer.de/t/mitarbeiter/e_bb.htm

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15 June, 2007

Dr. Lesbia Smith
64 Rathnelly Avenue
Toronto, ON M4V 2M6

Dear Dr. Smith:

Re: Response to Comments Provided by EOHS +Plus on the Durham and York Region – Residual Waste Study - Generic Risk Assessment

Jacques Whitford would like to thank you for the insightful comments and suggestions raised in your peer review letter dated June 10, 2007. These comments were general in nature and were considered through out the final report as it was drafted. Wherever practicable these comments were adopted in the report, however, due to time constraints it was not possible to incorporate all comments. An example of this is that the technical appendices do not all contain explanations or introductions.

Thank you for your effort in preparing your peer review.

Yours very truly,

JACQUES WHITFORD LIMITED

Original Signed By

Chris Ollson, Ph.D.
Director, Environmental & Occupational Health Science
Jacques Whitford Limited