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MEMORANDUM

TO:	Dave Wake MTO, Murray Thompson URS	33900-6
FROM:	Harriet Phillips and Doug Chambers	13 March 2009
SUBJ:	Response to Comments from MOE Standards Development Branch	

The attached table provides our response to the comments provided in the memo of March 5, 2009 from the Ministry of the Environment Standards Development Branch.

If you have any questions on the responses, please do not hesitate to contact us.

Response to Comments from Standards Development Branch (SDB) on the Human Health Risk Assessment (HHRA) of the DRIC Study by SENES Consultants Limited dated December 2008.

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1	The Air Quality Impact Assessment (AQIA) is continually referenced throughout this human health risk assessment. In order for the reviewer to understand the assessment of risk it is important that a Human Health Risk Assessment report contain all the information needed to appropriately and thoroughly characterize the risks. (Otherwise it is not a complete risk assessment.) Therefore, we suggest that the relevant information from the AQIA be incorporated into the HHRA.	Relevant information from the Air Quality Impact Assessment will be included in an updated document to assist the MOE in their review. However, it should be noted that the HHRA is a Supporting Document to a federal and Provincial Environmental Assessment and as such is not considered a stand alone document.
2	SDB did find some errors in the calculations reviewed and thus encourages the proponent to verify each calculation. Further, all data inputs were taken from the AQIA which was not reviewed by SDB for accuracy. As a result SDB is dependent upon the MOE AQIA reviewer accepting, and verifying if possible, the background concentrations, accuracy of the data used by the consultant including the modelled emissions estimates, as valid and accurate. The results and conclusions of the human health risk assessment are dependent upon the quality and accuracy of the information contained in the AQIA, and therefore it is important that MOE staff conduct a thorough review of the AQIA document.	There are no errors in the calculations provided in the document. All calculations have undergone QA/QC as per our ISO 90001 procedures. No changes are necessary. The MOE AQIA reviewer did not have any issues relating to the model used in the AQIA including the background concentrations and the results. Thus the air concentrations used in the HHRA are valid.
3	Statements in the HHRA indicate that the Technically and Environmentally Preferred Alternative (TEPA) is not expected to present an unacceptable risk to human health. This statement is based on calculations demonstrating that both TEPA and 'No Build' are expected to contribute, for some contaminants, an amount equivalent	Apparently there is some confusion as to what was presented in the HHRA. All predicted concentrations in the report include background. The predicted concentrations are composed primarily of background with traffic related inputs representing a small fraction of the background exposure. There is no doubling of the air concentrations. For example, for SO_2 the background or ambient concentration in the Windsor area is 32 $\mu g/m^3$ and the current

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	to the background concentrations in the Windsor area. This would represent an increase of 100%, a doubling of the current air concentrations, of these compounds.	roadway which is Talbot Road and Huron Church Road ("No Build) or the Windsor-Essex Parkway add an additional 0.1 to 0.7 µg/m^3 to the background air concentration.
4	**1. Objective of HHRA**: It is unclear to the reviewer why the focus of the HHRA has been limited to the phase following construction, since risk to human health could occur from all phases of a project from construction through to decommissioning. However, the scope of the DRIC is defined as: *"The primary objective of this HHRA is to determine whether chemical concentrations emitted from vehicles on the proposed road way for the TEPA have the potential for unacceptable health effects to people located in the immediate area in comparison to the 'Future No Build' scenario."* This is a very narrow scope given the project will span from Highway 401 to the Detroit River, and construction is likely to occur over many years. Construction of such a massive project would have its own set of impacts to the area, as would maintenance activities and decommissioning. Further, this assessment of risk will assist in determining if the proposed risk mitigation measures will be effective in removing the risk. If the mitigation measures do not perform *in situ* as predicted, the HHRA will be invaluable in determining corrective measures.	It is agreed that the scope of the HHRA is very narrow; however, over the course of the four year environmental assessment study and numerous public meetings the DRIC study team heard that the public was interested in Air Quality and potential health issues related to the Parkway after construction. . It has been agreed with the MOE SDB that construction is not part of this current HHRA but will be dealt with in the future when the design plans are finalized. At that time the MOE will be involved in discussions as to the scope and type of assessment needed.
5	In addition, the report does not provide support for how	The immediate area was described in the EA document and Air Quality

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	the 'immediate area' was selected nor does it clearly define the 'immediate area'. In addition to including this information, it is important to also present it visually using maps and/or figures.	Impact Assessment document. From a Provincial approval process the immediate area is the area surrounding the Parkway only. More description and mapping will be provided in an updated document.
6	Considerations in Assessing the Construction Phase: - Exposure of workers to hazards of environmental origin would have to be assessed, the associated parameters would differ from residential receptors; - Dust/PM_{10} (particulate matter) generated from unpaved road surfaces would be different than that generated when the project is complete and in use; - Vehicle use and composition would be different for each phase; - As a result of a larger volume of 'dust' being generated consideration should be given as to whether the indoor (and outdoor) environment of the residential receptor will be impacted. (It would be expected that construction activities would contribute to indoor dust levels).	It has been agreed with the MOE SDB that construction is not part of this current HHRA but will be dealt with in the future when the design plans are finalized. At that time the MOE will be involved in discussions as to the scope and type of assessment needed.
7	**2. Inconsistency and lack of clarity on scope of assessment**: The Introduction (p. 1) states: *"This report provides a discussion of the assessment of the technically and environmentally preferred crossing, plaza, and access road related to potential adverse effects to humans in the vicinity of the roadway…"* However in more than one section of the HHRA it is	The HHRA only focuses on the Parkway as that is the scope of the Provincial EA. The Plaza and Crossing are part of the federal undertaking. In addition the Plaza and Crossing are located near industrial areas.

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	stated that the Plaza and Crossing were not assessed in the HHRA since there were no nearby receptors. The report must be consistent and transparent when discussing what was assessed and what was not assessed. Further comments on the appropriateness of excluding the Plaza and Crossing are provided in this memo.	
8	Two scenarios in two different locations are being compared in this HHRA: 1 No-Build: The current roadway, through the city of Windsor, used to gain access to the Ambassador Bridge. The 'No-Build' scenario is not well described in this HHRA so the reviewer could not distill how many lanes are presently there, and therefore whether the assumptions regarding increased traffic and congestion are accurate. 2 The proposed parkway (TEPA) which does not yet exist. It is not clear if there are currently any roads in that area, i.e., are smaller local roads being turned into a parkway? With the purpose of this parkway being to take (the majority?) international vehicular traffic away from the currently used Huron Church Road which leads to the Ambassador Bridge? The maps and figures provided only highlight the TEPA and receptors along TEPA. The same detail should be provided for the current 'No Build' area.	The "No Build" Scenario is documented in the Air Quality Impact Assessment document as well as the EA document. Talbot Road (Highway 3) and Huron Church Road are currently the roads used to access the Ambassador Bridge. Highway 3 is a 4 lane rural highway and Huron-Church Road is a 6-lane arterial road. The transportation modelling was done by IBI and is provided in another supporting document. This information was used in the AQIA to determine the predicted concentrations. The MOE Air Quality Reviewer did not have issues relating to the traffic inputs into the AQIA. A more detailed description of the "No Build" scenario will be provided in the updated report including maps. The TEPA consists of the proposed Parkway which is a six lane highway which will be built in the same corridor as Highway 3 and a section of Huron Church Road up to the EC Row Expressway. At EC Row, the Parkway will then divert from Huron Church and be located in the same corridor as EC Row until it connects with the Plaza and Crossing. Traffic will have the choice of either going along the Parkway to the new Plaza and Crossing or going along Huron Church to the Ambassador Bridge. A more detailed description of the TEPA will be provided in the updated report including maps.
9	In addition given the above it is expected that there would be two calculation scenarios for both TEPA and 'No Build' for a total of four scenarios to compare risks. For example: a comparison of TEPA and 'No Build' should	The appropriate scenarios have been examined in the HHRA. It has been agreed with the MOE SDB that while the Huron Church access road to the Ambassador Bridge is outside the area of analysis for the EA and HHRA that calculations will be provided for this stretch of road in the updated report.

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	include: o receptors along the current roadway (1), such as residents, giving risk comparisons for the 3 horizon years based on TEPA and No Build; and o receptors along the TEPA (2), such as recreational users and adjacent residents, under the current 'No Build' and with the TEPA, also for the 3 horizon years. Presumably since there is currently no traffic associated with the TEPA, at location 2, the current air quality would be experiencing a lower load of air pollutants than when the TEPA is built and the (presumed) 'majority' of international traffic will be using it.	Residential receptors were evaluated for the "No Build" and TEPA. These receptors were selected since they represent the most exposed individuals along the roadways as they are assumed to be exposed 24 hrs per day, 7 days per week for 365 days per year for a 75 year lifetime. Recreational users will be exposed for a much shorter time and are thus encompassed by the residential receptors. However, additional calculations will be provided for recreational users of the trails on the green space.
10	Are there plans to 'decommission' the Ambassador Bridge? If not, then would it not be logical to assume that at some point in the future its use will increase to capacity? And would that occur by 2035?	There are no plans to decommission the Ambassador Bridge. The Ambassador Bridge is not within the scope of the EA. In addition, the Parkway has been overdesigned and it will not reach capacity in 2035.
11	Salt and sand are applied to Ontario roads during winter months, a certain amount of which will eventually be washed off the roadways (or the bridge) and reach local water ways, whether directly or through storm sewers. There is no discussion as to whether or not this will impact water wells, aquifers or other water bodies that drinking water may be taken from.	The collection of social data via questionnaires, comment forms, and context sensitive solutions workshops undertaken as part of the study did not identify any recreational uses of the watercourses influenced by potential project effects on surface and groundwater resources. Review of MOE records indicate that there may be a few wells within approximately 250 m of the corridor but these are all in locations that are now serviced by watermains. Mapping received from utility companies and municipalities indicate that the vast majority of the water in the study area (all three municipalities) is supplied by Windsor Utilities Commision watermains and the intake for this utility is located upstream of the discharge of the stormwater discharge from the Parkway into the Detroit River. It is likely that there are some wells in the

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		vicinity of the receiving watercourses between the study area and the Detroit River. It is understood that wells in the vicinity of the project area draw water from the underlying bedrock aquifer. With respect to the influence of the roadway run-off on this subsurface aquifer, it is noted that the proposed roadway is separated from the underlying bedrock aquifer by a layer of low permeability clayey silt to silty clay (aquitard) that, depending on the final roadway elevation, will be some 15 to 30 m thick below the roadway.
12	The document refers to transborder air pollution (p. ES-1, 5). One would presume we have an obligation to determine whether this project will have a negative or positive affect on air quality further afield from the TEPA.	Transboundary pollution is the driver of air quality in Windsor and has been recognized as such by the Ministry of the Environment (MOE) in their publications "*Preliminary Air Quality Assessment Related to Traffic Congestion at Windsor's Ambassador Bridge, 2004*", "*Transboundary Air Pollution in Ontario, 2005"*, and the annual Air Quality in Ontario publications. The Preliminary Air Quality Assessment Related to Traffic Congestion at Windsor's Ambassador Bridge states: *"Transboundary air pollutants from the United States account for up to 50 per cent of smog in Southwestern Ontario. In Windsor, this value may be as high as 90 per cent."* The AQIA indicates that the traffic related concentrations are highest within 50m of the roadway and drop-off rapidly the further away from the roadway to background concentrations. Thus the project has no effect on background further afield from the TEPA.
13	The problem formulation stage is where the risk assessor presents the Chemicals of Potential Concern (COPCs) and explains the criteria used to select them and the criteria and rationale used to determine a final list of Chemicals of Concern (COCs). While Table 2.1 presents the selected COC's, there is no discussion on how the COCs were selected, nor the rationale for	A number of chemicals were considered in the development of the COCs. The AQIA document contains a discussion of these chemicals. Chemicals associated with vehicle tailpipe emissions and vehicular movements on roads were considered in the selection of COCs. The U.S. AP-42 document was consulted for emission factors related to vehicular emissions. For example, VOCs, PAHs, CO, NO_x, SO_2, CO_2, PM_{10}, $PM_{2.5}$ were all considered. A detailed discussion of all the chemicals considered and the rationale for

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	choosing those selected. The first step in this HHRA should be to identify all COPCs as a result of constructing, operating and decommissioning this parkway (and service roads). From this 'list' the reviewer distills the COCs. For both steps a clear rationale must be provided.	dropping chemicals from further consideration will be provided in the updated document. It should be noted that the COCs that were evaluated in the HHRA document will remain unchanged.
14	NO_x and volatile organic compounds (VOCs) are carried forward as chemicals of concern, which is appropriate since it is known that these compounds are emitted in vehicle exhaust. However, it is also known that, in the presence of sunlight, they combine to produce ozone, which is known to have human health impacts. Why has ozone not been included in this assessment? In order to be considered complete, an assessment for a project involving vehicle emissions should address ozone and the potential for associated human health effects.	Ozone was considered as a potential COC; however the average wind speed in Windsor is 4.36 m/s. Therefore, in an hour, the pollutant travels 16 km during average conditions. Even allowing for a low wind speed of 1.5 m/s provides for a distance of 5 km that ozone would travel within an hour. This is well beyond the area of study for the DRIC project. Both $PM_{2.5}$ and NO_x (ozone precursors) were assessed and beyond a few hundred metres their maximum impact is not detectable relative to ambient conditions. Therefore ozone was not considered as a COC.
15	The reviewer noted the absence of compounds known to be associated with vehicular emissions, from the table listing the chemicals of concern selected for assessment. For example, since the project is the construction of a parkway to handle cross border traffic of which a large portion is the transportation of goods via diesel trucks, (p. 1 states: '*The City of Windsor also has a relatively high fraction of diesel powered transport trucks that are used to move goods into and out of Canada.*'), it would be expected that there would be some discussion of diesel emissions - for example, polycyclic aromatic hydrocarbons (PAHs), nitro-PAHs, and particulate	PAHs were considered in the original list of COCs; however there are no emissions factors to evaluate concentrations of PAHs. Naphthalene emission factors can be used as a surrogate for PAHs, these emission factors are the same order of magnitude as 1,3-butadiene. Naphthalene background concentrations in Windsor are approximately 1 $\mu g/m^3$ and based on 1,3-butadiene, the predicted incremental increase over background for naphthalene associated with vehicle emissions is in the order of 0.05 $\mu g/m^3$ resulting in an overall concentration of 1.05 µg/m3 and an increase of less than 10% over background and therefore PAHs were dropped from consideration.

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	matter. Some of the compounds in that family are considered to be human carcinogens. It would also be expected that compounds associated with the normal wear and tear of vehicles, which may include some metals and possibly asbestos from the brake linings of large trucks, would also at least make the list of COPCs.[1] Again, an important component of an HHRA is to carefully identify each COPC that could be emitted as a result of the undertaking.	
16	Section 2.2 presents a list of 21 receptor locations but no information is provided to explain how these receptors were selected. The list provided includes 15 location names and when the numbers in brackets, following those names, are counted there are a total of 21 'locations'. The numbers in brackets are not explained. While many of them are found on figure 2.1, this figure has more than 50 numbers on it, many of which are indistinguishable because they overlap. Further page 9 states: *"In general two different locations were selected within these areas through discussions with the Air Quality modelling team."* It is not clear to the reviewer what the above statement is referring to. Only six of the 15 named locations (p. 9) have two numbers in brackets, so it does not appear as though two different locations were selected. Also what	The information on all receptors was provided in the AQIA. Over 2400 receptors in the Windsor Airshed were examined within the Air Quality modelling. The first two rows of receptors were placed at 50 m intervals from each side of the existing road, followed by 100 m intervals up to 500 m away. The figure below provides an example of receptor locations relative to the Windsor-Essex Parkway. Another grid with 500 m x 500 m spacing was then overlaid to cover the rest of the modelling domain, which was essentially all of west Windsor and the surrounding communities. The numbers in brackets refer to the receptor location number from the Air Quality modelling. The text on page 9 should refer to **twenty one** different locations. Information on receptors from the AQIA will be added to the updated report.

[1] The provision of this information is not intended to be a substitute for the proponent conducting this step and working through all the steps of 'problem formulation' to determine all relevant COPCs and COCs.

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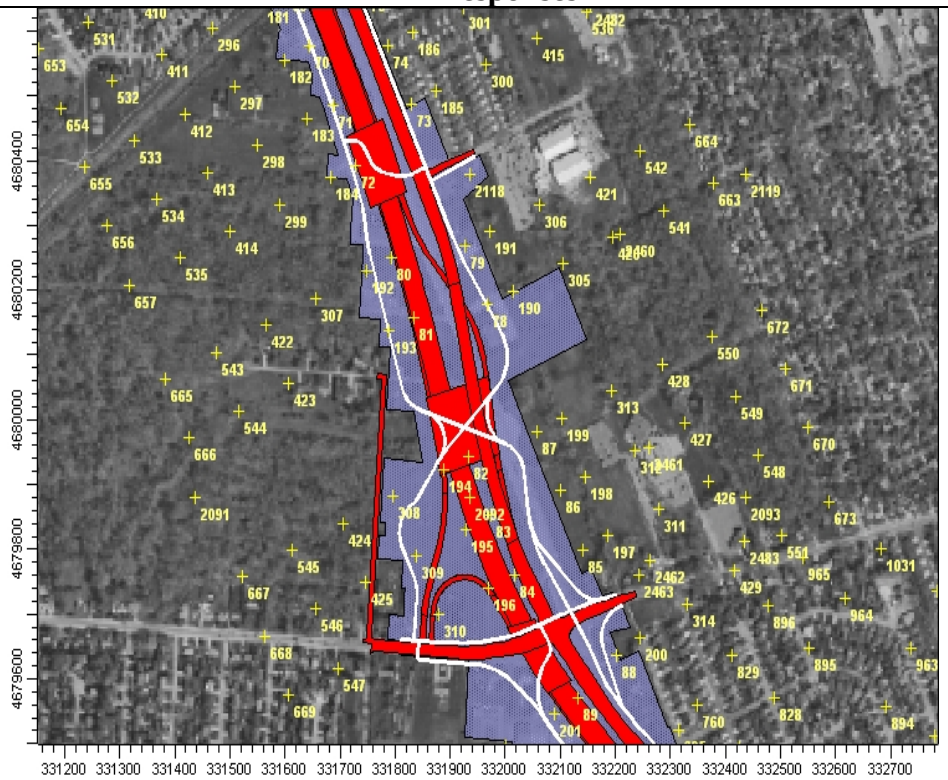
	was the criteria for selection and what was the pool from which they were selected?	
17	It is also stated in the HHRA that: "*These areas generally represented maximum concentration locations within these areas.*" Further explanation is required. For example, locations where maximum concentrations were not used must be identified, and it must be explained why this was done. In addition, in reference to 'these areas' it is confusing to use a term to define itself. As stated previously it should be clear to the reviewer which locations and receptors were	The receptors selected in the HHRA represented the maximum concentrations in different neighbourhoods along the Parkway. These maximum "neighbourhood" concentrations are lower than concentrations within the right-of-way of the Parkway hence the use of the word "generally". More clarification will be provided in the updated report.

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	assessed and why.	
18	In addition sensitive receptors have not been discussed nor characterized. This is surprising since the discussion of potential health effects for particulate matter, in the Hazard Assessment Section, discusses individuals with conditions that makes them predisposed to adverse effects from exposure. The HHRA must include a proper discussion of the potentially sensitive subgroups of the population.	Sensitive receptors have been discussed such as the LaSalle Home for the Aged. There are no special characteristics of these individuals different for those presented in the risk assessment. No other risk assessments to our knowledge have characteristics of sensitive receptors. The sensitivity of the receptors are captured within the use of the toxicity reference values which are selected to protect such sensitive receptors. This discussion will be added to the updated report.
19	One of the benefits put forward in selecting this 'alternative' is the additional green areas and a recreational trail that will be created on top of, and alongside, this parkway. Page P-1 states: *"a grade separated recreational trail system, and extensive green area"* and page 1 states: *"A landscaped parkland buffer to the right-of-way provides a trail system and linkages to both sides of the transportation corridor."* Yet there is no mention of potential receptors (which will include recreational users of all ages), that will be using this new recreational trail / green space. These receptors will be in the immediate vicinity of a roadway that will have heavy traffic (including diesel powered trucks) and so it would seem prudent to include them in the HHRA. In addition it would be expected that a number of these potential receptors will be exercising which will increase exposure via the inhalation route.	Residential receptors were evaluated since they represent the most exposed individuals along the roadways as they are assumed to be exposed 24 hrs per day, 7 days per week for 365 days per year for a 75 year lifetime. Recreational users will be exposed for a much shorter time and are thus encompassed by the residential receptors. However, additional calculations will be provided for recreational users of the trails on the green space.
20	In several places it states *"The Plaza and Crossing were*	The HHRA only focuses on the Parkway as that is the scope of the Provincial

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	not assessed in the HHRA since there were no nearby receptors." However the TEPA is being built to move people, and therefore they will end up at the Plaza and Crossing during which time they will be continually exposed to vehicle emissions. As a result it is expected that both of these locations will have a variety of receptors exposed for 'short durations' and potentially longer exposure scenarios. The reviewer also suggests that the HHRA should clarify whether or not back-up due to Customs delays has been considered. In addition there will be workers, at the Plaza, who could be exposed for many years. The rationale for excluding these two locations from this assessment is inadequate.	EA. The Plaza and Crossing are part of the federal undertaking. In addition the Plaza and Crossing are located near industrial areas.
21	The timeline for this project has not been provided. For example, when is construction assumed to begin and be completed? Exposures have been calculated for 2015, 2025 and 2035. What was the basis for selecting those years? How would different horizons change the exposure assessment?	The timelines for the project have been discussed in the Environmental assessment and have been selected based on a thirty year horizon which is common industry practice for transportation projects. Construction is scheduled for completed in 2013. 2005 was selected as the base year with horizons selected 10, 20 and 30 years into the future.
22	Pages 9 & 11, Table 2.4 is referred to but not included in this document.	No Table 2.4 exists. The text will be corrected.
23	The 'area of consideration' is not clearly defined. The area being considered in this assessment should be explained both in writing and using maps/figures. The three figures in their present format do not clearly identify the area that is being assessed and the receptor locations.	The area of consideration is the area around the Parkway and has been described in the Air Quality Impact Assessment and the EA Report. More information from these documents, including maps, will be provided in the updated document.
24	P. 11 It is stated:	The use of 75 years of deposition provides the maximum concentrations of

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	"Section 3, provides maximum predicted soil concentrations after 75 years of deposition of VOCs;" Whereas it is useful to understand deposition after 75 years, it would be important to also understand deposition at the end of the horizon years forming the basis of this HHRA – namely years 2015, 2025 and 2035. If the parkway and bridge were completed today 2035 is 26 years from now.	COCs built up in the soil and is a conservative estimate of the soil concentrations. The use of this value ensures that exposures will not be underestimated. Soil concentrations for the different horizon years would be much lower than those accumulated after 75 years. It is common practice in risk assessments to use very conservative estimates such as soil concentrations after 75 years to provide a conservative estimate of risks.
25	Table 2.3, A wide range of values are reported in the literature for soil ingestion rates (SIRs). And in this context, MOE expects that the proponent would have included in this discussion consideration of the SIRs in the Ministry's October 2008 posting *"The Rationale for the Development of Generic Soil and Groundwater Standards for Use at Contaminated Sites in Ontario"* (http://www.ene.gov.on.ca/envision/env_reg/er/documents/2008/010-4642%203.pdf) to the Environmental Registry (ER). This document contains the SIRs the Ministry considers appropriate for use in assessing this exposure pathway. The Ministry notes that the SIRs used in this HHRA report are all lower, and therefore less conservative, than those contained in the ER posting. If the proponent wishes to use different SIRs a rationale must be provided to support the use of the SIRs selected.	The HHRA was done to address requirements of federal agencies under the Canadian Environmental Assessment Act. Hence the Health Canada receptor characteristics were selected for use in the risk assessment. There are many scientific debates surrounding the use of an appropriate soil ingestion rate for risk assessments and Health Canada, the U.S. EPA and the Ontario Ministry of the Environment (to name a few) all support different soil ingestion rates. It should be noted that since this is a comparative risk assessment which is comparing the "No Build" and the TEPA scenarios, the use of the different soil ingestion rates would not change the conclusion of the risk assessment. Nonetheless, a sensitivity analysis (i.e. additional calculations) will be carried out using the Ontario Ministry of the Environment soil ingestion rates.
26	In addition, Richardson, 1997 is cited as the source of the SIRs, but the Richardson, 1997 document[2] does not provide soil ingestion rates.	The source is Health Canada 2004a. *Federal Contaminated Site Risk Assessment in Canada. Part 1: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA).* Environmental Health Assessment

[2] Richardson, G.M. 1997. Compendium of Canadian Human Exposure Factors for Risk Assessment. O'Connor Associates

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		Services. Cat. No.: H46-2/04-367E.
27	It is stated more than once: *"it has been assumed that all resident receptors drink water obtained from the City of Windsor's potable water system and that this water source is not impacted by potential emissions from the proposed roadway." (p. 11, Table 2.2)* It should not be necessary to make an assumption regarding the source of resident's drinking water. The location of the parkway (associated infrastructure) and receptors are known, and thus it should be possible to obtain information from the City of Windsor with regards to the source of the receptor's water. The source of Windsor's drinking water should be identified.	The collection of social data via questionnaires, comment forms, and context sensitive solution workshop undertaken as part of the EA study did not identify any recreational uses of the watercourses influenced by potential project effects on surface and groundwater resources. Review of MOE records indicate that there may be a few wells within approximately 250 m of the corridor but these are all in locations that are now serviced by watermains. Mapping received from utility companies and municipalities indicate that the vast majority of the water in the study area (all three municipalities) is supplied by Windsor Utilities Commision watermains and the intake for this utility is located upstream of the discharge of the stormwater discharge from the Parkway into the Detroit River. It is likely that there are some wells in the vicinity of the receiving watercourses between the study area and the Detroit River. It is understood that wells in the vicinity of the project area draw water from the underlying bedrock aquifer. With respect to the influence of the roadway run-off on this subsurface aquifer, it is noted that the proposed roadway is separated from the underlying bedrock aquifer by a layer of low permeability clayey silt to silty clay (aquitard) that, depending on the final roadway elevation, will be some 15 to 30 m thick below the roadway.
28	The proponent should also be able to determine if the parkway crosses any waterways (or other water bodies) that might feed into aquifers or other sources of drinking water. (Also see earlier comment regarding salt migrating into waterways).	Currently, there are no systems in place to collect road runoff from Talbot Road or Huron Church Road and thus water from these roadways infiltrates into the surrounding aquifers. Stormwater management ponds will be located at various points along the Parkway. Water from these ponds will then feed via Turkey Creek into the Detroit River downstream of the intake for the municipal drinking water intake and thus there will be no influence on drinking water sources from the Parkway.
29	Indoor exposures should be discussed. The parkway (as well as construction and maintenance activities) will generate particulates some of which will end up in the indoor (residential/school etc.) environment. This will	The assessment considered that an individual in a residence would be exposed 24hrs a day, 7 days per week, 365 days per year outdoors. This is a conservative estimate of exposure. There are some literature sources indicating that indoor air concentrations of volatile organic chemicals (VOCs)

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	add to the inhalation and ingestion routes of exposure. This pathway must be discussed and if it is not going to be assessed a scientific basis for ruling it out must be provided.	can be potentially higher in indoor air than outdoor air, however, these concentrations are due to indoor sources of the VOCs. Since this is a comparative assessment of traffic related sources the use of the outdoor air exposure assumption of 24 hours a day is the appropriate measure. In addition it must be emphasized that background (ambient) air concentrations dominate the inhalation exposure pathway.
30	The proponent has been inconsistent in defining the exposure duration. The assessment switches back and forth from using a total duration of exposure of 30 and 75 years. The dose, and hence the hazard quotients and cancer risk levels, are directly impacted by the selection of the appropriate averaging time (duration of exposure) used in the assessment. For example[3]: p. 18, 3.3.1 states: '*for carcinogenic effects the air concentration is adjusted for the exposure duration (30 years of a 75 year lifetime).*' p. 51, 5.4.1 states: '*it was assumed that an adult residential receptor was assumed to live at their house 24 hours/day, 365 days/year for 30 years….*' While, p. 11, 2.2.1 states: '*the resident receptor ….. is assumed to be exposed for 75 years at the maximum concentration possible, 24 hours each day of the year. An exposure duration of 75 years was selected to represent a lifetime of exposure for a resident.*'	75 years is the exposure duration that has been used. This is a very conservative exposure calculation since population mobility statistics show that the average length of time that someone stays in a home is 5 years (U.S. EPA Exposure Factors Handbook 1997). The text will be corrected.

[3] The reviewer would like to emphasize to the proponent that these are just selected examples and it is the responsibility of the proponent to thoroughly edit and check the document to ensure consistency.

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	Pages ES-3 and 7 state: *'it was assumed that residents were exposed to vehicle emissions 24 hours a day, 7 days a week over their entire lifetime.'*	
31	P. 14 for the dermal exposure to soils and dust pathway it is stated: *"In environmental settings, this pathway is insignificant and thus, is not assessed further."* Rationale for this statement must be provided if the proponent is going to rule out this pathway. Many HHRAs have been conducted which examine and characterize this route of exposure. This pathway would be especially relevant during the construction and decommissioning phases.	As shown in the dose calculations of Appendix B of the HHRA, the dominant exposure pathway is consumption of backyard produce rather than soil exposure. In many cases the differences between these pathways are orders of magnitude. Thus the calculations of exposure support the exclusion of the dermal exposure pathway.
32	Further, it goes on to say: "*With the exception of formaldehyde, the predicted soil concentrations are so small that the dermal exposure pathway will be insignificant."* With this statement in mind it would seem that formaldehyde should have been carried forward for assessment via the dermal pathway. As there is a continuous source (emissions will essentially be 24 hours, 7 days a week) it is difficult for the Ministry to accept that any pathway would be ruled out. Dermal exposure would occur during gardening, which is assumed to occur given the assumption in this HHRA that 7.5% of vegetable intake is from backyard gardens.	For formaldehyde, Table B4-9 shows that backyard garden produce is the dominant exposure pathway over soil ingestion by four orders of magnitude. Therefore, the dermal exposure pathway will be even smaller than the soil ingestion pathway which supports the exclusion of the dermal exposure pathway.

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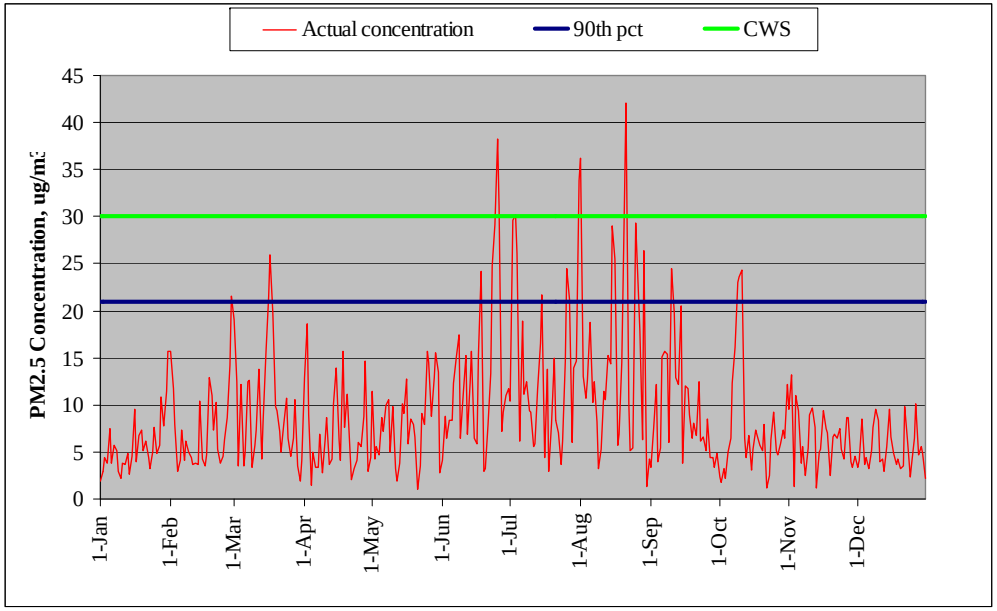
33	Figure 3.1 Conceptual Site Model (CSM). This model has not depicted all potential exposure pathways. A CSM normally depicts all potential exposure pathways and indicates which ones are complete, and therefore being assessed, and which ones are incomplete and are therefore not carried through in the assessment.	The CSM illustrates all the exposure pathways considered in the risk assessment namely inhalation of air, soils/dust and ingestion of soil and garden produce. A revised diagram indicating the incomplete pathways will be provided in the updated document.
34	P. 13, It is stated that the assumption was made that drinking water was obtained from a source not impacted by the proposed roadway. The reviewer expects that the proponent could do more than just make this assumption. It should be possible to determine the source, and location, of the selected receptors drinking water (personal wells, municipally supplied etc.). It is expected that within the 'area of consideration' the proponent could determine if there are rivers or lakes, that supply drinking water that would be impacted by runoff or deposition. See comments under Problem Formulation for further discussion.	See response to comment 27.
35	Carbon monoxide (CO), ammonia and PM10 are listed as COCs in Table 2.1. However, while background information is included for CO in Table 3.1, these three compounds are not discussed further in the Exposure Assessment and no explanation for dropping them is provided.[4]	As discussed in comment 13 a detailed rationale will be provided for all COC that have not been considered in the assessment.
36	P. 15, Units are missing from Table 3.1.	The units are µg/m^3.

[4] CO does make a brief appearance in the Hazard Assessment section, as it is listed in Table 4.2 and a brief paragraph is included describing the basis of the guideline.

#	SDB Comment	Responses
	P. 15, More information on the background concentrations needs to be provided. For example: a year of reference, as well as a reference indicating where this information was taken from. On page G-1, the Glossary defines background concentration as follows: *"representative amount of chemicals in air, water or soil to which people are routinely exposed (generally mean or 90th percentile concentration)."* The report does not indicate if the 90th percentile or mean was used. The proponent must indicate what measure of distribution was used and why. It is expected that this issue would warrant discussion in the Uncertainty Analysis.	A detailed discussion of background air concentrations was provided in the Air Quality Impact Analysis. A brief summary is provided below. An important consideration in the air quality analysis is the ambient concentration of a contaminant that would occur without the inclusion of the transportation element. This is commonly referred to as the "background" concentration. The Ministry of the Environment (MOE) typically requires the assessment be completed using a 90th percentile background concentration which is reflective of a background concentration that would actually be lower 90% of the time. Alternatively, 10% of the time the background concentrations will be higher. While the choice of the 90th percentile background may under-predict the absolute maximum concentrations reported, it tends to over-predict the concentrations (and the numbers of exceedances) because the background is artificially elevated as shown in the figure below which provides an example of $PM_{2.5}$. For $PM_{2.5}$ the 90th percentile background concentration is 21 µg/m^3 in Windsor based on data reported for the MOE's air quality stations in Windsor. As seen from the figure, the use of the 90th percentile value overestimates the background exposure for a significant part of the year. In addition, **Error! Reference source not found.** shows that the day-to-day variability in ambient (background) concentrations is typically several µg/m^3 and can be as high as 30 µg/m^3.

#	SDB Comment	Responses
		 This discussion will be added to an updated report and included in an uncertainty discussion.
37	It would be informative to discuss if and how background may be expected to change between the 3 horizon years. Was this taken into account in this HHRA? It is expected that this would be discussed in the Uncertainty Analysis.	It is difficult to determine how background will change between the three horizon years and therefore the background was fixed in the assessment. The uncertainty associated with this assumption will be discussed in the Uncertainty section.
38	P. 15, Table 3.1 the annual background concentration for NO2 has not been provided, while a 24-hour (and 1-hour) value has been given. The remainder of the HHRA presents predicted concentrations and hazard quotients (HQs) for NO_2 on an annual (and 1-hour) basis but not a 24-hour basis. Please clarify.	There are no health based criteria with which to evaluate NO2 on an annual basis and thus only an evaluation of the 1 hr and annual exposure can be completed.
39	P. 18, 2nd paragraph, 'off-site' is not defined. It should	The term "off-site" will be removed from the discussion.

#	SDB Comment	Responses
	be clear what area is considered 'offsite'. It would be beneficial to have a map depicting the 'off-site' area.	
40	There is inconsistency with units. Table B.3-1 indicates the air concentrations are in units of mg/m^3 while Table C.1-2 indicates the air concentration is in units of ug/m^3. The numerical value is the same in both cases. Units need to be reviewed and corrections made where appropriate.	All air concentrations are in units of $\mu g/m^3$.
41	P. 19 The predicted soil concentrations for formaldehyde are approximately 20,000 to 5,000,000 times greater than the soil concentrations predicted for the other VOCs (even though the maximum predicted air concentrations are not more than double those of the other VOCs). It would be helpful to include a discussion on the reason for this result. Does it relate to the different properties of formaldehyde versus the other VOCs? It is expected that this would be discussed further in the Risk Characterization section.	The reason why there are differences between formaldehyde predictions in soil and the other VOCs is that formaldehyde is a less volatile VOC. The Henry's Law constant is 5 orders of magnitude lower than 1,3-butadiene (for example) resulting in a soil loss constant 6 orders of magnitude lower than that of 1,3-butadiene (for example). This results in the high predicted soil concentrations. This discussion will be added to Section 3 where the soil concentrations are predicted. It should be highlighted that background is responsible for the majority of the predicted soil concentration for formaldehyde with traffic sources only adding a small increment.
42	P. 20 Table 3.5 footnote states the inhalation dose for formaldehyde is for the composite receptor because it is considered to be a carcinogen. The hazard assessment (and Table 4.1) presents 1,3-butadiene and acetaldehyde as inhalation carcinogens and benzene is known to be a human carcinogen. Why aren't the respective values presented for the composite receptor for these other three carcinogens? (This statement also applies to the dose tables in Appendix B).	The risk assessment assesses both the non-carcinogenic and carcinogenic for all COC that have both carcinogenic and non-carcinogenic endpoints. Appendix B provides the exposure calculations for both the non-carcinogenic and carcinogenic endpoints.
43	This section does not provide any discussion on the	This was inadvertently left out and will be added to the text.

#	SDB Comment	Responses
	potential human health effects from exposure to benzene or the toxicity reference value (TRV) that will be used in the HHRA. Benzene has also been omitted from Table 4.1.	
44	The rationale provided for formaldehyde, acetaldehyde, acrolein and 1,3-butadiene only provides a brief discussion of the basis of the TRVs selected by the proponent for these compounds. There is no discussion of potential human health effects from either short- or long-term exposure to these contaminants. This section of a HHRA must include a discussion of potential human health effects for all COC's.	The TRVs used in this comparative evaluation were obtained from the United States Environmental Protection Agency database known as the Integrated Risk Information System (IRIS). The IRIS database has a detailed discussion on how each of the values has been selected and the values in the database also undergo a rigorous peer review process. The values from this database are used in risk assessments in the US, internationally and in Canada. Thus the values selected are appropriate for the assessment. Since it is a comparative risk assessment and not a risk assessment to determine the absolute risks the discussion provided is appropriate. However, more discussion will be provided in the text.
45	The rationale used in selecting each TRV in this human health risk assessment must be discussed. Information which should be discussed includes: • Were any other agencies considered besides the one selected and discussed? • What factors informed the selection of the TRV (such as relevant endpoint, time/duration of the study versus the exposure time in the assessment)? • Is the TRV protective of sensitive subgroups of the population? The selection of a TRV to be used in a risk assessment is a critical step. As such, sufficient detail must be provided to enable the reviewer to determine that the basis of the selected TRV is appropriate for the exposure scenario	See Response to comment 44 above.

#	SDB Comment	Responses
	being assessed. Necessary detail must also be provided to assure the reviewer that the most relevant TRV has been selected. The current presentation of the Hazard Assessment section of this HHRA does not facilitate the reviewer in assessing the appropriateness of the conclusion. For a better understanding of the key considerations in selecting TRVs, please refer to *"The Rationale for the Development of Generic Soil and Groundwater Standards for Use at Contaminated Sites in Ontario"* using the following link: http://www.ene.gov.on.ca/envision/env_reg/er/documents/2008/010-4642%203.pdf	
46	As an example: The proponent has used the US EPA's carcinogenicity assessment for 1,3-butadiene. This was last revised by the United States Environmental Protection Agency (US EPA) in 2002. Recently the states of North Carolina and Texas have incorporated updated exposure data, in their cancer risk analysis of 1,3-butadiene. The Ministry recommends the proponent refer to the Texas Commission on Environmental Quality (TCEQ) for updated information on hazard assessment and dose-response analysis for 1,3-butadiene. The documents from North Carolina and Texas were discussed in the Ministry's Science Discussion Document released to stakeholders in the fall of 2008. It should also be noted that in Table 4.1 and the paragraph (p. 25) referring to the US EPA's inhalation unit risk the value has been incorrectly reported as 0.03 $(mg/m^3)^{-1}$ when it is in fact $3*10^{-5}$ per (ug/m^3). Further,	More discussion will be provided in the text. A value of 0.03 $(mg/m^3)^{-1}$ is the same as $3*10^{-5}$ per (ug/m^3). In the absence of an oral TRV, the oral TRV for butadiene is based on extrapolation of the rodent based unit cancer risks for inhalation exposure

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	why has an oral slope factor been provided? A quick look at IRIS found the following: *"Quantitative Estimate of Carcinogenic Risk from Oral Exposure - None. 1,3-Butadiene is a gas at room temperature and pressure, making oral exposure unlikely."* From a quick search it appears that the 1,3-butadiene document referenced was superceded by a final version published in 2002. Why is the 1998 document (which says 'Draft Do Not Cite or Quote') still referenced? In addition, in the reference section it is listed as 1998c, but the references only cite one US EPA document from 1998. These comments are provided not to focus on 1,3-butadiene, but to convey the need to provide the rationale for selection of a TRV.	provided in Section 9.5 of the U.S. EPA (1998) document which range from 4 x 10^{-3}/ppm to 0.29/ppm. The average of these values was approximately 0.097/ppm which when adjusted to mg/m^3 using a conversion factor of 1 ppm = 2.25 mg/m^3 and extrapolation to a mg/kg d basis using an inhalation rate of 15.8 mg/m^3 and a body weight of 70.7 results in an oral slope factor of approximately 1.8 per mg/kg d.
47	US EPA inhalation unit risk values have also been incorrectly reported for formaldehyde and acetaldehyde.	The inhalation unit risk value for formaldehyde and acetaldehyde is 0.0022 $(mg/m^3)^{-1}$ and not 0.022 $(mg/m^3)^{-1}$ as reported in Table 4.1. This is a typographical error; however all the calculations were carried out with the correct unit risk value of 0.0022 $(mg/m^3)^{-1}$.
48	For sulphur dioxide Table 4.2 presents two 24-hour guideline values, one of which is an interim guideline. The discussion on SO_2 provides a second interim guideline. It appears from examining the Risk Characterization section of the HHRA that the highest (interim) 24hour guideline was used. This selection must be explained. In addition, presumably there are target dates for moving from the interim guideline to the	This is a comparative risk assessment between the "No Build" and TEPA scenarios and thus the overall conclusions are the same no matter what guideline is being used.

#	SDB Comment	Responses
	guideline. Has this been considered in the three horizon years examined in this assessment?	
49	It should be noted that WHO air quality guidelines (AQGs) are not considered to be toxicity reference values (TRVs). WHO AQGs are targets meaning that they are not derived solely based on the potential for human health impacts. The following are portions of paragraphs taken from the WHO Global Update 2005 referenced in this assessment: *"The WHO air quality guidelines are designed to offer guidance in reducing the health impacts of air pollution. …. These guidelines are intended to inform policy-makers and to provide appropriate targets for a broad range of policy options for air quality management in different parts of the world.* *Several key findings that have emerged in recent years merit special mention. Firstly, the evidence for ozone (O3) and particulate matter (PM) indicates that there are risks to health at concentrations currently found in many cities in developed countries. Moreover, as research has not identified thresholds below which adverse effects do not occur, it must be stressed that the guideline values provided here cannot fully protect human health.* *As noted above, the epidemiological evidence indicates that the possibility of*	The NO_2 guidelines are health based values with no safety factors built in and are not targets. The 24 hr SO_2 values are targets; however, there are no other values presently available to evaluate effects relating to SO_2 and the WHO values are the most current values available. A review of other risk assessments that consider SO_2 exposure also use the SO_2 values provided by the WHO. In terms of $PM_{2.5}$, the WHO was not used to determine any health based values. Instead information from a report published by the California Environmental Protection Agency Air Resources Board in 2008 was used and is a very current reference. The report was endorsed by a number of scientific advisors including Dr. Jonathan Levy, Dr. Barst Ostro and Dr. Arden Pope all well known scientists in the fine particulate area. In addition, the information on the document was peer reviewed by 12 experts including scientists such ad Dr. Doug Dockery, Dr. Kaz Ito, Dr. Morton Lippmann, Dr. Daniel Krewski and others. Thus the use of a threshold of 7 $\mu g/m^3$ is supported by these experts and they indicate that this level is the best information due to *the lack of long-term data at low ambient concentrations of $PM_{2.5}$*.

#	SDB Comment	Responses
	adverse health effects remains even if the guideline value is achieved, …."	
50	P. 34 states 7 ug/m^3 was used as the health based level for particulate matter. The tables show calculations using both 7 and 15 ug/m^3, thus an explanation should be provided indicating why two different values have been used.	Two different values have been used to show the diverse thoughts on $PM_{2.5}$, a value of 7 µg/m^3 is used by the California Air Resources Board (2008) and the value of 15 µg/m^3 was considered by the CCME in determining the Canada Wide Standard for $PM_{2.5}$. Some discussion on the use of these two values was provided on pg 39 of the report.
51	In addition, SDB would like to caution using the term 'health-based' throughout the document. The HHRA has noted that for some compounds research has not yet been able to identify an exposure level below which adverse health effects are not expected to occur. And so, care must be taken, when discussing the results of risk calculations that fall below the selected risk target, not to imply that those risk values imply public health is protected.	The NO_2 guidelines are health based values with no safety factors built in and are not targets. The 24 hr SO_2 values are targets; however, there are no other values presently available to evaluate effects relating to SO_2 and the WHO values are the most current values available. A review of other risk assessments that consider SO_2 exposure also use the SO_2 values provided by the WHO. SO_2 exposure is dominated by background with the traffic inputs only adding incrementally to the background concentration. The discussion on SO_2 on page 38 states that *"there is no difference in the health risks between the future "No Build" and the TEPA scenario."* No statement has been provided to imply that public health is protected.
52	Pages ES-4, and 54 state: *"Hazard quotients for non-carcinogenic VOCs (predicted exposure dose / chronic toxicity reference value) for background, Future "No Build" and the TEPA scenarios were below 0.2 for benzene and 1,3-butadiene."* The above named compounds are carcinogens, and therefore calculation of a 'hazard quotient' is inappropriate to express the risk from exposure to these substances. The standard practice in toxicology, for calculating cancer risks, is to calculate incremental lifetime cancer risk (ILCR) values. If these VOCs also	As discussed in the response to Comment 42, the risk assessment assesses both the non-carcinogenic and carcinogenic for all COC that have both carcinogenic and non-carcinogenic endpoints.

#	SDB Comment	Responses
	have non-carcinogenic effects, which are being assessed, then perhaps the proponent meant to say "for the non-carcinogenic effects of….."	
53	P. 7 (also p. ES-3 & 35) state: *"…. the Ontario Ministry of the Environment and the US EPA concur that a hazard quotient value below one (1) (for assessing gaseous air pollutants since they include background), ….. are not significant."* This statement is problematic from a number of perspectives. First, it is not clear as to what 'includes background' means and represents. Second, the Ministry expects the risk characterization to be done both including and excluding background.	It is agreed with the MOE that the inclusion of background and other exposure pathways in the calculation warrants the comparison to a value of 1.
54	P. 37 it is incorrect to state: *"A hazard quotient value for gaseous air pollutant of less than 1 indicates that the predicted air concentrations are less than the reference concentrations and as such there are no measurable health impacts expected."* The 'reference concentrations' used for the gaseous air pollutants are targets and as such 'compliance' with them does not ensure there will be no impacts to human health. As stated previously the hazard assessment section reports several times that several studies indicate there are no threshold levels below which health impacts have not been seen for some of these compounds.	This is a generic discussion provided and has nothing to do with the discussion of the various pollutants and as such is correct in a general sense.

#	SDB Comment	Responses
55	A quick check of a few hazard quotient calculations provided in Tables 5.1 and 5.2 found errors. While the titles of the tables state that background is included in the calculated values, one can only derive the same value when the predicted air concentration from Table 3.2 (which appears to exclude background) is divided by the selected reference value. An example calculation should be provided detailing how background was incorporated into the hazard quotients.	There are no errors in the calculations provided in the document. All calculations have been undergone QA/QC as per our ISO 90001 procedures. No changes are necessary. An example calculation of the hazard quotient which is simply the predicted air concentration (provided in Table 3.2) divided by the TRV will be provided in Appendix C.
56	Section 5.3 discusses hazard quotients and incremental risks for the five VOCs assessed in this assessment. In each chemical specific analysis it states something along the lines of: *"Background exposure accounts for the majority of the (chemical name) risks."* With the final conclusion for this section being: *"In summary, the predicted VOC concentrations for the Future No Build and TEPA scenarios are essentially the same as background. Therefore the TEPA scenario does not result in an increased risk of adverse health effects when compared to background or the Future No Build scenario."* With regards to the first statement, a comparison of Tables 3.1 and 3.3 finds the emissions to be roughly equal to background (although the issue of units needs to be addressed). As a result the reviewer finds the first	The reviewer is confused. All tables with predicted concentrations include background and there is no doubling of the load in the air shed. For example the background concentration of SO_2 is 32 µg/m^3 and predicted concentrations range from 32.1 to 32.7 µg/m^3.

#	SDB Comment	Responses
	statement to be misleading because if the emissions will contribute an amount equal to background, that would represent a doubling of the 'load' to the airshed.	
57	With regard to the second statement, that VOC emissions are similar to background, this represents a doubling, or 100% increase, in those particular compounds due to both 'No Build' and TEPA. Given that 'No Build' and TEPA are in two different locations this means that for the airshed surrounding the TEPA site, this potentially represents a doubling of VOC emissions.[5]	The reviewer is confused. All tables with predicted concentrations include background and there is no doubling of the load in the air shed. For example the background concentration of acrolein is 0.15 µg/m^3 and predicted concentrations for the 'No build" range from 0.151 to 0.163 µg/m^3. For the TEPA the concentrations range from 0.151 to 0.158 µg/m^3. The "No Build" and the TEPA are not in different locations, they are in the same location, the "No Build" represents the use of Talbot Road and Huron Church Road and the TEPA represents a newly build Parkway in the same road corridor as Talbot Road and Huron Church Road.
58	P. 40 states Ballpark location (assuming this is Ball Field 2479) is the highest concentration for PM. However looking at Table 3.2 the location with the highest PM is Bellwood Estates.	The statement on page 4 indicates that the Ballfield has the highest PM concentration for the Future "No Build" scenario. Table 3.2 shows that this statement is correct. The reviewer is referring to the TEPA.
59	P. 43 & 44 Table 5.6 presents the hazard quotients calculated for 1,3-butadiene. There are two problems with this. The first being 1,3-butadiene is a carcinogen and therefore the ILCR value should be calculated instead of an HQ. The second being that the information necessary to calculate an HQ was not provided in the hazard assessment section.	1,3-Butadiene has both carcinogenic and non-carcinogenic properties and both were evaluated in this assessment as shown in Table 5.6 (non-carcinogenic properties) and Table 5.7 (carcinogenic properties). The non-carcinogenic properties were based on inhalation exposure and were compared to a reference concentration provided in the U.S. EPA IRIS database of 2 x 10^{-3} mg/m^3 for an endpoint of ovarian atrophy. This value was inadvertently left out of Table 4.1.
60	P. 43 states the background concentration for benzene is 2.3 ug/m^3 while Table 3.1 presented a value of 2.7 (no units provided). P. 46 states the background concentration for formaldehyde is 4.3 ug/m^3 while Table 3.1 presented a value of 4.1 (no units provided).	The background concentrations provided in Table 3.1 were the maximum measured background concentrations while the background concentrations referred to in the text were the 90th percentile background concentrations as shown in the following table.

[5] The issue of local airshed needs to be explored and defined in this assessment. As mentioned previously the 'area of concern' needs to be defined

<table>
<tr><th>#</th><th>SDB Comment</th><th>Responses</th></tr>
<tr><td></td><td>P. 48 states the background concentration for acetaldehyde is 2.3 ug/m^3 while Table 3.1 presented a value of 2.4 (no units provided).
P. 49 states the background concentration for acrolein is 0.15 ug/m^3 while Table 3.1 presented a value of 0.16 (no units provided).</td><td>90th percentile Background Concentrations in µg/m^3<table><tr><td>NO_2</td><td>70</td></tr><tr><td>$PM_{2.5}$</td><td>21</td></tr><tr><td>PM_{10}</td><td>42</td></tr><tr><td>SO_2</td><td>32</td></tr><tr><td>CO</td><td>1000</td></tr><tr><td>Acrolein</td><td>0.15</td></tr><tr><td>Benzene</td><td>2.3</td></tr><tr><td>Acetaldehyde</td><td>2.3</td></tr><tr><td>Formaldehyde</td><td>4.3</td></tr><tr><td>1,3 Butadiene</td><td>0.16</td></tr></table></td></tr>
<tr><td>61</td><td>The fact that the compounds assessed in this HHRA have similar effects on human health, and therefore may have a compounding effect, has not been discussed or assessed. Further, the tables present the risk from each pathway per compound but the total risk per individual per compound is not calculated. The result is that this underestimates the total risk. Lastly, there is no discussion of a hazard index.</td><td>Risks are not presented by pathway in Tables 5.5, 5.7, 5.9 and 5.11. In addition it is unclear what the comment on “hazard index” refers to as it is not found in the text.</td></tr>
<tr><td>62</td><td>P. 53 it is stated: “The hazard quotients associated with NO_2 for both the short-term (1 hour) and long-term (annual) were similar to background.” First, how can a ‘hazard quotient’ be similar to background – is this to mean that it is the same as the HQ of background? Please see earlier comment – if so, this means a 100% increase in the estimated hazard. Second, an annual background concentration is not provided for NO_2 (Table 3.1).</td><td>The statement means that it is similar to background hazard quotients. It does not mean 100% increase in the estimated hazard as explained several times above. The NO_2 background is 34 µg/m^3.</td></tr>
</table>

#	SDB Comment	Responses
63	A sample calculation is provided for benzene. Table C.1-2 provides the parameter values necessary for calculating exposure. The values of the parameters have not been provided for any of the other COC's assessed. Chemical and receptor-specific parameters must be provided for all COC's.	Parameter values will be provided for all COCs.
64	To be considered complete a human health risk assessment must include an example of all the calculations conducted to quantify and assess risk. This has not been done for the gaseous air pollutants in Appendix C.	An example calculation of the hazard quotient which is simply the predicted air concentration (provided in Table 3.2) divided by the TRV will be provided in Appendix C.
65	P. 9, Section 2.2 states three types of residents have been selected for this HHRA (adult, toddler and infants), however Table 2.3 outlines 5 receptors infant, toddler, child, teen, and adult. Receptor selection is not consistently defined.	All five receptors have been considered in the assessment. Section 2.2 will be corrected.
66	P. 18, section 3.3.2 a composite receptor is referred to but not defined until Table C1-3.	A composite receptor encompasses all life stages and is used to evaluate carcinogenic effects. This will be defined in Section 2.
67	P. 24 states: *"As seen from the table, some emissions from the BWGGS have both carcinogenic and non-carcinogenic properties."* It is not clear what is being communicated by the above statement. What does the acronym BWGGS stand for?	This statement is meant to refer to vehicular emissions and will be corrected.
68	P. 25; The last sentence of the second paragraph under 'Gaseous Air Pollutants' should be rephrased. It currently reads:	This will be corrected.

#	SDB Comment	Responses
	"Thus, ….to warrant changing the annual NO_2 annual guideline of 40 ug/m^3."	
69	P. 28 (& 34) It would be more logical to have the paragraphs discussing the CARB threshold value to either be sequential or combined into one paragraph.	This will be considered.
70	If there is uncertainty about an agency's averaging time (e.g., CARB) they should be contacted to determine the averaging time if the value is to be used in the assessment.	No response.
71	P. 35 & 36 why has the calculation of the HQ for gaseous air pollutants and non-carcinogenic chemicals been separated by the risk calculation for carcinogens? Further, because the calculation is the same it is not necessary to repeat 'example' equations. Equations 5-1, 5-4 and 5-5 are essentially the same.	The separation is done for different types of COCs with gaseous pollutants being considered as 1 group and VOCs as a separate group since only the inhalation route is evaluated for the gaseous pollutants and VOCs consider other exposure routes.
72	P. 39 Table 5.2 why is the column heading SO_2 when all other columns for this particular gaseous air pollutant are SO_x? Column headings should be consistent.	This will be corrected.
73	P. 41, section 5.3 states: *'long-term risks for the COC other than gaseous air pollutants identified in Section 2.2'*. COC's are identified in Section 2.1.	This will be corrected.
74	P. 43, 3rd line should refer to the incremental risk not hazard quotient.	This will be corrected.
75	P. 45 states: *"Table 5.7 presents the results for …*	It is meant to be 1,3-butadiene and not benzene.

#	SDB Comment	Responses
	exposure to benzene …" However, the remainder of this paragraph actually discusses 1,3-butadiene and Table 5.7 is labelled as 1,3-butadiene.	
76	P. 47 Table 5.9 the title of this table says formaldehyde but the row heading says 1,3-butadiene.	It is meant to be formaldehyde.
77	P. 49 states: *"It should be noted that all hazard quotients … indicating that there may be a potential risk associated with the non-carcinogenic effects of acetaldehyde."* However, the remainder of this paragraph discusses acrolein.	It is meant to be acrolein.
78	Appendix B – Terminology should remain consistent. The tables here now refer to the Parkway in presenting incremental lifetime risk and hazard quotients. Why has it been changed from TEPA? Please be consistent or provide an explanation for the change in terminology.	Parkway and TEPA are the same since the TEPA is the Technically Environmentally Preferred Alternative which is the Parkway.
79	When tables are spread over more than one page please ensure that column headers appear on each page. This was not done with Tables B.4-11, B.4-12 etc.	This is a style issue but will be considered.
80	It has not been explained why some numbers appear in bold type.	This refers to incremental risks exceeding a value of 1×10^{-6} or hazard quotient values exceeding a value of 2. Footnotes will be added to the tables.
81	Appendix C – P. 151 2nd sentence of the footnote refers to the *'non-characteristics'* of benzene. Please explain what this means.	It is meant to read the non-carcinogenic characteristics.
82	Also it would be better if table footnotes appeared on the same page as the table(s) they are referring to.	This is a style issue.