APPENDIX N

Human Health and Ecological Risk Assessment
Brandon Generating Station
Licence Review

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EXECUTIVE SUMMARY

The Brandon Generating Station (Brandon G.S.), located on the eastern boundary of the City of Brandon, on the southern shore of the Assiniboine River, is an important part of Manitoba Hydro's integrated system. This report provides a risk assessment to the Environmental Impact Statement (EIS) that has been prepared as part of the Environment Act Licence Review (EALR) for the coal-fired operation of Brandon Unit 5 (Unit #5). The report documents an evaluation of the potential health effects in people residing and/or working in the immediate area from the substances emitted to the air from the proposed facility. The emissions to water have not been considered in this risk assessment since the emissions from the Unit #5 stack will not substantially change water concentrations in the Assiniboine River; therefore this assessment focussed on emissions to air. No other pathways of potential exposure have been identified. An assessment of effects on ecological receptors was also undertaken.

Methodology

The methodology used in assessing human health risks followed guidelines outlined by various regulatory agencies including Environment Canada, Health Canada, the Canadian Council of Ministers of the Environment (CCME), and the United States Environmental Protection Agency as shown in Figure 1. Results are expressed in terms of hazard quotients and cancer risk levels for long-term exposures and in terms of concentration ratio values for short-term exposures. The hazard quotient is defined as the ratio of exposure to a long-term toxicity value and the concentration ratio is defined as the ratio of the predicted concentration to a short-term concentration protective of human health. In general, Health Canada concurs that a hazard quotient below 0.2 or a cancer risk level equal to or less than one-in-one hundred thousand ($<1 \times 10^{-5}$), is not significant. For concentration ratios, background exposure is considered, therefore, concentration ratios below 1 are considered not to be significant. Risk levels below these regulatory limits were interpreted as reflecting no significant adverse health impacts.

The potential exposure pathways for human receptors were assumed to include the inhalation of particulate matter and gaseous particles both indoors and outdoors, the ingestion of soil and dust both indoors and outdoors and the ingestion of locally (backyard garden) grown produce. These pathways are illustrated in Figure 2.

Figure 1

Human Health Risk Assessment Process

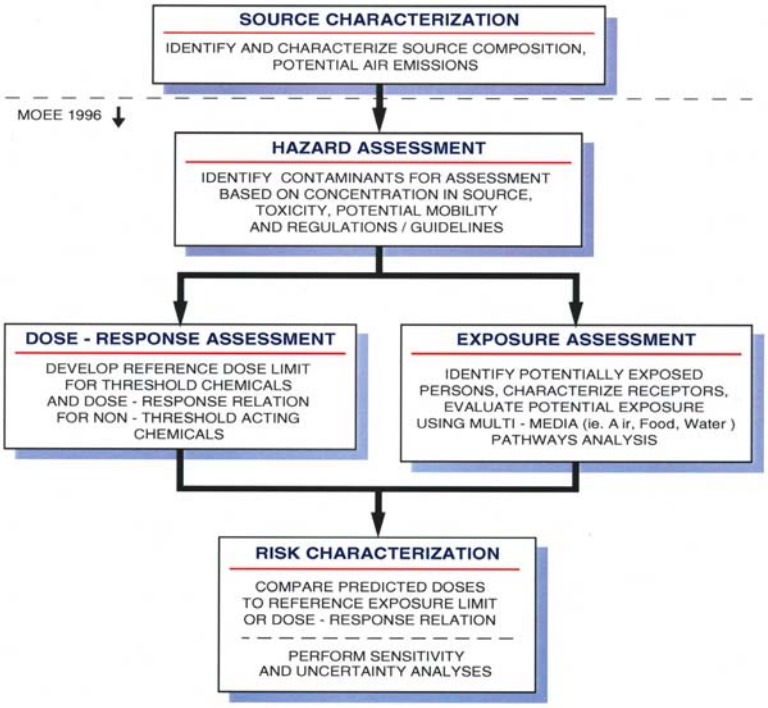
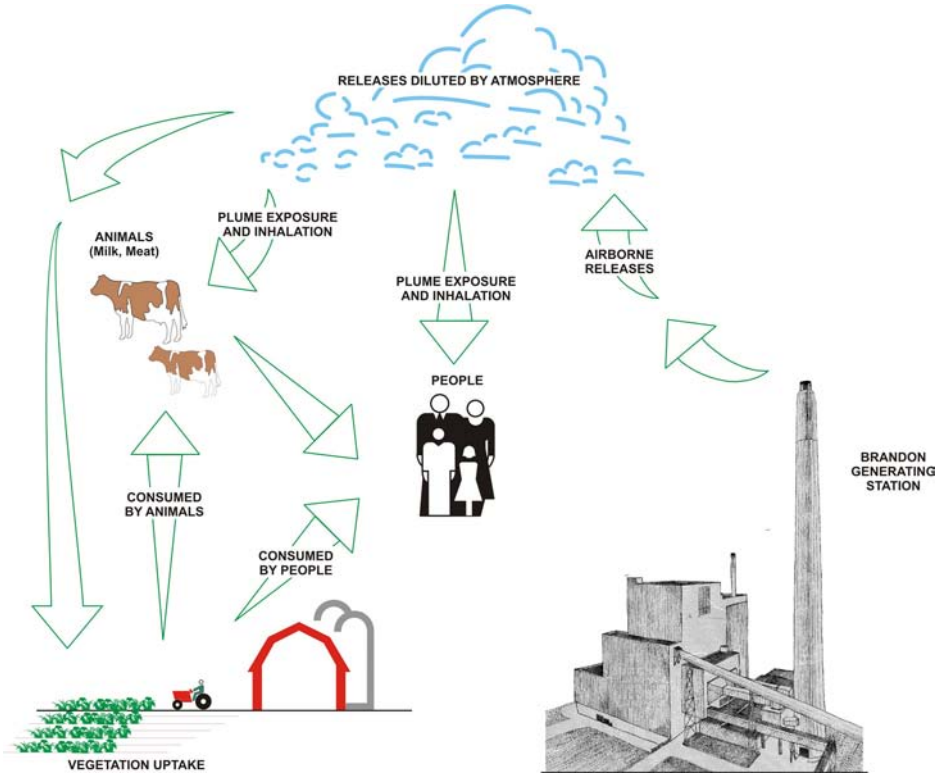


Figure 2

Conceptual Model And Potential Pathways Of Exposure For Human Receptors



The predicted long-term (on an annual basis) and short-term (1-hour and 24-hour) air concentrations are conservatively estimated from full operating conditions (i.e., 100 % capacity factor). Dispersion modelling was performed for three operating scenarios (referred to as OS1, OS2 and OS3) which represent, respectively:

- OS1 – lowest emission rates for common air contaminants under the most efficient operating conditions using current coal supplies;
- OS2 – highest emission rates for common air contaminants under the least efficient operating conditions using current coal supplies;
- OS3 – highest emission rates for all contaminants under the least efficient operating conditions using the upper-bound coal properties of alternative coal supplies that might be used in the future.

The chemicals of concern identified were combustion gases (carbon monoxide, nitrogen oxides and sulphur dioxide), volatile organic compounds, polycyclic aromatic hydrocarbons (PAHs), and particulate matter.

Short-term Effects

Potential short-term effects (e.g., respiratory health impacts) arising from Unit #5 at the Brandon G.S. were shown to be below the *a priori* concentration ratio of 1. These results indicate that no measurable short-term adverse health outcomes would be expected in sensitive individuals from the current operations of Unit #5 at the Brandon G.S. The SO_2 concentrations would not increase by more than 33 % over current levels with a switch to one of the alternative coal suppliers identified by Manitoba Hydro. The concentration ratio of SO_2 were shown to be less than 1 as long as the SO_2 concentrations remain below 33% over the current levels.

Long-term Effects

Long-term concentration ratio values for all the combustion gases, including background levels measured in Winnipeg/Brandon, are below the concentration ratio of 1. These long-term concentrations are based on the maximum predicted annual average concentrations (at the maximum point of impingement) with the plant operating at a hypothetical 100 % capacity factor. The highest concentration ratio (0.30) is obtained for NO_2. It should be noted that the WHO annual guidelines are based on the protection of the most sensitive individuals within a population. The WHO in their recent guideline for SO_2 (2005) indicated that an annual value for SO_2 is not necessary since compliance with the 24-hour guideline will assume low annual values. The predicted 24-hour SO_2 concentrations are below the most stringent SO_2 guidelines which implies that there should be no long-term adverse effects from exposure to SO_2.

Therefore, no measurable adverse health effects would occur from long-term exposure to the combustion gases emitted from Unit #5 of the Brandon G.S.

For fine particulate matter, the maximum point of impingement for 24-hour average concentrations occurs at the facility property line near the northwest corner of the plant boundary. A secondary point of elevated concentration occurs within the City of Brandon. For the fugitive dust emissions from the coal storage area, the maximum 24-hour average and annual average concentrations occur at the facility property line on the south side of the plant, while those for the ash storage area occur at the property line along the north boundary of the plant, near the Assiniboine River. Overall, the predicted adverse effects of $PM_{2.5}$ from coal-fired operations at the Brandon G.S. are negligible (i.e., below the measurement capability of $PM_{2.5}$ monitors). While the fugitive coal dust emissions potentially could exceed the PM_{10} health reference level of 25 $\mu g/m^3$ on perhaps one day per year, background PM_{10} levels in the area due to other sources are much more significant contributors to observed PM_{10} levels. Therefore, the predicted incremental $PM_{2.5}$ and PM_{10} concentrations from the Brandon G.S. are not discernable from the normal variability in existing air quality.

The cancer risk values for long-term exposure to carcinogenic chemicals, VOCs and PAHs are all below the regulatory risk level of one-in-one hundred thousand (1×10^{-5}). Therefore, it was concluded that no measurable adverse health impacts would be expected to occur in the vicinity of the Brandon G.S.

Ecological Assessment

A screening level ecological risk assessment was performed for representative ecological receptors (e.g., vegetation, as well as wild animals) to cover a range of possible exposure scenarios following the guidance set out by the CCME.

The first step of the process involved screening of the various chemicals against available CCME Soil Quality Guidelines (Parkland) and toxicity data for vegetation. The results of the screening indicated that dioxin was the only chemical to carry through the assessment as all other predicted soil concentrations were below the Soil Quality Guidelines or vegetation toxicity values.

The assessment considered terrestrial vegetation, earthworms, white-footed mice, cows, horses, robins and owls. These receptors covered a wide range of exposure. The robin and the white-footed mouse were found to be the most exposed species. The predicted exposures were low, and the assessment indicated that it is unlikely that any ecological receptors will be adversely impacted by the emissions from Unit #5 of the Brandon G.S.

Certainty of Results

An evaluation of the uncertainties in various measurements and methods used in the current assessment indicated that the risks have been over-estimated as a result of the assumptions made about exposure (which were generally cautious). The results of this uncertainty analysis support the overall conclusion that no measurable adverse impacts would occur in the human or ecological community surrounding the Brandon G.S.

Conclusion

In summary, the results of the human health and ecological risk analysis determined that there will be no incremental measurable, adverse impacts on the humans or the environment from the operation of the Unit #5 stack at the Brandon G.S.

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1.0 INTRODUCTION

The Brandon Generating Station (Brandon G.S.) operates one coal-fired boiler (Unit #5, 105 MW rated capacity). The station is located on the eastern boundary of the City of Brandon, on the south shore of the Assiniboine River. Manitoba Hydro continues to rely on non-hydroelectric resources such as Unit #5 to support, optimize, and diversify its overall portfolio of generating options. The purpose of the present risk assessment report is to support the licence review for Unit #5. The risk assessment only deals with emissions to air.

The procedures followed in the assessment were in agreement with procedures outlined by various regulatory agencies including Environment Canada, Health Canada, the Canadian Council of Ministers of the Environment, and the United States Environmental Protection Agency. These procedures include consideration of exposure through inhalation as well as ingestion of chemicals through direct deposition to food crops, as well as deposition to soils and uptake by food crops and livestock. A screening level ecological risk assessment was also carried out.

The possibility of short-term and long-term adverse human health outcomes is assessed based on exposures that would occur from predicted air concentrations at nine different receptor locations as well as at the maximum point of impingement. The locations are chosen to provide a range of exposure scenarios such as residential, park and hospital land use as well as a range of air concentrations. This is done to ensure that the maximum point of exposure is captured as well as other levels of exposure. Conservative estimates of exposure are used in the assessment to ensure that risks are not underestimated.

The predicted long-term (on annual basis) and short-term (1-hour and 24-hour) air concentrations are conservatively estimated from full operating conditions (i.e., 100 % capacity factor). There is the possibility that Manitoba Hydro may have to purchase sub-bituminous coal from other mines; therefore, the stack emission rates for some trace inorganic elements, as well as the sulphur and ash content, may be higher than those from the current sub-bituminous coal (i.e., from the Spring Creek mine). A survey of coal quality from a total of 10 alternative coal sources was used to identify the range of possible emission rates that may result from the use of other coal sources. The potential maximum emission rates from the use of alternative sources of coal were used to define the Operating Scenario 3 (OS3), for Unit #5 operations. This set of properties for OS3 is not intended to represent a specific coal from a specific mine, but rather is intended to account for the upper-bound range of properties associated with potential use of alternative coal suppliers. This set of properties therefore provides a conservative estimate of the emissions that would result if Manitoba Hydro must switch coal suppliers in the future. In this assessment, the predicted long-term air concentrations were estimated for the least efficient operating scenario (burner row combination ABC) using the worst combination of the

characteristics of future coals from 10 alternative mines. The potential long-term risks were only assessed for this scenario, which is termed Operating Scenario 3 (OS3) (see Section 3.0 of the Air Quality Assessment Report in Appendix K for details). In addition, the maximum concentrations of trace elements in coal and ash from alternative coal sources were also used to determine the modelled air concentration from fugitive coal and ash emissions.

The predicted short-term air concentrations of combustion gases were based on the characteristics of current coal from the Spring Creek mine under the most (described as Operating Scenario 1 (OS1)) and the least efficient operating scenarios (Operating Scenario 2 (OS2)), as described in the Air Quality Assessment Report in Appendix K. The OS1 is based on burner row combination BCD, and the OS2 is based on burner row combination ABC. Emissions are somewhat different depending on which rows of burner combination are used. However, emissions generated from using burner row combination A, B, and C are the highest.

Table 1.1-1 summarizes the air contaminants, burner row combinations and coal properties that were evaluated for each of the three "Operating Scenarios".

Table 1.1-1
Operating Scenarios Considered in the Risk Assessment

Operating Scenario (OS)		Air Contaminants	Description
Preferred Burner Combination	OS1	CO, NO_2, SO_2, SPM, PM_{10} & $PM_{2.5}$	Burner row combination BCD using current coal properties for ash and sulphur content
Alternate Burner Combination	OS2	CO, NO_2, SO_2, TSP, PM_{10} & $PM_{2.5}$	Burner row combination ABC using current coal properties for ash and sulphur content
Upper Bound Emission Estimate + Future Coal	OS3	SO_2, TSP, PM_{10}, $PM_{2.5}$, trace organic & inorganic constituents	Burner row combination ABC using upper bound future coal properties for ash, sulphur content and trace inorganic constituents

The chemicals of concern emitted from the facility are combustion gases (carbon monoxide, nitrogen oxides and sulphur dioxide), volatile organic compounds (benzene and formaldehyde), metals, polycyclic aromatic hydrocarbons (PAHs) and particulate matter.

The human health assessment results are expressed as deterministic hazard quotients and cancer risk levels for long-term exposures as well as concentration ratio values for both short-term and long-term exposures to combustion gases. In general, regulatory agencies such as Health Canada concur that a hazard quotient or concentration ratio values below 0.2 per pathway or an incremental cancer risk level of one in one hundred thousand (1×10^{-5}) are not significant.

The current human health assessment was divided into four different steps as provided in the various regulatory frameworks. They are the problem formulation stage, in which the various chemicals of concern, receptors, exposure pathways, and scenarios are identified. The second step in the process involves the exposure assessment where predicted exposures are calculated for the various receptors and chemicals of concern. The third step involves the determination of exposure limits for the chemicals of concern and is known as the hazard assessment. The final step in the process is an integration of the exposure and hazard assessment steps and is termed risk characterization.

1.1 SITE CHARACTERIZATION

The Brandon G.S. is located on the eastern outskirts of the City of Brandon, on the southern shore of the Assiniboine River. Land use on the north shore of the river is primarily agricultural. Residential and commercial areas are located a few kilometres to the west. Industrial developments are located to the south of the plant, while agricultural and undeveloped rangeland is located to the east. The closest residence is located approximately 1 km northwest of the plant. Agriculture Canada operates a research station on the western outskirts of the City of Brandon, approximately 7 km to the northwest of the Brandon G.S.

There are two large natural areas near the Brandon G.S including the Brandon Hills, which is located 13 km south of the site, and the Douglas marshes approximately 15 km east of Brandon area. The Brandon Hills are the largest tract of forested land near the site and is surrounded by different habitats such as prairie, parkland, and boreal forest. The Douglas Marsh area has a large wetland area that is habitat to a large number of aquatic birds. There are also some residual tracts of tall-grass prairie habitat. The Kemnay Sand Hills area located approximately 22 km west of the plant is a recreational area. Spruce Woods Provincial Park is located over 45 km east of the Brandon G.S. The park encloses unique habitats of mixed-grass prairie, riverbank forests, and shifting sand dunes which are home to cactus and rare species of snakes.

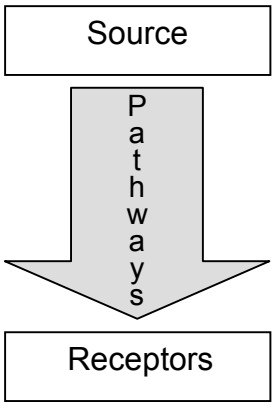
2.0 SCREENING FOR CHEMICALS OF CONCERN

A wide array of chemicals has been analyzed in the emissions from the Brandon G.S. The following screening procedure is used to determine a final list of chemicals of potential concern (COPCs) that need to be evaluated in more detail in the human health and ecological risk assessment. The procedure used to develop the list of COPCs followed the Chemical Selection Criteria outlined by the United States Environmental Protection Agency (U.S. EPA) in *Risk Assessment Guidance for Superfund* (U.S. EPA 1991*)*. Before performing the screening, the source of the chemicals needs to be characterized.

2.1 SOURCE CHARACTERIZATION

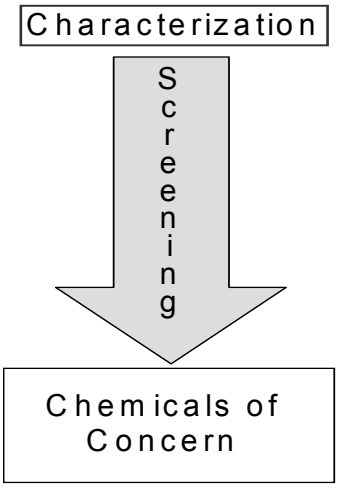
This first task characterized: (i) the source, (ii) potential pathways for movement of chemicals from the source to receptors, and (iii) potential human and ecological receptors (see Figure 2.1-1). The types and concentrations of chemicals in the air emissions were identified from recent stack testing as well as typical emission data provided by the U.S. EPA (AP-42). Potential human and ecological receptors were identified by examining current land use around the Brandon G.S. The information provided in the 1992 EIA (SENES 1992) was used to aid in the identification of potential ecological receptors.

Figure 2.1-1
Source-Pathway-Receptor Conceptual Model



The Source-Pathway-Receptor Conceptual Model was then reduced to the major chemicals of concern, major pathways and critical receptors via a screening process. Figure 2.1-2 illustrates the process used to reduce the list of chemicals identified from the source and to identify those of greatest potential concern.

Figure 2.1-2
Task 2: Screening



The screening of chemicals of concern was carried out using the recommended U.S. EPA screening procedure. Pathways were grouped according to source/exposure combinations and the most significant combinations were selected for evaluation. Representative human and ecological receptors were selected based on the locations of the most exposed receptors. In this screening, a child receptor at the highest exposed location(s) was chosen. A child receptor was chosen since this is the most potentially sensitive receptor. For ecological receptors, the

availability of literature data on toxicity and exposure was also considered in the receptor selection. There is not a lot of toxicological information available for many terrestrial ecological receptors including horses, rabbits, cows, foxes and vegetative species. In general, toxicity data for mice or rats are scaled up to larger species; generic vegetation toxicity values are used to assess impacts on trees, shrubs, flowers, crops and grasses. Thus, a mouse was chosen as a representative terrestrial mammal for this assessment because there is a large toxicological information base available. Additionally, mice have relevance in the food chain as a species that consumes soil and grass and their low body weight increases their sensitivity to contaminant levels. Generic vegetation data was used to screen for trees, shrubs, and grasses.

2.1.1 Chemical Releases

The sources of airborne chemical releases are the stack, the coal pile and the ash lagoon. The chemicals emitted to the air by the stack are sulphur dioxide (SO_2), nitrogen oxides (NO_2 and NO) and particulate matter. The particulate matter contains trace metals as well as trace polycyclic aromatic hydrocarbons such as benzo(a)pyrene.

The following table (Table 2.1-1) outlines the various chemicals that were measured in the stack gases[1] as well as other chemicals that were provided by the U.S. EPA AP-42 *Guidance on Coal-Fired Operations*. Predicted concentrations at the maximum point of impingement location are provided in the table. These concentrations were based on OS3. This location is chosen since it represents an area where the maximum concentrations of the chemicals occur. These concentrations have been derived from air quality modelling as described in the Appendix K of the Environmental Impact Statement (EIS).

Table 2.1-1
Calculated Air Concentrations At Maximum Point Of Impingement For Operating Scenario 3

Chemical	Air Concentration ($\mu g/m^3$)			
	Stack	Coal	Ash	Total
Particulate Matter	4.12×10^{-2}	0.541	1.26	1.84
PM10	3.83×10^{-2}	0.235	1.12	1.38
PM2.5	2.35×10^{-2}	0.104	0.108	0.229
Sulphur Dioxide	0.71	0.00	0.00	0.537
Oxides of Nitrogen	0.41	0.00	0.00	0.644
Carbon Monoxide	5.14×10^{-2}	0.00	0.00	5.14×10^{-2}
HF	4.76×10^{-4}	0.00	0.00	4.76×10^{-4}
Hydrochloric acid	5.19×10^{-4}	0.00	0.00	5.19×10^{-4}
Aluminum	2.23×10^{-3}	3.49×10^{-3}	5.52×10^{-2}	6.09×10^{-2}
Antimony	3.90×10^{-7}	9.19×10^{-7}	3.58×10^{-6}	4.89×10^{-6}

[1] ORTECH Environmental, 2005. Comprehensive Stack Gas Emission Testing Program at Manitoba Hydro, Brandon Generating Station, Unit No. 5. Prepared for Manitoba Hydro, Winnipeg, MB.

Table 2.1-1 (Cont'd)
Calculated Air Concentrations at Maximum Point of Impingement

Chemical	Air Concentration ($\mu g/m^3$)			
	Stack	Coal	Ash	Total
Arsenic	4.63×10^{-6}	8.11×10^{-7}	2.39×10^{-5}	2.93×10^{-5}
Barium	1.04×10^{-3}	4.44×10^{-4}	5.79×10^{-3}	7.28×10^{-3}
Beryllium	5.77×10^{-7}	3.24×10^{-7}	8.23×10^{-6}	9.14×10^{-6}
Boron	1.78×10^{-4}	2.70×10^{-5}	5.80×10^{-4}	7.85×10^{-4}
Bismuth	0.00	0.00	3.03×10^{-7}	3.03×10^{-7}
Cadmium	4.58×10^{-7}	1.46×10^{-7}	1.16×10^{-6}	1.76×10^{-6}
Calcium	0.00	5.66×10^{-3}	1.55×10^{-1}	1.61×10^{-1}
Chromium	1.68×10^{-5}	3.32×10^{-6}	1.81×10^{-4}	2.01×10^{-4}
Cobalt	1.01×10^{-6}	1.62×10^{-6}	1.30×10^{-5}	1.57×10^{-5}
Copper	2.56×10^{-5}	7.82×10^{-6}	2.40×10^{-4}	2.74×10^{-4}
Iron	8.22×10^{-4}	2.84×10^{-3}	1.77×10^{-2}	2.14×10^{-2}
Lead	5.28×10^{-6}	1.70×10^{-6}	3.19×10^{-5}	3.89×10^{-5}
Lithium	1.82×10^{-5}	2.76×10^{-6}	0.00	2.09×10^{-5}
Magnesium	0.00	1.38×10^{-3}	2.39×10^{-2}	2.53×10^{-2}
Manganese	2.60×10^{-5}	1.36×10^{-5}	7.28×10^{-4}	7.67×10^{-4}
Elemental Mercury (gas)	0	0.00	0.00	0.00
Oxidized Mercury (gas)	0	0.00	0.00	0.00
Particle-bound Mercury	5.01×10^{-9}	4.86×10^{-8}	1.24×10^{-7}	1.78×10^{-7}
Molybdenum	1.32×10^{-5}	1.48×10^{-6}	1.18×10^{-5}	2.65×10^{-5}
Nickel	1.35×10^{-5}	2.57×10^{-6}	1.46×10^{-4}	1.62×10^{-4}
Palladium	2.60×10^{-6}			2.60×10^{-6}
Phosphorous	0.00	8.15×10^{-6}	0.00	8.15×10^{-6}
Potassium	0.00	2.51×10^{-4}	2.87×10^{-5}	2.80×10^{-4}
Selenium	3.89×10^{-6}	6.76×10^{-7}	2.02×10^{-6}	6.59×10^{-6}
Silver	1.18×10^{-6}	2.76×10^{-7}	3.22×10^{-6}	4.68×10^{-6}
Sodium	0.00	1.63×10^{-3}	1.81×10^{-2}	1.97×10^{-2}
Strontium	2.12×10^{-4}	2.36×10^{-4}	3.53×10^{-3}	3.97×10^{-3}
Thallium	1.21×10^{-5}	6.00×10^{-7}	1.96×10^{-5}	3.23×10^{-5}
Thorium	4.22×10^{-6}	1.16×10^{-6}	0.00	5.39×10^{-6}
Tin	1.26×10^{-4}	2.26×10^{-6}	2.65×10^{-5}	1.55×10^{-4}
Titanium	0.00	2.28×10^{-4}	3.74×10^{-3}	3.97×10^{-3}
Uranium	6.67×10^{-7}	1.85×10^{-6}	4.83×10^{-5}	5.08×10^{-5}
Vanadium	1.91×10^{-5}	1.17×10^{-5}	3.45×10^{-4}	3.76×10^{-4}
Zinc	3.58×10^{-6}	7.46×10^{-6}	6.83×10^{-5}	7.93×10^{-5}
Acetaldehyde	3.52×10^{-5}	0.00	0.00	3.52×10^{-5}
Acetophenone	9.28×10^{-7}	0.00	0.00	9.28×10^{-7}
Acrolein	1.79×10^{-5}	0.00	0.00	1.79×10^{-5}
Benzene	8.04×10^{-5}	0.00	0.00	8.04×10^{-5}
Benzyl chloride	4.33×10^{-5}	0.00	0.00	4.33×10^{-5}
di-(2-Ethylhexyl) phthalate	4.51×10^{-6}	0.00	0.00	4.51×10^{-6}
Bromoform	2.41×10^{-6}	0.00	0.00	2.41×10^{-6}
Carbon disulfide	8.04×10^{-6}	0.00	0.00	8.04×10^{-6}

Table 2.1-1 (Cont'd)
Calculated Air Concentrations at Maximum Point of Impingement

Chemical	Air Concentration (µg/m^3)			
	Stack	Coal	Ash	Total
2-Chloroacetophenone	$4.33x10^{-7}$	0.00	0.00	$4.33x10^{-7}$
Chlorobenzene	$1.36x10^{-6}$	0.00	0.00	$1.36x10^{-6}$
Chloroform	$3.65x10^{-6}$	0.00	0.00	$3.65x10^{-6}$
Cumene	$3.28x10^{-7}$	0.00	0.00	$3.28x10^{-7}$
Cyanide	$1.55x10^{-4}$	0.00	0.00	$1.55x10^{-4}$
2,4-Dinitrotoluene	$1.73x10^{-8}$	0.00	0.00	$1.73x10^{-8}$
Dimethyl Sulphate	$2.97x10^{-6}$	0.00	0.00	$2.97x10^{-6}$
Ethyl benzene	$5.81x10^{-6}$	0.00	0.00	$5.81x10^{-6}$
Ethyl Chloride	$2.60x10^{-6}$	0.00	0.00	$2.60x10^{-6}$
1,2-Dichloroethane	$2.47x10^{-6}$	0.00	0.00	$2.47x10^{-6}$
Ethylene Dibromide	$7.42x10^{-8}$	0.00	0.00	$7.42x10^{-8}$
Formaldehyde	$1.48x10^{-5}$	0.00	0.00	$1.48x10^{-5}$
Hexane	$4.14x10^{-6}$	0.00	0.00	$4.14x10^{-6}$
Isophorone	$3.59x10^{-5}$	0.00	0.00	$3.59x10^{-5}$
Bromomethane	$9.89x10^{-6}$	0.00	0.00	$9.89x10^{-6}$
Chloromethane	$3.28x10^{-5}$	0.00	0.00	$3.28x10^{-5}$
2-Butanone	$2.41x10^{-5}$	0.00	0.00	$2.41x10^{-5}$
Methyl Hydrazine	$1.05x10^{-5}$	0.00	0.00	$1.05x10^{-5}$
Methyl Methacrylate	$1.24x10^{-6}$	0.00	0.00	$1.24x10^{-6}$
tert Butyl methyl ether	$2.16x10^{-6}$	0.00	0.00	$2.16x10^{-6}$
Dichloromethane	$1.79x10^{-5}$	0.00	0.00	$1.79x10^{-5}$
Phenol	$9.89x10^{-7}$	0.00	0.00	$9.89x10^{-7}$
Propionaldehyde	$2.35x10^{-5}$	0.00	0.00	$2.35x10^{-5}$
Tetrachloroethylene	$2.66x10^{-6}$	0.00	0.00	$2.66x10^{-6}$
Toluene	$1.48x10^{-5}$	0.00	0.00	$1.48x10^{-5}$
1,1,1-Trichloroethane	$1.24x10^{-6}$	0.00	0.00	$1.24x10^{-6}$
Styrene	$1.55x10^{-6}$	0.00	0.00	$1.55x10^{-6}$
m-Xylene	$2.29x10^{-6}$	0.00	0.00	$2.29x10^{-6}$
Vinyl acetate	$4.70x10^{-7}$	0.00	0.00	$4.70x10^{-7}$
Acenaphthene	$4.21x10^{-7}$	0.00	0.00	$4.21x10^{-7}$
Anthracene	$1.84x10^{-8}$	0.00	0.00	$1.84x10^{-8}$
Benzo(a)anthracene	$2.63x10^{-9}$	0.00	0.00	$2.63x10^{-9}$
Benzo(k)fluoranthene	$4.97x10^{-9}$	0.00	0.00	$4.97x10^{-9}$
Biphenyl	$1.99x10^{-7}$	0.00	0.00	$1.99x10^{-7}$
Benzo(a)pyrene	$3.80x10^{-9}$	0.00	0.00	$3.80x10^{-9}$
Benzo(b)fluoranthene	$4.09x10^{-9}$	0.00	0.00	$4.09x10^{-9}$
Benzo(g,h,i)perylene	$1.17x10^{-8}$	0.00	0.00	$1.17x10^{-8}$
Chrysene (Current Scenario)	$1.11x10^{-8}$	0.00	0.00	$1.11x10^{-8}$
Dibenzo(a,h)anthracene	$2.13x10^{-10}$	0.00	0.00	$2.13x10^{-10}$
Fluoranthene	$4.97x10^{-8}$	0.00	0.00	$4.97x10^{-8}$
Fluorene	$5.84x10^{-8}$	0.00	0.00	$5.84x10^{-8}$
Indeno(1,2,3-cd)pyrene	$2.02x10^{-9}$	0.00	0.00	$2.02x10^{-9}$
Naphthalene	$1.75x10^{-7}$	0.00	0.00	$1.75x10^{-7}$

TABLE 2.1-1 (Cont'd)
Calculated Air Concentrations at Maximum Point of Impingement

Chemical	Air Concentration ($\mu g/m^3$)			
	Stack	**Coal**	**Ash**	**Total**
Phenanthrene	$3.21x10^{-7}$	0.00	0.00	$3.21x10^{-7}$
Pyrene	$3.21x10^{-8}$	0.00	0.00	$3.21x10^{-8}$
Quinoline	$2.42x10^{-9}$	0.00	0.00	$2.42x10^{-9}$
2,3,7,8-TCDD	$4.07x10^{-11}$	0.00	$2.15x10^{-10}$	$2.56x10^{-10}$
2,3,7,8-TCDF	$3.61x10^{-12}$	0.00	$2.40x10^{-10}$	$2.44x10^{-10}$

As seen from Table 2.1-1, over 90 compounds have been identified as being emitted from the Brandon G.S. To aid in the assessment, this list was reduced by a screening procedure to identify the chemicals of potential concern (COPCs).

2.2 SCREENING PROCEDURE FOR HUMAN HEALTH RISK

The procedure used to develop the list of COPCs followed the Chemical Selection Criteria outlined by the U.S. EPA in *Risk Assessment Guidance for Superfund* (U.S. EPA 1991). In summary, the selection criteria specify that:

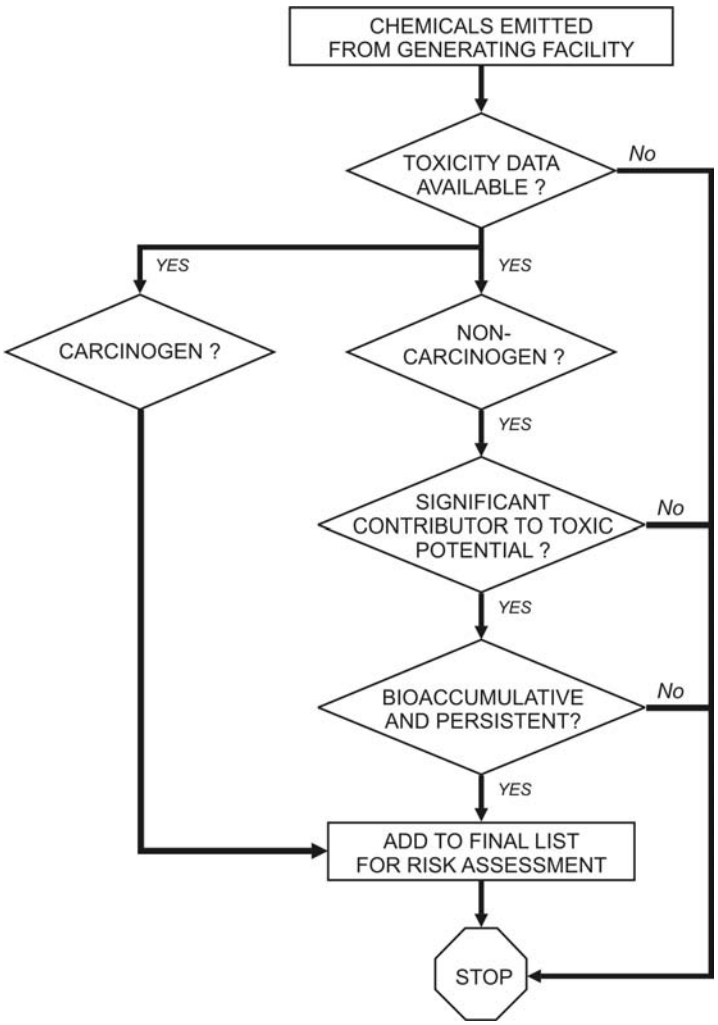
(i) chemicals present at concentrations less than the detection limit, where the detection limit is sufficiently low, are considered not present at the site;
(ii) all known or probable human carcinogens which are present must be evaluated for human health;
(iii) chemicals which have the potential to bioaccumulate and are also persistent and toxic must be evaluated;
(iv) for chemicals that have the potential to be degraded to other toxic chemicals, the breakdown products must be assessed; and
(v) where two or more chemicals that are similar in physical, chemical and biological properties and that have the same toxic end points are present (such as PAHs and dioxins and furans), it is acceptable to evaluate one representative contaminant to reduce the scope of the exercise. However, if this route is taken, modelling has to be conducted with the most toxic contaminant, using the highest concentration among the chemicals and the physical chemical properties of the most mobile chemical in the group.

2.2.1 Screening Steps

The procedure followed for screening chemicals of concern for human health is illustrated in Figure 2.2-1. As discussed previously, a child receptor is used in this process. There are 6 major steps shown in this figure:

1. Determination of the availability of toxicity data from standard referred agencies, generally the U.S. EPA.
2. Determination of the potential to break down into toxic products.
3. Assessment for carcinogenicity.
4. Determination of a toxic potential for non-carcinogenic chemicals.
5. Assessment of ability of non-carcinogenic chemicals to be persistent or bioaccumulate.
6. Determination of a final list of COPCs for further assessment.

Figure 2.2-1
Screening Procedure for Human Health



These steps are discussed in more detail in subsequent sections.

2.2.2 Determination of the Availability of Toxicity Reference Values

In this step, toxicity reference values (TRVs) are obtained from published toxicity assessments by reputable regulatory agencies. Regulatory agencies such as Health Canada, the United States Environmental Protection Agency (U.S. EPA) and the World Health Organization (WHO) were consulted. This study made no attempt to derive any toxicity data from the primary literature. The regulatory agencies provided TRV information based on the following:

- **Slope Factor (SF)** - (for carcinogens) comprises a plausible upper bound estimate of the probability of a response per unit intake of a contaminant over a lifetime. For carcinogens, no threshold is assumed to exist (i.e., every dose presents some risk); or
- **Reference Dose (RfD)** - (for non-carcinogens) comprises an estimate of the daily exposure level for a chemical for the entire population, including sensitive receptors, that is not anticipated to present an appreciable risk of an adverse effect during a portion of a lifetime.

Some of the sources of information for the TRVs were:

- **IRIS** - The U.S. EPA's on-line database (Integrated Risk Information System, IRIS) was a prime source of information. This database is regularly updated by the U.S. EPA;
- **HEAST** - The data contained in Health Effects Assessment Summary Tables (HEAST) were used to supplement the toxicological data. These tables are issued bi-annually by the U.S. EPA;
- **Health Canada** – Health Canada (HC) has provided a list of TRVs for a number of chemicals that are potentially found at federally contaminated sites. This list provides no endpoints for chemical exposure and is not updated on a regular basis;
- **ATSDR** – The data used in the Agency for Toxic Substances and Disease Registry (ATSDR) were used in the absence of toxicological data from the above sources; and
- **NCEA** – United States Environmental Protection Agency National Centre for Environmental Assessment. These data were used in the absence of toxicological data from the above reputable regulatory agencies.

When data is available from more than one information source, a chain of precedence is established. The IRIS database is the first choice; if data are not available in IRIS then other potential sources considered included HEAST and HC. The IRIS database is generally chosen over the HC and HEAST because this database is updated on a more frequent basis. However, all toxicity data are examined and the most conservative estimate of toxicity was used from the

sources reviewed. Data from ATSDR and NCEA are only used if these other sources had no data available. Nominal values used to estimate reference dose from reference concentrations are the standard factors of 70 kg body weight, an inhalation rate of 20 m^3/d and a drinking water intake of 2 L/d.

Available toxicity reference values for most of the identified chemicals are provided in order to review the relevant data and examine which chemicals are carcinogenic or non-carcinogenic. A summary of the toxicity reference values for each of the chemicals for oral and inhalation pathways is provided in Table 2.2-1. Where both non-carcinogenic and carcinogenic effects are documented for a chemical, both the RfD and SF are given. It is important to emphasize that in the development of Table 2.2-1, only TRVs available from the reputable regulatory agencies are used. As seen from the table, the majority of the TRVs were obtained from the U.S. EPA IRIS database which also provides endpoints for adverse effects.

Table 2.2-1
Toxicity Reference Values for Chemicals Emitted
From the Brandon G.S.

	SF Oral		RfD Oral		SF Inhalation		RfD Inhalation	
	1/(mg/kg-d)		(mg/kg-d)		1/(mg/kg-d)		(mg/kg-d)	
Aluminium	n/a		1.00	n	n/a		$1.40x10^{-3}$	n
Antimony	n/a		$4.00x10^{-4}$	i	n/a		n/a	
Arsenic	1.5	i	$3.00x10^{-4}$	i	$1.51x10^{1}$	i	n/a	
Barium	n/a		0.07	i	n/a		$1.43x10^{-4}$	h
Beryllium	n/a		0.002	i	8.40	i	$5.71x10^{-6}$	i
Boron	n/a		0.2	i	n/a		$5.71x10^{-3}$	h
Bismuth	n/a		n/a		n/a		n/a	
Cadmium	n/a		$5.00x10^{-4}$	i	6.30	i	n/a	
Calcium	n/a		n/a		n/a		n/a	
Chlorine	n/a		n/a		n/a		n/a	
Chromium (assume total)	n/a		n/a		$4.20x10^{1}$	i	n/a	
Chromium III	n/a		1.50	i	n/a		n/a	
Chromium VI	n/a		0.003	i	$2.90x10^{2}$	i	$2.20x10^{-6}$	i
Cobalt	n/a		0.02	*	n/a		$8.57x10^{-6}$	A
Copper	n/a		0.04	h	n/a		n/a	
Fluorine	n/a		n/a		n/a		n/a	
Iron	n/a		$3.00x10^{-1}$	n	n/a		n/a	
Lead	n/a		0.0036	HC	n/a		n/a	
Lithium	n/a		n/a		n/a		n/a	
Magnesium	n/a		n/a		n/a		n/a	
Manganese	n/a		$1.40x10^{-1}$	i	n/a		$1.40x10^{-5}$	i
Mercury (elemental)	n/a		n/a		n/a		$8.60x10^{-5}$	i
Mercury and compounds	n/a		$3.00x10^{-4}$	i	n/a		$3.00x10^{-4}$	i
Molybdenum	n/a		$5.00x10^{-3}$	i	n/a		n/a	

	SF Oral		RfD Oral		SF Inhalation		RfD Inhalation	
	1/(mg/kg-d)		(mg/kg-d)		1/(mg/kg-d)		(mg/kg-d)	
Nickel	n/a		$2.00x10^{-2}$	i	n/a		n/a	
Palladium	n/a		n/a		n/a		n/a	
Phosphorus	n/a		n/a		n/a		n/a	
Potassium	n/a		n/a		n/a		n/a	
Selenium	n/a		$5.00x10^{-3}$	i	n/a		n/a	
Silver	n/a		$5.00x10^{-3}$	i	n/a		n/a	
Sodium	n/a		n/a		n/a		n/a	
Strontium	n/a		$6.00x10^{-1}$	i	n/a		n/a	
Thallium (using Thallium nitrate toxicity value)	n/a		$9.00x10^{-5}$	i	n/a		n/a	
Thorium	n/a		n/a		n/a		n/a	
Tin	n/a		$6.00x10^{-1}$	h	n/a		n/a	
Titanium	n/a		4.00	n	n/a		$8.60x10^{-3}$	n
Uranium	n/a		$2.00x10^{-4}$	n	n/a		n/a	
Vanadium	n/a		$9.00x10^{-3}$	i	n/a		n/a	
Zinc	n/a		$3.00x10^{-1}$	i	n/a		n/a	
Acetaldehyde	n/a		n/a		$7.70x10^{-3}$	i	$2.57x10^{-3}$	i
Acetophenone	n/a		$1.00x10^{-1}$	i	n/a		n/a	
Acrolien	n/a		$2.00x10^{-2}$	h	n/a		$5.71x10^{-6}$	i
Benzene	$5.50x10^{-2}$	i	$4.00x10^{-3}$	i	$2.70x10^{-2}$	i	$8.60x10^{-3}$	i
Benzyl chloride	$1.70x10^{-1}$	i	n/a		$1.70x10^{-1}$	r		
Bis(2-ethylhexyl)phthalate (DEHP)	$1.40x10^{-2}$	i	$2.00x10^{-2}$	i	$1.40x10^{-2}$	r	$2.00x10^{-2}$	r
Bromoform	$7.90x10^{-3}$	i	$2.00x10^{-2}$	i	$3.85x10^{-3}$	i	$2.00x10^{-2}$	r
Carbon disulphide	n/a		$1.00x10^{-1}$	i	n/a		$2.00x10^{-1}$	i
2-Chloroacetophenone	n/a		$8.60x10^{-6}$	r	n/a		$8.57x10^{-6}$	i
Chlorobenzene	n/a		$2.00x10^{-2}$	i	n/a		$1.70x10^{-2}$	n
Chloroform	n/a		$1.00x10^{-2}$	i	$8.05x10^{-2}$	i	$1.40x10^{-2}$	n
Cumene	n/a		$1.00x10^{-1}$	i	n/a		$1.10x10^{-1}$	i
Cyanide	n/a		$2.00x10^{-2}$	i	n/a		$8.57x10^{-4}$	i
2,4-Dinitrotoluene	n/a		$2.00x10^{-3}$	i	n/a		$2.00x10^{-3}$	r
Dimethyl Sulphate	n/a		n/a		n/a		n/a	
Ethyl benzene	n/a		$1.00x10^{-1}$	i	n/a		$2.90x10^{-1}$	i
Ethyl chloride	$2.90x10^{-3}$	n	$4.00x10^{-1}$	n	$2.90x10^{-3}$	r	2.86	i
Ethylene dichloride	n/a		n/a		n/a		n/a	
Ethylene dibromide	2.00	i	$9.00x10^{-3}$	i	2.00	i	$2.60x10^{-3}$	i
Formaldehyde	n/a		$1.50x10^{-1}$	i	$4.60x10^{-2}$	i	n/a	
Hexane (n-Hexane)	n/a		n/a		n/a		$5.71x10^{-2}$	i
Isophorone	$9.50x10^{-4}$	i	$2.00x10^{-1}$	i	$9.50x10^{-4}$	r	$2.00x10^{-1}$	r
Methylbromide	n/a		$1.40x10^{-3}$	i	n/a		$1.40x10^{-3}$	i
Methyl chloride	n/a		n/a		n/a		n/a	
Methyl ethyl ketone	n/a		$6.00x10^{-1}$	i	n/a		1.40	i
Methyl hydrazine	1.10	h	n/a		1.10	r	n/a	
Methyl methacrylate	n/a		1.40	i	n/a		$2.00x10^{-1}$	i

	SF Oral		RfD Oral		SF Inhalation		RfD Inhalation	
	1/(mg/kg-d)		(mg/kg-d)		1/(mg/kg-d)		(mg/kg-d)	
Methyl tert-butyl ether	n/a		n/a		n/a		$8.57x10^{-1}$	i
Methylene chloride	$7.50x10^{-3}$	i	$6.00x10^{-2}$	i	$1.65x10^{-3}$	i	$8.57x10^{-1}$	h
Phenol	n/a		$3.00x10^{-1}$	i	n/a		$3.00x10^{-1}$	r
Propionaldehyde	n/a		n/a		n/a		n/a	
Tetrachloroethylene	n/a		$1.00x10^{-2}$	i	n/a		$1.00x10^{-2}$	r
Toluene	n/a		$2.00x10^{-1}$	i	n/a		$1.10x10^{-1}$	i
1,1,1-Trichloroethane	n/a		$2.80x10^{-1}$	n	n/a		n/a	
Styrene	n/a		$2.00x10^{-1}$	i	n/a		$2.90x10^{-1}$	i
Xylenes	n/a		$2.00x10^{-1}$	i	n/a		$2.90x10^{-2}$	i
Vinyl acetate	n/a		1.00	h	n/a		$5.71x10^{-2}$	i
Acenaphthene	n/a		0.06	i	n/a		0.06	r
Anthracene	n/a		0.3	i	n/a		0.3	r
Benzo(a)anthracene	$7.30x10^{-1}$	n	n/a		$7.30x10^{-1}$	r	n/a	
Benzo(a)pyrene	7.30	i	n/a		7.30	r	n/a	
Benzo(b)fluoranthene	$7.30x10^{-1}$	n	n/a		$7.30x10^{-1}$	r	n/a	
Chrysene	$7.30x10^{-3}$	n	n/a		$7.30x10^{-3}$	r	n/a	
Indeno(1,2,3-cd)pyrene	$7.30x10^{-1}$	n	n/a		$7.30x10^{-1}$	r	n/a	
Benzo(k)fluoranthene	0.073	n	n/a		0.073	r	n/a	
Biphenyl (using 1,1 biphenyl)	n/a		0.05	i			0.05	r
Benzo(g,h,i)perylene	n/a		n/a		n/a		n/a	
Dibenzo(a,h)anthracene	7.30	n	n/a		7.30	r	n/a	
Fluoranthene	n/a		$4.00x10^{-2}$	i	n/a		$4.00x10^{-2}$	r
Fluorene	n/a		0.04	i	n/a		0.04	r
Naphthalene	n/a		$2.00x10^{-2}$	i	n/a		$8.57x10^{-4}$	i
Phenanthrene	n/a		n/a		n/a		n/a	
Pyrene	n/a		$3.00x10^{-2}$	i	n/a		$3.00x10^{-2}$	r
Quinoline	3	i	n/a		3	r	n/a	
Total Dioxins (using 2,3,7,8-TCDD)	$1.50x10^{5}$	h	n/a		$1.50x10^{5}$	h	n/a	
Total Furans (using 2,3,7,8-TCDF)	n/a		$1.00x10^{-3}$	i	n/a		$1.00x10^{-3}$	r

Note:

i- IRIS (U.S. EPA 2005a)
n- NCEA (U.S. EPA 2001)
h- HEAST (U.S. EPA 1997)
r- route to route extrapolation
HC-Health Canada (2004)
A –ATSDR (2001)
*- U.S. EPA Region 6 (2005b)
n/a- data not available

Chemicals that had no toxicity data (see Table 2.2-1) reported from the above agencies are not carried through the quantitative screening procedure. These are bismuth, calcium, chlorine, fluorine, lithium, magnesium, palladium, titanium, ethylene dichloride, methyl chloride and

propionaldehyde. Benzo(g,h,i)perylene and phenanthrene do not have toxicity data, but are generally assessed using benzo(a)pyrene as a surrogate. The omission of these chemicals adds to the uncertainty in the screening assessment and the predicted total risk estimate since not all detected chemicals were included. A discussion on uncertainties is included in this assessment. Essential human nutrients such as lithium, iron, magnesium, phosphorus, potassium, and sodium as well as calcium (which do not have toxicity data), need not be considered further in the assessment according to the U.S. EPA (1991).

Health Canada (2004) recommends an oral Total Daily Intake (TDI) for 2,3,7,8-TCDD-TEQ of 2×10^{-9} mg/kg-d. HEAST treats dioxins as carcinogenic compounds. To be conservative, dioxins are considered as carcinogenic compounds in this assessment.

As seen from the tables above, mercury is emitted from the stacks at the Brandon G.S; however the values are very low. Mercury is a naturally occurring element that is generally found in low concentrations in air, water, and soil. Therefore, mercury residues are naturally present to some degree in plant, fish, animal, and human tissues. Mercury levels of fish in the Assiniboine River are reported to be elevated and there is a fish consumption restriction for fish caught in the Assiniboine River. The incremental increase in mercury in the Assiniboine River and consequently in the fish tissue due to the emissions from the Brandon G.S. stacks are predicted to be so small that they would result in levels in the river and in fish that would not be measurable. Therefore, mercury is not considered further in the assessment.

2.2.3 Determination of the Potential to Break Down into Toxic Products

Biodegradation of chemicals can reduce their concentrations and produce breakdown products. This can be an important consideration if the breakdown products are more toxic than the original chemicals. This is generally not the case and concentrations decrease within each step in the degradation process so that even a more toxic breakdown product does not always increase risk.

Biodegradation depends on the size and composition of the microbial population, the pH, salinity or temperature of the water, redox conditions, the presence of essential elements and nutrients and the concentration of the contaminant. As well, inhibitors such as a heavy metal or other toxic materials can limit the growth of micro-organisms. None of the chemicals in this assessment have the potential to break down into more toxic products.

2.2.4 Assessment for Carcinogenicity

All chemicals that are identified as carcinogens must be carried through the risk assessment for human health. Slope factors were obtained using the methodology discussed in the previous step. As seen in Table 2.2-1, eighteen of the chemicals on the list are considered to be carcinogenic. They are arsenic, beryllium, cadmium, chromium, acetaldehyde, benzene, benzyl chloride, di-(2-ethylhexyl)phthalate, bromoform, chloroform, ethyl chloride, ethylene dibromide, formaldehyde, isophorone, dichloromethane, methyl hydrazine, benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene, quinoline, and dioxins.

2.2.5 Determination of Toxic Potential for Non-Carcinogenic Chemicals

In this screening procedure, non-carcinogenic chemicals that are most likely to contribute significantly to risks are identified. Two important factors for determining potential effect for the inclusion of a chemical in the risk assessment are its measured concentration and its toxicity. Therefore, in this screening procedure a toxic potential is calculated for all chemicals other than essential nutrients with identified non-carcinogenic toxicity values. This toxic potential compares the maximum air or soil concentration to the toxicity value; both the oral and inhalation routes of exposure are assessed, where toxicity data for both routes are available. The toxic potential is not equivalent to a risk and is only used in this screening procedure to identify chemicals that would contribute significantly to the risk. The toxic potential results are shown in Attachment C.2. The screening procedure was limited to maximum point of impingement, where chemical concentrations are the highest. The procedure is briefly outlined below.

The screening of the chemicals for the child receptor uses the soil concentrations calculated from the equations presented in Attachment A. The child characteristics used for screening purposes include a body weight of 34 kg, an inhalation rate of 14 m^3/d and a soil ingestion intake of 100 mg/day.

The chemical exposure rate from soil ingestion is calculated using the following equation (2-1):

$$Chemical\ Exposure\ Rate = \frac{Chemical\ Concentration\ in\ Soil \times Soil\ Ingestion\ Rate}{Body\ Weight} \qquad (2\text{-}1)$$

where:

chemical exposure rate	= mg chemical/(kg bw day);
chemical concentration in soil	= mg chemical/kg soil for tilled and forage soil;
soil ingestion rate	= 100 mg soil/day for a child receptor;

body weight = 34 kg for a child receptor.

This calculated exposure rate is compared directly to the oral TRVs, as discussed in Section 2.2.1 and presented in Table 2.2-1. If the exposure rate from soil ingestion is greater than the oral TRV, then this non-carcinogenic chemical is carried through to the human health assessment.

For the inhalation pathways, the predicted air concentrations at the maximum point of impingement are directly compared to the inhalation toxicity reference concentration. If the estimated concentration in air is greater than the reference concentration, the non-carcinogenic chemical is carried through the assessment. The reference concentration is calculated from the inhalation TRV using the following equation (2-2):

$$Reference\,Concentration = \frac{RfD_i \times Body\,Weight}{Inhalation\,Rate} \times \frac{1000\,\mu g}{1\,mg} \times \frac{1\,d}{24\,hr} \qquad (2\text{-}2)$$

where:

reference concentration = µg chemical/m^3;
RfD_i = reference dose (mg chemical/(kg body weight/d));
inhalation rate = 0.58 m^3/hr for a child receptor;
body weight = 34 kg for a child receptor.

From Attachment C, it can be seen that all non-carcinogenic chemicals are dropped from further assessment since their toxic potentials are well below one (1).

2.2.6 Assessment of Ability of Non-Carcinogenic Chemicals to be Persistent or Bioaccumulate

Two factors that are considered before the final list of COPCs is determined are the persistence and bioaccumulation of the chemicals. To assess bioaccumulation, the following criteria suggested by Environment Canada are employed (Environment Canada and Health Canada, 1994):

- Bioconcentration Factor (BCF) or Bioaccumulation Factor (BAF) >500
- $3 < \text{Log } K_{ow} < 7$, where K_{ow} = octanol-water partition coefficient

Chemicals that are identified as having no potential to bioaccumulate, and are found not to be persistent are dropped from the list of chemicals.

Persistence is evaluated according to the criteria suggested by Environment Canada (Environment Canada and Health Canada 1994):

- half life in air, surface water and soil >50 days
- half life in groundwater >100 days
- half life in sediment >180 days

None of the non-carcinogenic chemicals are added to this list.

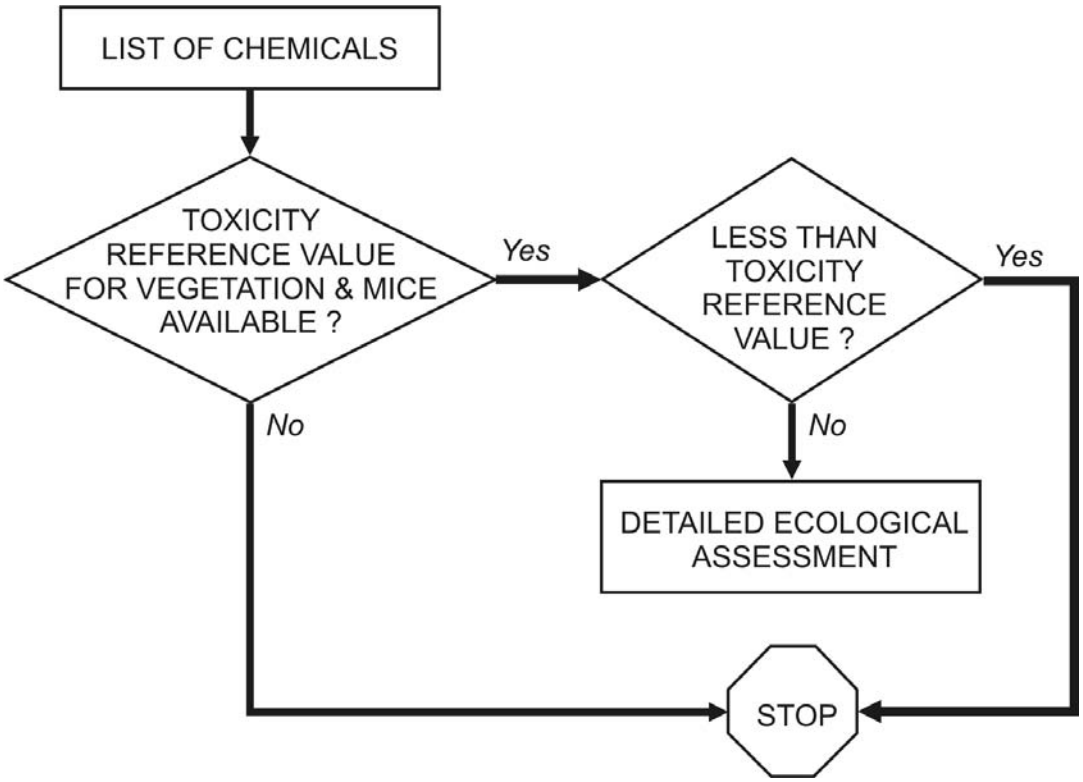
2.3 SCREENING FOR ECOLOGICAL RISK

This section discusses the screening procedure for ecological receptors. As discussed previously, toxicity data for vegetation are used to screen for trees, shrubs, crops and grasses and toxicity data for mice are used to screen for terrestrial ecological receptors.

2.3.1 Approach to Screening

The first step in the selection of chemicals for detailed ecological evaluation is to use a screening process similar to that for human health. The purpose of the screening is to determine which of the chemicals measured in air pose a potential to be toxic to ecological receptors. This step was carried out as shown in Figure 2.3-1.

Figure 2.3-1
Illustration of Screening Ecological Assessment



Two major groups of ecological receptors potentially exposed to the chemicals were identified. These included terrestrial vegetation and terrestrial animals. In this screening process, a white-footed mouse was used for the terrestrial ecological receptor and generic plant species were used for the terrestrial vegetation. Therefore, the first step in this process is to determine the assessment endpoints for the various chemicals based on these two receptors. Assessment endpoints are an explicit expression of the actual environment value that is to be protected (U.S. EPA 1999). To evaluate an assessment endpoint, measures of effects are determined from available literature and may include measures related to impaired reproduction, growth and survival. These measures of effects are compiled from various sources including the U.S. Department of Energy database (Efroymson *et al.* 1997; Sample *et al*. 1996; Suter and Tsao 1996), the Canadian Environmental Quality Guidelines (CCME 2003) and PSL documents.

These screening ecotoxicity values (TRVs) are usually based on generic assessment endpoints such as the protection of communities from changes in structure or function and have been assumed to be applicable to this study. Table 2.3-1 outlines the available screening ecotoxicity values of the chemicals emitted from the coal-fired operations of the Brandon G.S. TRVs used, when available, for the screening processes are No Observable Adverse Effect Levels (NOAELs) for a mouse species, and CCME soil guidelines or vegetation TRVs.

Table 2.3-1
Toxicity Reference Values for Ecological Risk Assessment

Chemical	Mouse TRV	Vegetation TRV	Chemical	Mouse TRV	Vegetation TRV
	(mg/kg-d)	(mg/kg soil)		(mg/kg-d)	(mg/kg soil)
Aluminum	2.086	50[a]	Cyanide	128.9	0.9
Antimony	0.135	5[a]	2,4-Dinitrotoluene	n/a	n/a
Arsenic	0.136	17	Dimethyl Sulphate	n/a	n/a
Barium	10.8	2000[b]	Ethyl benzene	n/a	1.2
Beryllium	1.32	10[a]	Ethyl chloride	n/a	n/a
Boron	55.9	0.5[a]	Ethylene dichloride	n/a	n/a
Bismuth	n/a	n/a	Ethylene dibromide	n/a	n/a
Cadmium	1.926	10	Formaldehyde	45.4	n/a
Calcium	n/a	n/a	Hexane	n/a	n/a
Chlorine	n/a	n/a	Isophorone	n/a	n/a
Chromium	5466	64	Methylbromide	n/a	n/a
Chromium VI	6.55	n/a	Methyl chloride	n/a	n/a
Cobalt	n/a	20[a]	Methyl ethyl ketone	3537	n/a
Copper	30.4	63	Methyl hydrazine	n/a	n/a
Fluorine	n/a	n/a	Methyl methacrylate	n/a	n/a
Iron	n/a	n/a	Methyl tert-butyl ether	n/a	n/a
Lead	15.98	300	Methylene chloride	11.7	n/a
Lithium	18.8	2[a]	Phenol	n/a	20

Chemical	Mouse TRV	Vegetation TRV
	(mg/kg-d)	(mg/kg soil)
Magnesium	n/a	n/a
Manganese	176	500[a]
Mercury	2.6	12
Molybdenum	0.28	2[a]
Nickel	79.89	50
Palladium	n/a	n/a
Phosphorus	n/a	n/a
Potassium	n/a	n/a
Selenium	0.399	1
Silver	n/a	2[a]
Sodium	n/a	n/a
Strontium	525	n/a
Thallium	0.015	1.4
Thorium	n/a	n/a
Tin	25.3	50[a]
Titanium	n/a	n/a
Uranium	3.261	5[a]
Vanadium	0.389	130
Zinc	319.5	200
Acetaldehyde	n/a	n/a
Acetophenone	n/a	n/a
Acrolien	n/a	n/a
Benzene	28.5	0.5
Benzyl chloride	n/a	n/a
Bis (2-ethylhexyl)phthalate (DEHP)	19.8	n/a
Bromoform	n/a	n/a
Carbon disulphide	n/a	n/a
2-Chloroacetophenone	n/a	n/a
Chlorobenzene	n/a	n/a
Chloroform	30	n/a
Cumene	n/a	n/a

Chemical	Mouse TRV	Vegetation TRV
	(mg/kg-d)	(mg/kg soil)
Propionaldehyde	n/a	n/a
Tetrachloroethylene	1.51	0.2[b]
Toluene	28.1	0.8
1,1,1-Trichloroethane	1123	n/a
Styrene	n/a	300[a]
Xylenes	2.269	1[b]
Vinyl acetate	n/a	n/a
Acenaphthene	1.08	n/a
Anthracene	1.08	n/a
Benzo(a)anthracene	1.08	1.2[c]
Benzo(a)pyrene	1.08	0.7
Benzo(b)fluoranthene	n/a	1.2[c]
Benzo(k)fluoranthene	1.08	1.2[c]
Benzo(ghi)perylene	n/a	n/a
Biphenyl	1.08	1.3
Chrysene	n/a	1.2[c]
Dibenzo(a,h)anthracene	1.08	1.2[c]
Fluorene	1.08	n/a
Fluoranthene	n/a	n/a
Indeno(1,2,3-cd)pyrene	n/a	1.2[c]
Naphthalene	n/a	0.6
Phenanthrene	n/a	n/a
Pyrene	n/a	n/a
Quinoline	1.08	n/a
2,3,7,8-TCDD	0.000002	n/a
2,3,7,8-TCDD	n/a	n/a

Note: n/a – not available

* - TRVs are based on CCME ecological component (SQG_E) guideline for parkland, unless otherwise stated (in the case value from this source is not available)

a- Will and Suter (1995)

b- CCME soil quality guidelines (2003)

c- U.S. EPA Region 6 Protocol (1999) – Terrestrial plant toxicity reference values (Table E-5)

It is important to note that ecological assessments are limited since data may not be relevant or available for all the chemicals emitted from the Brandon G.S. Thus, the availability and

appropriateness of TRVs often constrains an ecological assessment. As seen from Table 2.3-1, TRVs are not available for a number of the chemicals emitted from the Brandon G.S. These chemicals will not be assessed further in the ecological risk assessment. The lack of toxicity data on these chemicals adds to the uncertainty in the ecological assessment.

The second step in this process involves the estimation of exposure levels. In this step, the maximum predicted concentrations of chemicals at maximum point of impingement are used and the bioavailability of the chemicals was assumed to be 100 %. An Exposure Ratio (ER) approach is used which compares either the estimated contaminant intake (mg/kg body weight/d) or the estimated contaminant concentration (mg/kg soil, mg/kg food) to the NOAEL.

Thus, the Exposure Ratio (ER) can be expressed as:

$$ER = \left(\frac{Dose}{NOAEL}\right) \quad or \quad = \left(\frac{Estimated\ Concentration}{NOAEL}\right) \tag{2-3}$$

An Exposure Ratio of less than one (1) indicates that the chemical alone is unlikely to cause adverse effects and further evaluation is not indicated. For groups of chemicals, the summing of individual ERs is sometimes appropriate. A sum of less than 1 indicates that the group of chemicals is unlikely to cause adverse ecological effects and further evaluation is not indicated.

2.3.2 Screening Based on Terrestrial Vegetation

Chemical soil concentrations are calculated based on the equations provided in Attachment A. The screening process involves the comparison of the estimated soil concentrations at the maximum point of impingement (areas of maximum chemical concentration) to available Canadian Soil Quality Guidelines and vegetation TRVs. As discussed previously, this study was not required to develop toxicity data from the primary literature but to use data from existing regulatory agencies. Thus, chemicals with no CCME soil criteria but with TRVs based on vegetation from the U.S. Department of Energy database were also assessed (see Figure 2.3-2). The CCME parkland category is chosen to represent the most appropriate soil criteria. The ecological component of the criteria is protective of both plant and animal species. It must be noted that TRVs do not exist for a number of the organic chemicals and thus they cannot be assessed in the vegetation screening process. Table 2.3-2 shows the predicted soil concentration of those chemicals with available soil quality guidelines and vegetation TRVs. Since these guidelines and TRVs include both background and incremental soil concentrations, a comparison of the predicted incremental soil concentrations to 20 % of the Soil Quality

Guidelines and vegetation TRVs was done. This implies that the incremental concentrations accounts for 20 % of the total soil concentrations. As seen from Figure 2.3-2, if the maximum predicted soil concentration is below 20 % of the appropriate Soil Quality Guidelines or screening TRV for vegetation, then this chemical is dropped from further assessment. As seen from Table 2.3-2, none of the predicted soil concentrations at maximum point of impingement are above 20 % of the CCME Soil Quality Guidelines and vegetation TRVs and thus none of the chemicals need to undergo further assessment.

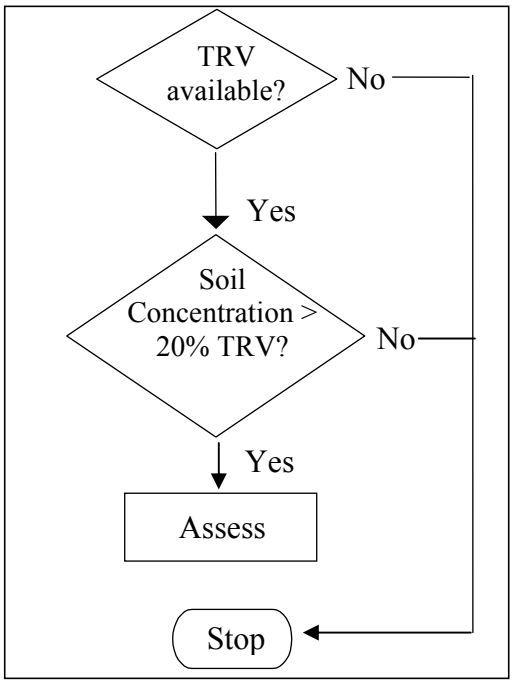
Table 2.3-2

Comparison of Maximum Predicted Soil Concentrations at Maximum Point of Impingement With Vegetation Toxicity Reference Values and Canadian Soil Quality Guidelines*

Chemicals	Vegetation TRV/Soil Quality Guidelines	Soil Concentrations	
		Tilled	Forage
	(mg/kg)	(mg/kg)	(mg/kg)
Aluminum	10	0.83	5.63
Antimony	1	1.31×10^{-5}	1.31×10^{-5}
Arsenic	3.4	1.11×10^{-10}	1.11×10^{-10}
Barium	400	0.38	2.24
Beryllium	2	1.94×10^{-8}	1.94×10^{-8}
Boron	0.1	8.76×10^{-3}	8.76×10^{-3}
Cadmium	2	7.22×10^{-10}	7.22×10^{-10}
Chromium (Total)	12.8	1.43×10^{-10}	1.43×10^{-10}
Cobalt	4	2.92×10^{-4}	5.87×10^{-4}
Copper	12.6	8.42×10^{-3}	2.52×10^{-2}
Lead	60	1.94×10^{-3}	1.17×10^{-2}
Lithium	0.4	3.87×10^{-5}	3.87×10^{-5}
Manganese	100	6.02×10^{-3}	8.56×10^{-3}
particle-bound mercury	2.4	2.34×10^{-9}	2.34×10^{-9}
Molybdenum	0.4	4.49×10^{-3}	1.58×10^{-2}
Nickel	10	4.37×10^{-3}	1.23×10^{-2}
Selenium	0.2	2.68×10^{-4}	2.69×10^{-4}
Silver	0.4	1.00×10^{-5}	1.00×10^{-5}
Thallium	0.28	3.84×10^{-3}	1.02×10^{-2}
Tin	10	0.046	0.26
Uranium	1	1.24×10^{-4}	1.49×10^{-4}
Vanadium	26	6.35×10^{-3}	1.99×10^{-2}
Zinc	40	1.00×10^{-3}	1.89×10^{-3}
Benzene	0.1	6.54×10^{-18}	6.54×10^{-18}
Cyanide	0.18	1.42×10^{-10}	1.53×10^{-10}
Ethyl benzene	0.24	3.73×10^{-18}	3.73×10^{-18}
Phenol (using pentachlorophenol chemical properties)	4	2.37×10^{-12}	1.48×10^{-11}
Tetrachloroethylene	0.04	1.42×10^{-18}	1.42×10^{-18}
Toluene	0.16	7.11×10^{-18}	7.11×10^{-18}
Styrene	60	1.33×10^{-17}	1.33×10^{-17}
m-Xylene	0.2	1.63×10^{-18}	1.63×10^{-18}
Benzo(a)pyrene	0.14	1.33×10^{-6}	1.02×10^{-5}
Benzo(b)fluoranthene	0.24	1.22×10^{-6}	3.30×10^{-6}
Chrysene	0.24	3.80×10^{-6}	2.29×10^{-5}
Indeno(1,2,3-cd)pyrene	0.24	7.07×10^{-7}	5.52×10^{-6}
Napthalene	0.12	1.96×10^{-9}	1.96×10^{-9}
Benzo(a)anthracene	0.24	9.21×10^{-7}	7.08×10^{-6}
Benzo(k)fluoranthene	0.24	1.74×10^{-6}	1.34×10^{-5}
Biphenyl	0.26	7.42×10^{-5}	5.43×10^{-4}
Dibenzo(a,h)anthracene	0.24	7.47×10^{-8}	5.74×10^{-7}

Note: *A factor of 0.2 is applied to the TRV/guidelines for the purpose of comparing to the predicted incremental soil concentrations.

Figure 2.3-2
Screening Procedure for Terrestrial Vegetation



2.3.3 Screening Based on Terrestrial Animals

Wildlife

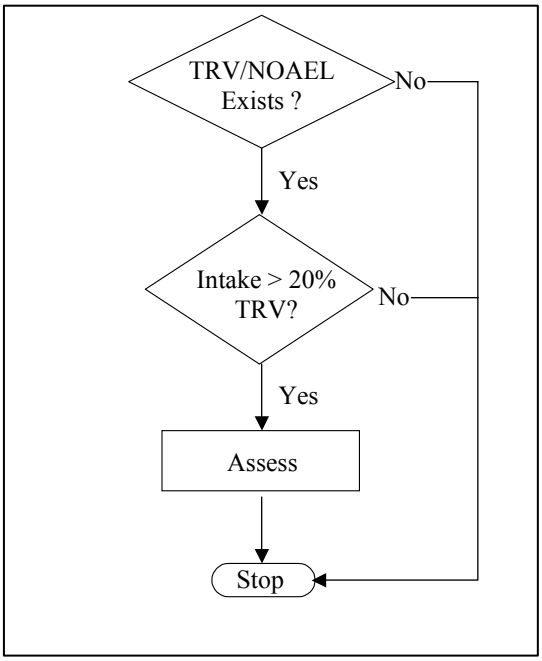
All chemicals carried forward with appropriate available TRVs for terrestrial animals are carried through to this step of the screening assessment, with the exception of those chemicals that are below the CCME soil criteria in the previous step of the assessment. The screening procedure is illustrated in Figure 2.3-3.

In this assessment, an estimate of the maximum potential intake by terrestrial mammals is necessary in order to have a comparison to the TRVs. Thus, to determine the intake, it is necessary to estimate the potential concentration in vegetation. The soil screening concentrations obtained above are converted into an estimated vegetation concentration by the use of a soil-to-vegetation transfer coefficient.

Thus,

$$\left[\begin{array}{c}\textit{Screening Vegetation}\\ \textit{Concentration (mg/kg)}\end{array}\right] = \left[\begin{array}{c}\textit{Screening Soil}\\ \textit{Concentration (mg/kg)}\end{array}\right] \times \left[\begin{array}{c}\textit{Soil to Veg Transfer}\\ \textit{Coefficient (mg/kg)}\end{array}\right]$$

Figure 2.3-3
Screening Procedure for Terrestrial Wildlife



Soil-to-vegetation transfer factors are calculated differently for metals and organic compounds. Baes *et al*. (1984) provides soil-to-vegetation elemental transfer coefficients for vegetative (B_v) and non-vegetative (B_r) portions of food crops and feed. These were adapted according to the following equation provided in SENES (1992).

$$B = 0.2\left(0.1 \times B_v + 0.9 \times B_r\right) \qquad (2\text{-}4)$$

where:

B	=	soil-vegetation transfer factor [(mg/kg vegetation)/(mg/kg soil)];
0.2	=	conversion from dry weight to wet weight;
0.1,0.9	=	approximate balance of vegetative, non-vegetative food portions in typical diet;
B_v	=	soil-vegetation transfer factor [(mg/kg vegetation)/(mg/kg soil)] for vegetative food portions;
B_r	=	soil-vegetation transfer factor [(mg/kg vegetation)/(mg/kg soil)] for non-vegetative food portions.

For organic chemicals, the soil-to-vegetation transfer factor is calculated using an equation from Travis and Arms (1988):

$$Soil - Vegetation\,Transfer\,Coefficient = 10^{(1.588 - 0.0578 \log Kow)} \qquad (2\text{-}5)$$

where:

K_{ow} = Octanol to water partition coefficient

In order to determine an intake to wildlife, namely mice, it was assumed that the diet is 100 % vegetation. This is a conservative assumption since the diet of these small mammals includes other food sources. Mice are chosen for this screening exercise since they will be directly affected by the presence of the chemicals. Food chain effects (i.e., predator species) are not assessed in this preliminary screening as the chemicals identified for assessment do not biomagnify. Intake to these animal species are calculated based on the food ingestion rate and body weight.

In other words,

$$Wildlife\ Intake\ (mg/(kg\,d)) = \frac{Screening\ Vegetation\ Concentration\ (mg/kg) \times Intake\ (kg/d)}{Body\ weight\ (kg)}$$

An exposure ratio is then calculated by dividing the *Wildlife Intake* by the TRV for mice (which is the NOAEL as discussed previously). Table 2.3-3 shows a summary of the exposure ratios for the various chemicals at the maximum point of impingement (the location of maximum soil concentration). As seen from this table, more TRVs for mice are available for organic chemicals than are available for vegetation. A TRV exists for benzo(a)pyrene, which is considered to be the most toxic of the PAHs, and thus, it is used as a surrogate for all PAHs. Similarly, the TRV for 2,3,7,8-TCDD is considered a surrogate for all dioxins and furans. From the table it can be seen that the exposure ratio values are well all below one (1) with the exception of 2,3,7,8-TCDD, therefore, all these chemicals, with the exception of 2,3,7,8-TCDD, can be dropped from further consideration.

Table 2.3-3
Exposure Ratio Values for Mice at Maximum Point of Impingement

Chemical	Predicted Intake (Tilled) (mg/kg-d)	Predicted Intake (Forage) (mg/kg-d)	Toxicity Value for Mice (mg/kg-d)	Exposure Ratio (Tilled)	Exposure Ratio (Forage)
Aluminum	$3.57x10^{-5}$	$2.43x10^{-4}$	2.09	$1.7x10^{-5}$	$1.1x10^{-4}$
Antimony	$2.70x10^{-8}$	$2.70x10^{-8}$	0.135	$2.0x10^{-7}$	$2.0x10^{-7}$
Arsenic	$4.63x10^{-14}$	$4.63x10^{-14}$	0.136	$3.4x10^{-13}$	$3.4x10^{-13}$
Barium	$4.76x10^{-4}$	$2.80x10^{-3}$	10.8	$4.4x10^{-5}$	$2.6x10^{-4}$
Beryllium	$2.00x10^{-12}$	$2.00x10^{-12}$	1.32	$1.5x10^{-12}$	$1.5x10^{-12}$
Boron	$8.44x10^{-4}$	$8.45x10^{-4}$	55.9	$1.5x10^{-5}$	$1.5x10^{-5}$
Cadmium	$6.01x10^{-12}$	$6.01x10^{-12}$	1.93	$3.1x10^{-12}$	$3.1x10^{-12}$
Chromium (Total)	$3.01x10^{-14}$	$3.01x10^{-14}$	5470	$5.5x10^{-18}$	$5.5x10^{-18}$
Copper	$9.77x10^{-5}$	$2.93x10^{-4}$	30.4	$3.2x10^{-6}$	$9.6x10^{-6}$
Lead	$1.06x10^{-6}$	$6.39x10^{-6}$	16.0	$6.6x10^{-8}$	$4.0x10^{-7}$

Chemical	Predicted Intake (Tilled) (mg/kg-d)	Predicted Intake (Forage) (mg/kg-d)	Toxicity Value for Mice (mg/kg-d)	Exposure Ratio (Tilled)	Exposure Ratio (Forage)
Lithium	$1.02x10^{-8}$	$1.02x10^{-8}$	18.8	$5.4x10^{-10}$	$5.4x10^{-10}$
Manganese	$1.85x10^{-5}$	$2.63x10^{-5}$	176	$1.1x10^{-7}$	$1.5x10^{-7}$
Mercury	$2.76x10^{-11}$	$2.76x10^{-11}$	2.60	$1.1x10^{-11}$	$1.1x10^{-11}$
Molybdenum	$1.57x10^{-5}$	$5.53x10^{-5}$	0.28	$5.6x10^{-5}$	$1.9x10^{-4}$
Nickel	$1.15x10^{-5}$	$3.23x10^{-5}$	79.9	$1.4x10^{-7}$	$4.0x10^{-7}$
Selenium	$2.94x10^{-7}$	$2.95x10^{-7}$	0.399	$7.4x10^{-7}$	$7.4x10^{-7}$
Strontium	$9.58x10^{-4}$	$1.28x10^{-3}$	525	$1.8x10^{-6}$	$2.4x10^{-6}$
Thallium	$1.26x10^{-7}$	$3.36x10^{-7}$	0.015	$8.4x10^{-6}$	$2.2x10^{-5}$
Tin	$1.71x10^{-5}$	$9.71x10^{-5}$	25.3	$6.7x10^{-7}$	$3.8x10^{-6}$
Uranium	$2.42x10^{-8}$	$2.90x10^{-8}$	3.26	$7.42x10^{-9}$	$8.91x10^{-9}$
Vanadium	$9.04x10^{-7}$	$2.83x10^{-6}$	$3.89x10^{-1}$	$2.32x10^{-6}$	$7.28x10^{-6}$
Zinc	$4.22x10^{-5}$	$7.95x10^{-5}$	$3.20x10^{2}$	$1.32x10^{-7}$	$2.49x10^{-7}$
Benzene	$4.2x10^{-17}$	$4.2x10^{-17}$	$2.9x10^{1}$	$1.5x10^{-18}$	$1.5x10^{-18}$
di-(2-Ethylhexyl) phthalate	$4.4x10^{-11}$	$3.5x10^{-10}$	$2.0x10^{1}$	$2.2x10^{-12}$	$1.8x10^{-11}$
Chloroform	$1.8x10^{-18}$	$1.8x10^{-18}$	$3.0x10^{1}$	$6.0x10^{-20}$	$6.0x10^{-20}$
Cyanide	$1.2x10^{-9}$	$1.3x10^{-9}$	$1.3x10^{2}$	$9.4x10^{-12}$	$1.0x10^{-11}$
Formaldehyde	$1.65x10^{-14}$	$1.65x10^{-14}$	45.4	$3.6x10^{-16}$	$3.6x10^{-16}$
2-Butanone	$3.82x10^{-16}$	$3.82x10^{-16}$	3540	$1.1x10^{-19}$	$1.1x10^{-19}$
Dichloromethane	$4.87x10^{-18}$	$4.87x10^{-18}$	11.7	$4.2x10^{-19}$	$4.2x10^{-19}$
Tetrachloroethylene	$8.62x10^{-18}$	$8.62x10^{-18}$	1.51	$5.7x10^{-18}$	$5.7x10^{-18}$
Toluene	$4.22x10^{-17}$	$4.22x10^{-17}$	28.1	$1.5x10^{-18}$	$1.5x10^{-18}$
1,1,1-Trichloroethane	$1.48x10^{-18}$	$1.48x10^{-18}$	1120	$1.3x10^{-21}$	$1.3x10^{-21}$
m-Xylene	$9.06x10^{-18}$	$9.06x10^{-18}$	2.27	$3.9x10^{-18}$	$3.9x10^{-18}$
Acenaphthene	$7.91x10^{-4}$	$5.78x10^{-3}$	1.08	$7.33x10^{-4}$	$5.36x10^{-3}$
Anthracene	$3.20x10^{-5}$	$2.34x10^{-4}$	1.08	$2.97x10^{-5}$	$2.17x10^{-4}$
Benzo(a)anthracene	$3.66x10^{-6}$	$2.81x10^{-5}$	1.08	$3.39x10^{-6}$	$2.60x10^{-5}$
Benzo(a)pyrene	$5.08x10^{-6}$	$3.90x10^{-5}$	1.08	$4.70x10^{-6}$	$3.61x10^{-5}$
Benzo(k)fluoranthene	$6.55x10^{-6}$	$5.04x10^{-5}$	1.08	$6.07x10^{-6}$	$4.66x10^{-5}$
Biphenyl	$3.71x10^{-4}$	$2.71x10^{-3}$	1.08	$3.43x10^{-4}$	$2.51x10^{-3}$
Dibenzo(a,h)anthracene	$2.67x10^{-7}$	$2.05x10^{-6}$	1.08	$2.47x10^{-7}$	$1.90x10^{-6}$
Fluorene	$1.06x10^{-4}$	$7.74x10^{-4}$	1.08	$9.80x10^{-5}$	$7.17x10^{-4}$
Quinoline	$5.50x10^{-6}$	$4.23x10^{-5}$	1.08	$5.09x10^{-6}$	$3.91x10^{-5}$
TCDD, 2,3,7,8-	$3.72x10^{-8}$	$7.27x10^{-8}$	$2.00x10^{-6}$	$1.86x10^{-2}$	$3.64x10^{-2}$

2.3.4 Summary of Screening Level Ecological Assessment

The results of the screening of chemicals of potential concern for ecological assessment are summarized in Tables 2.3-2 and 2.3-3. The only chemical carried through to the ecological assessment is 2,3,7,8-TCDD.

3.0 PROBLEM FORMULATION

The primary objective of this human health assessment is to determine whether the chemical concentrations emitted from the coal-fired operation of the Brandon Generating Station would likely pose unacceptable health impacts to people located in the immediate area surrounding the generating station.

The problem formulation was derived from information provided by Manitoba Hydro as well as information available from the U.S. EPA on the different chemicals that could be potentially emitted from a coal-fired generating station. Previous Environmental Impact Assessments (EIAs) of the Brandon Generating Station (SENES 1992) also served as the foundation for the assessment.

3.1 IDENTIFICATION AND SELECTION OF CHEMICALS OF CONCERN

The screening of chemicals of potential concern (COPC) was discussed in Section 2. A summary of the COPC for the human assessment are listed in Table 3.1-1.

Table 3.1-1
COPC Selected for This Assessment

Combustion Gases	**Volatile Organic Compounds**
Carbon Monoxide (CO)	Acetaldehyde
Nitrogen Oxides (NO_x)	Benzene
Sulphur dioxide (SO_2)	Benzyl chloride
	di-(2-Ethylhexyl) phthalate
Metals	Bromoform
Arsenic	Chloroform
Beryllium	Ethyl Chloride
Cadmium	Ethylene Dibromide
Chromium	Formaldehyde
	Isophorone
Dixoins	Methyl Hydrazine
2,3,7,8-TCDD	Dichloromethane
Particulate Matter	**Polycyclic Aromatic Hydrocarbons**
Total Suspended Particulate	Benzo(a)pyrene
PM_{10}	Benzo(b)fluoranthene
$PM_{2.5}$	Chrysene
	Indeno (1,2,3-cd) pyrene
	Benzo(a)anthracene
	Benzo(k)fluoranthene
	Dibenzo(a,h)anthracene
	Quinoline

The predicted long-term air concentrations (annual averages) of these chemicals are estimated by modeling the theoretical maximum operating conditions (i.e., 100 % capacity factor). The predicted short-term air concentrations (1-hr and 24-hr) were conservatively estimated at the maximum sustained generation rates.

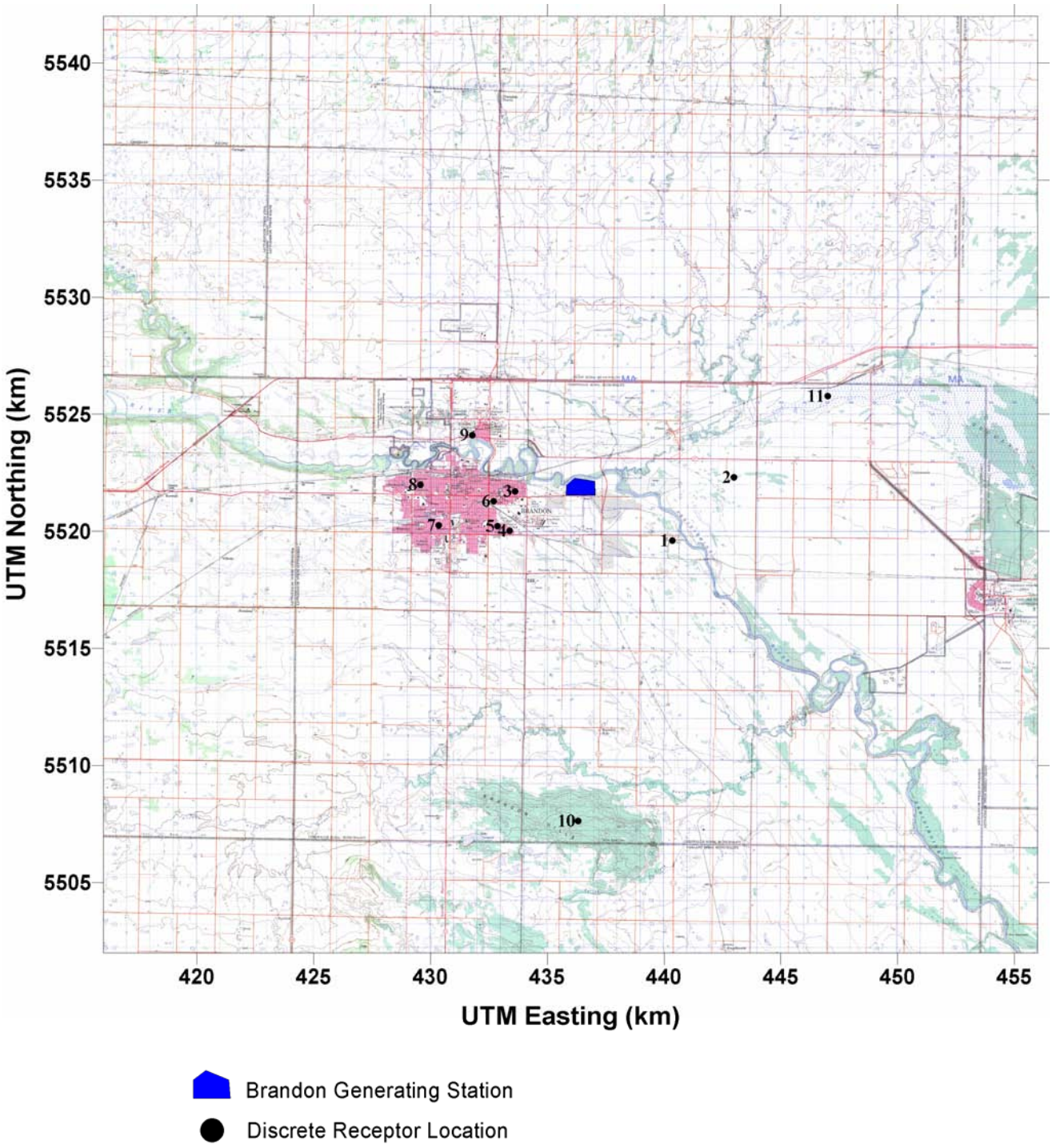
3.2 RECEPTOR SELECTION

Nine locations plus the maximum point of impingement (POI) were chosen for receptors that are most likely to be impacted by the emissions from Unit #5 at the Brandon G.S. These locations are representative of a range of different exposure scenarios. These receptor locations are shown in Figure 3.2-1. The POI location is considered in the assessment of short-term (acute) effects. Four categories of human receptors have also been selected (resident, industrial worker, school, and hospital/health centre patient); only adult and composite receptors (an individual present at that location all their life) were considered in the assessment as only carcinogenic chemicals were being evaluated. A summary of the receptor locations and categories of receptors are shown in Table 3.2-1.

Table 3.2-1
Summary of Locations and Receptors

Receptor Location	Description	Category of Receptors
R1	Chemical Plant (industrial receptor)	Adult Worker
R2	Residence at eastern edge of Brandon	Adult and Composite Resident
R3	Riverview Elementary	Adult and Composite
R4	Inglewood St. Residence	Adult and Composite Resident
R5	Green Acres Elementary School	Adult and Composite
R6	Hospital	Adult and Composite
R7	Meadows Elementary School	Adult and Composite
R8	Valleyview Elementary School	Adult and Composite
R9	Kirkaldy Heights Elem/Jr. High School	Adult and Composite
R9	Agriculture Land	Plant/Crops
R10	Hill 15 km South of Brandon	Ecological Receptor
R11	Marshy Area East of Brandon	Ecological Receptor
	Maximum point of impingement	Adult and Composite Resident, Infant, Short-Term Exposure for Sensitive Receptors

Figure 3.2-1
Receptor Locations



Receptor Location 1 is a chemical plant. Potential exposure to adult workers was conservatively assumed to estimate the maximum exposure in this area.

Receptor Locations 2 and 4 are grouped together as residences with adult and composite receptors. An infant is included at the hypothetical residence of maximum impingement of emissions from the generating station. As this location represents the maximum potential exposure at a residence, the dose to the infant at the maximum POI represents the maximum possible dose to an infant in the entire study area. The maximum points of impingement of emissions from the generating station also represent the maximum short-term (acute) exposure location. Receptor locations 10 and 11 are only considered in the ecological risk assessment as those are ecological areas.

In addition to residences, schools are present at Receptor Locations 3, 5, 7 and 8. Therefore, the exposure to adults outside the school for 8 hours a day, 5 days a week is also considered in this assessment. Since only carcinogenic effects are being evaluated, it was assumed that a child going to the school would also work at the school in later years and thus the composite receptor evaluated considers childhood exposure.

A hospital is located at Receptor Location 6. For this receptor location, an adult patient is assumed to be in the hospital 24 hours a day, 365 days a year.

3.3 HUMAN RECEPTOR CHARACTERIZATION

The assumptions of the behavioural characteristics for the human receptors are shown in Table 3.2-2 and their physical characteristics are presented in Tables 3.2-3 to 3.2-7. The assumed lifestyle characteristics are conservative and represent a range of exposures. The different exposure pathways (water, meat, soil, vegetation, etc.) will be discussed in more detail in Section 4.1. The exposure durations in Table 3.2-3 to 3.2-7 have been chosen to provide a conservative estimate (over-estimate) of exposure. Exposure to infants is discussed separately in Section 4.3.3.

Table 3.2-2
Assumed Lifestyle Characteristics
of the Human Receptors

Receptor	Ingestion of Water	Ingestion of Meat	Ingestion of Milk	Ingestion of Vegetation	Ingestion of Soil
Receptor 1 (Industrial Worker)	Assumed to drink municipal water	Assumed to not eat any beef from study area	Assumed to not drink any milk from study area	Assumed to not eat any produce from study area	Assumed to ingest soil from study area
Receptor 2 (Resident)	Assumed to drink municipal water	Assumed to obtain 5 % of beef from study area	Assumed to obtain 5 % of milk from study area	Assumed to obtain 5 % of produce from study area	Assumed to ingest soil from study area
Receptor 3 (School/Park)	Assumed to drink municipal water	Assumed to not eat any beef from study area	Assumed to not drink any milk from study area	Assumed to not eat produce from study area	Assumed to ingest soil from study area
Receptor 4 (Resident)	Assumed to drink municipal water	Assumed to obtain 5 % of beef from study area	Assumed to obtain 5 % of milk from study area	Assumed to obtain 5 % of produce from study area	Assumed to ingest soil from study area
Receptor 5 (School/Park)	Assumed to drink municipal water	Assumed to not eat any beef from study area	Assumed to not drink any milk from study area	Assumed to not eat produce from study area	Assumed to ingest soil from study area
Receptor 6 (Hospital)	Assumed to drink municipal water	Assumed to not eat any beef from study area	Assumed to not drink any milk from study area	Assumed to not eat produce from study area	Assumed to ingest soil from study area
Receptor 7 (School/Park)	Assumed to drink municipal water	Assumed to not eat any beef from study area	Assumed to not drink any milk from study area	Assumed to not eat produce from study area	Assumed to ingest soil from study area
Receptor 8 (School/Park)	Assumed to drink municipal water	Assumed to not eat any beef from study area	Assumed to not drink any milk from study area	Assumed to not eat produce from study area	Assumed to ingest soil from study area
Receptor 9 (School/Park)	Assumed to drink municipal water	Assumed to not eat any beef from study area	Assumed to not drink any milk from study area	Assumed to not eat produce from study area	Assumed to ingest soil from study area

Table 3.2-3
Summary of Receptor Characteristics
Adult Industrial Worker & School

Parameter	Units	Value	Reference	Fraction from Location
Breathing Rate	(m^3/d)	20	Richardson 1997	1.0
Soil Ingestion	(mg/d)	80	Richardson 1997	1.0
Meat Ingestion	(g/d)	182	Richardson 1997	0
Milk Ingestion	(g/d)	181	ATG 1999	0
Vegetation Ingestion	(g/d)	339	Richardson 1997	0
Body Weight	(kg)	70	Richardson 1997	-
Time Spent at Location				**Duration of Exposure**
8 hr/day		260 day/yr		30 yrs

Table 3.2-4
Summary of Receptor Characteristics
Adult Resident

Parameter	Units	Value	Reference	Fraction from Location
Breathing Rate	(m^3/d)	20	Richardson 1997	1.0
Soil Ingestion	(mg/d)	80	Richardson 1997	1.0
Meat Ingestion	(g/d)	182	Richardson 1997	0.05
Milk Ingestion	(g/d)	181	ATG 1999	0.05
Vegetation Ingestion	(g/d)	339	Richardson 1997	0.05
Body Weight	(kg)	70	Richardson 1997	-
Time Spent at Location				**Duration of Exposure**
24 hr/day		365 day/yr		70 yrs

Table 3.2-5
Summary of Receptor Characteristics
Child Resident

Parameter	Units	Value	Reference	Fraction from Location
Breathing Rate	(m^3/d)	14	Richardson 1997	1.0
Soil Ingestion	(mg/d)	100	Richardson 1997	1.0
Meat Ingestion	(g/d)	123	Richardson 1997	0.05
Milk Ingestion	(g/d)	353	ATG 1999	0.05
Vegetation Ingestion	(g/d)	259	Richardson 1997	0.05
Body Weight	(kg)	34	Richardson 1997	-
Time Spent at Location				**Duration of Exposure**
24 hr/day		365 day/yr		30 yrs

Table 3.2-6
Summary of Receptor Characteristics
Child Park (School)

Parameter	Units	Value	Reference	Fraction from Location
Breathing Rate	(m^3/d)	28.8	Richardson 1997	1.0
Soil Ingestion	(mg/d)	500	Richardson 1997	1.0
Meat Ingestion	(g/d)	123	Richardson 1997	0
Milk Ingestion	(g/d)	353	ATG 1999	0
Vegetation Ingestion	(g/d)	259	Richardson 1997	0
Body Weight	(kg)	34	Richardson 1997	-

Time Spent at Location		Duration of Exposure
8 hr/day	260 day/yr	30 yrs

Table 3.2-7
Summary of Receptor Characteristics
Adult Hospital

Parameter	Units	Value	Reference	Fraction from Location
Breathing Rate	(m^3/d)	14	Richardson 1997	1.0
Dust Ingestion	(mg/d)	0.56	Richardson 1997	1.0
Meat Ingestion	(g/d)	182	Richardson 1997	0
Milk Ingestion	(g/d)	181	ATG 1999	0
Vegetation Ingestion	(g/d)	339	Richardson 1997	0
Body Weight	(kg)	70	Richardson 1997	-

Time Spent at Location		Duration of Exposure
24 hr/day	365 day/yr	70 yrs

4.0 EXPOSURE ASSESSMENT

The primary objective of the exposure assessment is to predict, using a series of conservative assumptions, the potential exposure to COPCs (in mg/kg-d) for the receptors at the nine different locations via the pathways identified in the following section (Section 4.1).

The concentrations of the different chemicals of concern were obtained by air dispersion modelling for the air pathways and pathways modelling for the soil pathways. A detailed discussion of the air dispersion modelling is provided in the Air Quality Assessment Report (Appendix K). A detailed discussion of the pathways model used to determine the exposures of humans to chemicals of concern is provided in Attachment A. A worked example for one COPC has also been provided (Attachment C) to illustrate how these equations were used to develop the exposures at the various receptor locations. Similar calculations were performed for all the COPCs at the different receptor locations. Only chemical- and receptor-specific parameters were modified accordingly.

4.1 EXPOSURE PATHWAYS

The potential pathways of exposure (Figure 4.1-1) for each of the receptor types were assumed to be the following: the inhalation of particulate matter and gaseous particles - outdoors; the ingestion of soil and dust outdoors; the ingestion of locally grown produce; the ingestion of beef; and the ingestion of cow's milk. It was assumed that drinking water was from a municipal source not impacted by emissions from the coal-fired generating station. Specific assumptions for each receptor location are outlined in Table 3.2-2. The pathways are described in more detail below.

The various pathways evaluated were as follows:

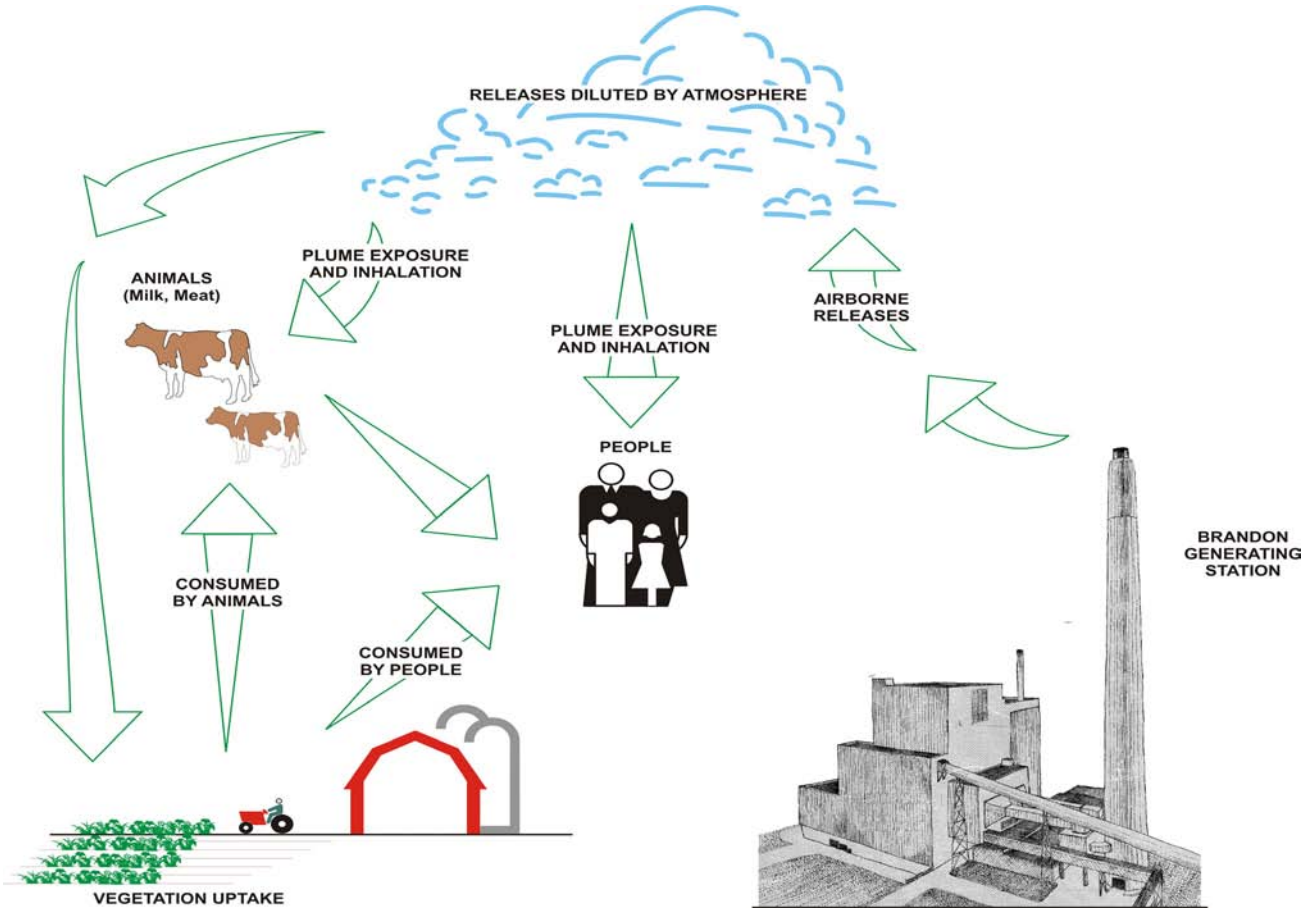
- Inhalation of Air: The emission of small amounts of chemicals of concern from the coal-fired operation at the Brandon G.S. will result in the direct exposure of the human population as the plume impinges down onto the ground level. Human receptors will therefore inhale both gaseous and particle-borne chemicals while outdoors.
- Inhalation of Soils and Dusts: Human exposure may occur through inhalation of soils and dusts outdoors as the gaseous and particle-borne chemicals deposit onto soils and surfaces. The rate of this deposition is a function of the local meteorological conditions such as wind speed and precipitation rates.
- Ingestion of Locally Grown Produce: As chemicals are deposited from air-borne emissions, they may contact leaves and fruit of locally grown (backyard gardens) produce, where they may remain on the surface or may be absorbed into the plant. Deposition of chemicals onto the soil may also result in accumulation in plants via root uptake. Humans are exposed to these chemicals by eating the produce from their backyard gardens. Cows are also exposed

by grazing on potentially contaminated vegetation. Humans are then exposed by consumption of locally grown beef and milk.

- Ingestion of Breast Milk: It is assumed that infants in residences around the Brandon G.S. would be exposed to chemicals via the breast milk of their mothers. It is assumed that the mothers would be exposed to the chemicals of concern via the consumption of locally grown produce as well as the inhalation of air and ingestion of soil and dust. This exposure pathway was only assessed for chemicals of concern with log $K_{ow} > 4$. Only benzo(a)pyrene falls into this category and will only be assessed at the receptor location where the maximum point of impingement occurs. Any other receptor location would result in a much lower exposure.
- Ingestion to Soils and Dusts: Human exposure may occur through ingestion of soils and dusts outdoors as the gaseous and particle-borne chemicals deposit onto soils and surfaces.
- Dermal Exposure to Soils and Dusts can be a pathway of exposure; however, soil concentrations in this assessment are very low and thus dermal exposure will be limited. Therefore, this pathway was not considered in the assessment.

Figure 4.1-1

Conceptual Model and Potential Pathways of Exposure for Human Receptors



4.2 Predicted Concentrations

Air dispersion modelling of emissions from the Unit #5 operations at the Brandon G.S. was carried out as described in the the Air Quality Assessment Report. (Appendix K) The predicted long-term and short-term air concentrations were estimated from the full operating conditions (i.e., operating at a theoretical maximum 100 % capacity factor).

Table 4.2-1a provides the maximum concentrations of the combustion gases at the maximum POI (see details in the Air Quality Assessment Report – Appendix K) for all three operating scenarios. Concentrations for CO, NOx, and NO_2 are identical for scenarios OS2 and OS3. On the other hand, predicted concentrations for particulate matter and SO_2 differ between the three operating scenarios. This is because future operations (OS3) of the Brandon G.S. may have to rely on coal purchases from other mines that might result in higher ash and sulphur content of the coal burned at the plant. Therefore, emissions of SO_2 and particulate matter may be higher in the future. The maximum predicted SO_2, SPM, PM_{10} and $PM_{2.5}$ concentrations for hypothetical future operations based on alternative coal sources are listed in Table 4.2-1b.

Table 4.2-1a

Maximum Predicted Ambient Air Concentrations for Current Operations of Unit #5 at the Maximum Point of Impingement

Emission	Maximum Incremental Concentration (µg/m^3)			
	1-hr	8-hr	24-hr	Annual Average
OS1 – Burner Row Combination B, C, D				
CO	16.1	3.5	na	0.04
NO_2 (Janssen Method)	91	na	7.9	0.14
NO_2 (100 % conversion of NO to NO_2)	243	na	17.8	0.65
SO_2	190	na	13.9	0.51
Total PM	6.9	na	0.5	0.019
PM_{10}	6.4	na	0.5	0.017
$PM_{2.5}$	4.1	na	0.3	0.011
OS2 – Burner Row Combination A, B, C				
CO	19.1	4.2	na	0.05
NO_2 (Janssen Method)	119	na	10.4	0.2
NO_2 (100 % conversion of NO to NO_2)	322	na	23.6	0.9
SO_2	200	na	14.6	0.5
Total PM	11.4	na	0.8	0.03
PM_{10}	10.6	na	0.8	0.03
$PM_{2.5}$	6.5	na	0.5	0.02

Note: na — not applicable.

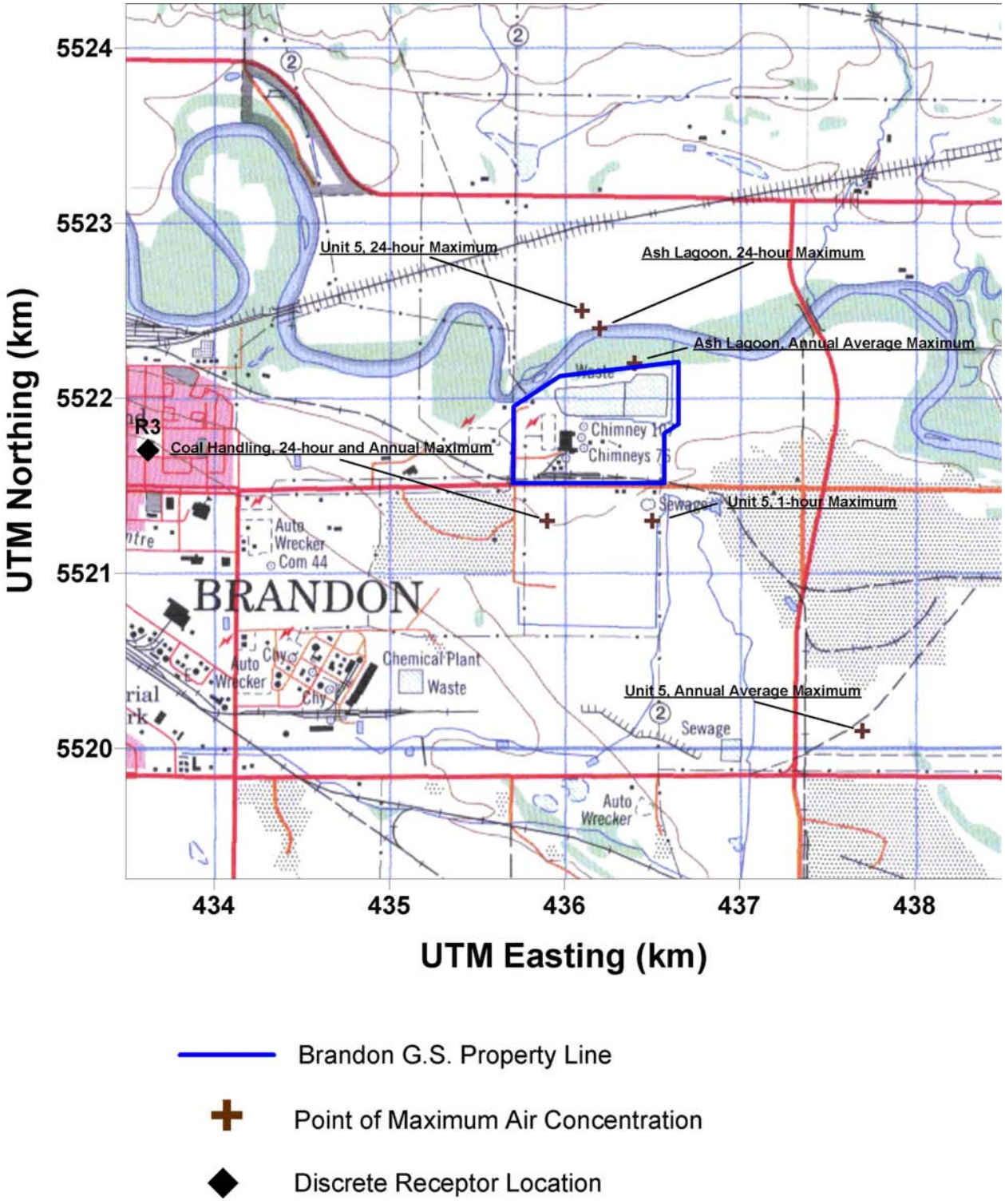
Table 4.2-1b
Maximum Predicted Ambient Air Concentrations for Future Operations of Unit #5 at the Maximum Point of Impingement

Emission	Maximum Incremental Concentration ($\mu g/m^3$)		
	1-hr	**24-hr**	**Annual Average**
OS3 – Burner Row Combination A, B, C			
SO_2	265.5	19.5	0.7
Total PM	15.4	1.1	0.04
PM_{10}	14.2	1.0	0.04
$PM_{2.5}$	8.7	0.6	0.02

Note:Only SO_2 and PM change with the future coal sources , ther CO and NO_x concentrations are the same as for the A,B, C bin combination provided in Table 4.2-1a.

The maximum point of impingement based on the annual average air concentration from the Unit #5 combustion stack occurs approximately 2 km southeast of the generating station**.** On the other hand, the maximum point of impingement for fugitive dust from the coal and ash storage areas occur at different locations along the facility property line (Figure 4.2-1). Nevertheless, for the sake of simplicity and to provide a conservative estimate of potential health risks, the maximum predicted annual average concentrations for the volatile organic compounds, PAHs and metals at the maximum point of impingement are assumed to occur at the same location. These maximum concentrations for the scenario OS3 are presented previously in Table 2.1-1. Maximum concentrations for the other receptor locations are provided in Attachment B.

Figure 4.2-1
Points Of Maximum Impingement for Unit #5 And Coal Handling/Processing Emissions, for Averaging Periods of Interest



4.3 EXPOSURE RATES

The predicted exposure rates for the nine different receptor locations and the maximum POI location based on annual or long-term exposure are provided in this section.

4.3.1 Inhalation

The estimated dose from inhalation is calculated based on the air concentrations of the chemicals of concern and these doses are provided in Attachment B. The equations used for calculating the exposure from inhalation are provided in detail in Attachment A. A summary of the inhalation dose (mg/kg-d) for each receptor location is given in Table 4.3-1 for the OS3. As expected, the dose from the inhalation pathway is the greatest for the composite and adult at the maximum POI location.

Table 4.3-1
Calculated Inhalation Dose (mg/kg-d) for All Receptor Locations– Operating Scenario 3

(mg/kg-d)	Receptor 1	Receptor 2		Receptor 3	Receptor 4		Receptor 5
	Industrial Worker	Resident		School	Resident		School
COPC	Adult	Adult	Composite	Adult	Adult	Composite	Adult
Arsenic	$1.29x10^{-9}$	$1.91x10^{-9}$	$2.04x10^{-9}$	2.10E-10	$6.43x10^{-10}$	$6.87x10^{-10}$	$2.31x10^{-10}$
Beryllium	$2.70x10^{-13}$	$4.01x10^{-13}$	$4.29x10^{-13}$	3.31E-11	$8.97x10^{-11}$	$9.60x10^{-11}$	$3.32x10^{-11}$
Cadmium	$2.91x10^{-13}$	$4.32x10^{-13}$	$4.62x10^{-13}$	1.93E-11	$6.16x10^{-11}$	$6.58x10^{-11}$	$2.19x10^{-11}$
Chromium (Total)	$7.89x10^{-13}$	$1.17x10^{-12}$	$1.25x10^{-12}$	8.81E-10	$2.49x10^{-9}$	$2.67x10^{-9}$	$9.14x10^{-10}$
Acetaldehyde	$1.43x10^{-13}$	$2.13x10^{-13}$	$2.28x10^{-13}$	1.37E-09	$4.60x10^{-9}$	$4.91x10^{-9}$	$1.63x10^{-9}$
Benzene	$3.02x10^{-15}$	$4.68x10^{-15}$	$5.00x10^{-15}$	3.12E-09	$1.05x10^{-8}$	$1.12x10^{-8}$	$3.71x10^{-9}$
Benzyl chloride	$1.87x10^{-13}$	$2.78x10^{-13}$	$2.97x10^{-13}$	1.68E-09	$5.64x10^{-9}$	$6.04x10^{-9}$	$2.00x10^{-9}$
di-(2-Ethylhexyl) phthalate	$3.53x10^{-13}$	$5.25x10^{-13}$	$5.61x10^{-13}$	1.75E-10	$5.89x10^{-10}$	$6.29x10^{-10}$	$2.08x10^{-10}$
Bromoform	$1.52x10^{-14}$	$2.25x10^{-14}$	$2.41x10^{-14}$	9.35E-11	$3.14x10^{-10}$	$3.36x10^{-10}$	$1.11x10^{-10}$
Chloroform	$1.72x10^{-13}$	$2.56x10^{-13}$	$2.74x10^{-13}$	1.41E-10	$4.76x10^{-10}$	$5.09x10^{-10}$	$1.68x10^{-10}$
Ethyl Chloride	$1.29x10^{-9}$	$1.91x10^{-9}$	$2.04x10^{-9}$	1.01E-10	$3.39x10^{-10}$	$3.62x10^{-10}$	$1.20x10^{-10}$
Ethylene Dibromide	$2.70x10^{-13}$	$4.01x10^{-13}$	$4.29x10^{-13}$	2.88E-12	$9.68x10^{-12}$	$1.03x10^{-11}$	$3.42x10^{-12}$
Formaldehyde	$2.91x10^{-13}$	$4.32x10^{-13}$	$4.62x10^{-13}$	5.75E-10	$1.94x10^{-9}$	$2.07x10^{-9}$	$6.85x10^{-10}$
Isophorone	$7.89x10^{-13}$	$1.17x10^{-12}$	$1.25x10^{-12}$	1.39E-09	$4.68x10^{-9}$	$5.00x10^{-9}$	$1.65x10^{-9}$
Methyl Hydrazine	$1.43x10^{-13}$	$2.13x10^{-13}$	$2.28x10^{-13}$	4.07E-10	$1.37x10^{-9}$	$1.47x10^{-9}$	$4.85x10^{-10}$
Dichloromethane	$3.02x10^{-15}$	$4.68x10^{-15}$	$5.00x10^{-15}$	6.95E-10	$2.34x10^{-9}$	$2.50x10^{-9}$	$8.27x10^{-10}$
Benzo(a)pyrene	$1.87x10^{-13}$	$2.78x10^{-13}$	$2.97x10^{-13}$	1.46E-13	$4.91x10^{-13}$	$5.25x10^{-13}$	$1.73x10^{-13}$
Benzo(b)fluoranthene	$3.53x10^{-13}$	$5.25x10^{-13}$	$5.61x10^{-13}$	1.58E-13	$5.29x10^{-13}$	$5.66x10^{-13}$	$1.86x10^{-13}$
Chrysene	$1.52x10^{-14}$	$2.25x10^{-14}$	$2.41x10^{-14}$	4.28E-13	$1.44x10^{-12}$	$1.54x10^{-12}$	$5.06x10^{-13}$
Indeno(1,2,3-cd)pyrene	$1.72x10^{-13}$	$2.56x10^{-13}$	$2.74x10^{-13}$	7.77E-14	$2.61x10^{-13}$	$2.79x10^{-13}$	$9.19x10^{-14}$
2,3,7,8-TCDD	$1.29x10^{-9}$	$1.91x10^{-9}$	$2.04x10^{-9}$	1.84E-15	$5.64x10^{-15}$	$6.03x10^{-15}$	$2.03x10^{-15}$
benz(a)anthracene	$2.70x10^{-13}$	$4.01x10^{-13}$	$4.29x10^{-13}$	1.01E-13	$3.40x10^{-13}$	$3.64x10^{-13}$	$1.20x10^{-13}$
benzo(k)fluoranthene	$2.91x10^{-13}$	$4.32x10^{-13}$	$4.62x10^{-13}$	1.91E-13	$6.42x10^{-13}$	$6.87x10^{-13}$	$2.26x10^{-13}$
dibenzo(a,h)anthracene	$7.89x10^{-13}$	$1.17x10^{-12}$	$1.25x10^{-12}$	8.22E-15	$2.76x10^{-14}$	$2.95x10^{-14}$	$9.72x10^{-15}$
Quinoline	$1.43x10^{-13}$	$2.13x10^{-13}$	$2.28x10^{-13}$	9.35E-14	$3.14x10^{-13}$	$3.35x10^{-13}$	$1.11x10^{-13}$

Table 4.3-1 (Cont'd)
Calculated Inhalation Dose (mg/kg-d) for All Receptor Locations– Operating Scenario 3

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
COPC	Adult	Adult	Adult	Adult	Adult	Composite
Arsenic	5.20E-10	2.27E-10	1.70E-10	1.03E-10	8.38E-09	8.96E-09
Beryllium	7.26E-11	2.99E-11	2.27E-11	1.56E-11	2.61E-09	2.79E-09
Cadmium	4.98E-11	2.21E-11	1.65E-11	9.64E-12	5.04E-10	5.38E-10
Chromium (Total)	2.02E-09	8.49E-10	6.42E-10	4.21E-10	5.75E-08	6.15E-08
Acetaldehyde	3.73E-09	1.72E-09	1.27E-09	7.03E-10	1.01E-08	1.08E-08
Benzene	8.51E-09	3.93E-09	2.91E-09	1.60E-09	2.31E-08	2.47E-08
Benzyl chloride	4.58E-09	2.12E-09	1.56E-09	8.63E-10	1.24E-08	1.33E-08
di-(2-Ethylhexyl) phthalate	4.78E-10	2.21E-10	1.63E-10	9.00E-11	1.29E-09	1.38E-09
Bromoform	2.55E-10	1.18E-10	8.72E-11	4.81E-11	6.92E-10	7.40E-10
Chloroform	3.86E-10	1.78E-10	1.32E-10	7.27E-11	1.05E-09	1.12E-09
Ethyl Chloride	2.75E-10	1.27E-10	9.39E-11	5.18E-11	7.45E-10	7.97E-10
Ethylene Dibromide	7.85E-12	3.63E-12	2.68E-12	1.48E-12	2.13E-11	2.28E-11
Formaldehyde	1.57E-09	7.26E-10	5.37E-10	2.96E-10	4.26E-09	4.55E-09
Isophorone	3.79E-09	1.75E-09	1.30E-09	7.15E-10	1.03E-08	1.10E-08
Methyl Hydrazine	1.11E-09	5.14E-10	3.80E-10	2.10E-10	3.02E-09	3.22E-09
Dichloromethane	1.90E-09	8.77E-10	6.48E-10	3.57E-10	5.14E-09	5.50E-09
Benzo(a)pyrene	3.98E-13	1.80E-13	1.34E-13	7.46E-14	1.09E-12	1.16E-12
Benzo(b)fluoranthene	4.28E-13	1.94E-13	1.44E-13	8.03E-14	1.17E-12	1.25E-12
Chrysene	1.16E-12	5.26E-13	3.91E-13	2.18E-13	3.17E-12	3.39E-12
Indeno(1,2,3-cd)pyrene	2.11E-13	9.55E-14	7.11E-14	3.96E-14	5.76E-13	6.16E-13
TCDD, 2,3,7,8-	4.56E-15	1.99E-15	1.49E-15	9.07E-16	7.30E-14	7.81E-14
benz(a)anthracene	2.75E-13	1.25E-13	9.27E-14	5.17E-14	7.51E-13	8.03E-13
benzo(k)fluoranthene	5.20E-13	2.35E-13	1.75E-13	9.76E-14	1.42E-12	1.52E-12
dibenzo(a,h)anthracene	2.23E-14	1.01E-14	7.52E-15	4.19E-15	6.09E-14	6.52E-14
Quinoline	2.54E-13	1.15E-13	8.55E-14	4.76E-14	6.93E-13	7.41E-13

4.3.2 Ingestion

The dose from ingestion can include the pathways of soil (dust), vegetation, beef, and milk. The concentration of chemicals in each of these compartments is ultimately based on the estimated concentration of chemicals in emissions from the generating facility. The transfer of chemicals to soil, vegetation, beef, and milk predicts resulting contaminant concentrations in these compartments, and the ingestion dose to human receptors is calculated. The detailed equations are provided in Attachment A. A summary of the ingestion dose (mg/kg-d) for each receptor location is given in Tables 4.3-2 for the OS3. The total ingestion dose shown is the sum of the dose from each individual ingestion pathway (e.g., soil, dust, vegetation, beef, milk). The dose from individual ingestion pathway is provided in Attachment B.

Table 4.3-2
Calculated Total Ingestion Dose (mg/kg-d) for All Receptor Locations – Operating Scenario 3

(mg/kg-d)	Receptor 1	Receptor 2		Receptor 3	Receptor 4		Receptor 5
	Industrial Worker	Resident		School	Resident		School
COPC	Adult	Adult	Composite	Adult	Adult	Composite	Adult
Arsenic	3.11×10^{-18}	1.16×10^{-10}	1.26×10^{-10}	6.01×10^{-18}	7.09×10^{-10}	7.69×10^{-10}	7.34×10^{-18}
Beryllium	5.42×10^{-16}	1.11×10^{-11}	1.20×10^{-11}	1.05×10^{-15}	6.74×10^{-11}	7.29×10^{-11}	1.28×10^{-15}
Cadmium	1.98×10^{-17}	6.59×10^{-12}	7.19×10^{-12}	3.81×10^{-17}	4.01×10^{-11}	4.38×10^{-11}	4.66×10^{-17}
Chromium (Total)	4.00×10^{-18}	9.43×10^{-10}	1.09×10^{-9}	7.73×10^{-18}	5.74×10^{-9}	6.66×10^{-9}	9.44×10^{-18}
Acetaldehyde	2.45×10^{-17}	6.10×10^{-14}	6.65×10^{-14}	1.85×10^{-17}	8.82×10^{-14}	9.62×10^{-14}	1.93×10^{-17}
Benzene	1.94×10^{-17}	1.03×10^{-14}	1.25×10^{-14}	1.46×10^{-17}	1.46×10^{-14}	1.78×10^{-14}	1.53×10^{-17}
Benzyl chloride	2.35×10^{-15}	8.61×10^{-13}	1.06×10^{-12}	1.77×10^{-15}	1.24×10^{-12}	1.53×10^{-12}	1.85×10^{-15}
di-(2-Ethylhexyl) phthalate	1.70×10^{-10}	2.14×10^{-8}	2.48×10^{-8}	1.28×10^{-10}	2.66×10^{-8}	3.08×10^{-8}	1.34×10^{-10}
Bromoform	1.72×10^{-20}	5.74×10^{-18}	7.08×10^{-18}	1.30×10^{-20}	8.30×10^{-18}	1.02×10^{-17}	1.36×10^{-20}
Chloroform	8.14×10^{-19}	7.37×10^{-16}	8.05×10^{-16}	6.15×10^{-19}	1.05×10^{-15}	1.15×10^{-15}	6.41×10^{-19}
Ethyl Chloride	4.71×10^{-20}	2.55×10^{-16}	2.85×10^{-16}	3.56×10^{-20}	3.24×10^{-16}	3.63×10^{-16}	3.71×10^{-20}
Ethylene Dibromide	1.56×10^{-18}	8.13×10^{-16}	9.97×10^{-16}	1.18×10^{-18}	1.17×10^{-15}	1.44×10^{-15}	1.23×10^{-18}
Formaldehyde	6.03×10^{-15}	1.51×10^{-11}	1.64×10^{-11}	4.55×10^{-15}	2.18×10^{-11}	2.37×10^{-11}	4.75×10^{-15}
Isophorone	3.38×10^{-14}	2.77×10^{-11}	3.40×10^{-11}	2.55×10^{-14}	4.00×10^{-11}	4.92×10^{-11}	2.66×10^{-14}
Methyl Hydrazine	4.31×10^{-13}	1.23×10^{-8}	1.52×10^{-8}	3.26×10^{-13}	1.79×10^{-8}	2.19×10^{-8}	3.40×10^{-13}
Dichloromethane	2.02×10^{-18}	2.75×10^{-15}	3.37×10^{-15}	1.52×10^{-18}	3.96×10^{-15}	4.86×10^{-15}	1.59×10^{-18}
Benzo(a)pyrene	2.04×10^{-13}	1.75×10^{-12}	2.14×10^{-12}	3.94×10^{-13}	1.06×10^{-11}	1.30×10^{-11}	4.81×10^{-13}
Benzo(b)fluoranthene	8.30×10^{-14}	1.35×10^{-12}	1.60×10^{-12}	1.60×10^{-13}	8.18×10^{-12}	9.69×10^{-12}	1.96×10^{-13}
Chrysene	4.87×10^{-13}	5.13×10^{-12}	6.23×10^{-12}	9.42×10^{-13}	3.11×10^{-11}	3.77×10^{-11}	1.15×10^{-12}
Indeno(1,2,3-cd)pyrene	1.10×10^{-13}	7.12×10^{-13}	8.72×10^{-13}	2.12×10^{-13}	4.31×10^{-12}	5.28×10^{-12}	2.59×10^{-13}
TCDD, 2,3,7,8-	5.53×10^{-16}	9.23×10^{-15}	1.09×10^{-14}	1.07×10^{-15}	5.61×10^{-14}	6.60×10^{-14}	1.31×10^{-15}
benz(a)anthracene	1.28×10^{-13}	1.27×10^{-12}	1.55×10^{-12}	2.47×10^{-13}	7.69×10^{-12}	9.36×10^{-12}	3.02×10^{-13}
benzo(k)fluoranthene	2.70×10^{-13}	2.20×10^{-12}	2.69×10^{-12}	5.22×10^{-13}	1.33×10^{-11}	1.63×10^{-11}	6.37×10^{-13}
dibenzo(a,h)anthracene	1.16×10^{-14}	7.99×10^{-14}	9.77×10^{-14}	2.24×10^{-14}	4.84×10^{-13}	5.92×10^{-13}	2.74×10^{-14}
Quinoline	1.30×10^{-13}	6.49×10^{-12}	7.73×10^{-12}	2.52×10^{-13}	3.90×10^{-11}	4.64×10^{-11}	3.07×10^{-13}

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
COPC	Adult	Adult	Adult	Adult	Adult	Composite
Arsenic	1.23×10^{-19}	8.25×10^{-18}	4.29×10^{-18}	1.77×10^{-18}	4.92×10^{-9}	5.33×10^{-9}
Beryllium	3.33×10^{-17}	1.44×10^{-15}	7.47×10^{-16}	3.09×10^{-16}	4.67×10^{-10}	5.06×10^{-10}
Cadmium	1.21×10^{-18}	5.23×10^{-17}	2.72×10^{-17}	1.13×10^{-17}	2.87×10^{-10}	3.13×10^{-10}
Chromium (Total)	2.46×10^{-19}	1.06×10^{-17}	5.52×10^{-18}	2.28×10^{-18}	3.98×10^{-8}	4.62×10^{-8}
Acetaldehyde	4.83×10^{-19}	2.20×10^{-17}	1.30×10^{-17}	7.85×10^{-18}	5.29×10^{-13}	5.77×10^{-13}
Benzene	2.46×10^{-19}	1.75×10^{-17}	1.03×10^{-17}	6.21×10^{-18}	8.20×10^{-14}	1.01×10^{-13}
Benzyl chloride	2.98×10^{-17}	2.12×10^{-15}	1.24×10^{-15}	7.53×10^{-16}	7.39×10^{-12}	9.10×10^{-12}
di-(2-Ethylhexyl) phthalate	3.04×10^{-12}	1.53×10^{-10}	8.98×10^{-11}	5.44×10^{-11}	6.75×10^{-8}	7.95×10^{-8}
Bromoform	2.18×10^{-22}	1.55×10^{-20}	9.11×10^{-21}	5.52×10^{-21}	4.95×10^{-17}	6.10×10^{-17}
Chloroform	1.61×10^{-20}	7.34×10^{-19}	4.31×10^{-19}	2.61×10^{-19}	6.00×10^{-15}	6.56×10^{-15}
Ethyl Chloride	5.98×10^{-22}	4.24×10^{-20}	2.49×10^{-20}	1.51×10^{-20}	1.00×10^{-15}	1.16×10^{-15}
Ethylene Dibromide	1.99×10^{-20}	1.41×10^{-18}	8.29×10^{-19}	5.02×10^{-19}	7.03×10^{-15}	8.63×10^{-15}
Formaldehyde	1.19×10^{-16}	5.43×10^{-15}	3.19×10^{-15}	1.93×10^{-15}	1.31×10^{-10}	1.42×10^{-10}

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
COPC	Adult	Adult	Adult	Adult	Adult	Composite
Isophorone	$4.29x10^{-16}$	$3.05x10^{-14}$	$1.79x10^{-14}$	$1.08x10^{-14}$	$2.40x10^{-10}$	$2.95x10^{-10}$
Methyl Hydrazine	$5.47x10^{-15}$	$3.88x10^{-13}$	$2.28x10^{-13}$	$1.38x10^{-13}$	$1.07x10^{-7}$	$1.32x10^{-7}$
Dichloromethane	$2.56x10^{-20}$	$1.82x10^{-18}$	$1.07x10^{-18}$	$6.48x10^{-19}$	$2.34x10^{-14}$	$2.87x10^{-14}$
Benzo(a)pyrene	$1.13x10^{-14}$	$5.41x10^{-13}$	$2.81x10^{-13}$	$1.16x10^{-13}$	$8.14x10^{-11}$	$1.00x10^{-10}$
Benzo(b)fluoranthene	$3.65x10^{-15}$	$2.20x10^{-13}$	$1.14x10^{-13}$	$4.73x10^{-14}$	$5.94x10^{-11}$	$7.07x10^{-11}$
Chrysene	$2.52x10^{-14}$	$1.29x10^{-12}$	$6.73x10^{-13}$	$2.78x10^{-13}$	$2.36x10^{-10}$	$2.89x10^{-10}$
Indeno(1,2,3-cd)pyrene	$6.12x10^{-15}$	$2.91x10^{-13}$	$1.51x10^{-13}$	$6.25x10^{-14}$	$3.29x10^{-11}$	$4.07x10^{-11}$
TCDD, 2,3,7,8-	$2.34x10^{-17}$	$1.47x10^{-15}$	$7.64x10^{-16}$	$3.16x10^{-16}$	$4.01x10^{-13}$	$4.73x10^{-13}$
benz(a)anthracene	$6.85x10^{-15}$	$3.39x10^{-13}$	$1.76x10^{-13}$	$7.30x10^{-14}$	$5.88x10^{-11}$	$7.22x10^{-11}$
benzo(k)fluoranthene	$1.51x10^{-14}$	$7.16x10^{-13}$	$3.72x10^{-13}$	$1.54x10^{-13}$	$1.02x10^{-10}$	$1.26x10^{-10}$
dibenzo(a,h)anthracene	$6.48x10^{-16}$	$3.08x10^{-14}$	$1.60x10^{-14}$	$6.62x10^{-15}$	$3.70x10^{-12}$	$4.56x10^{-12}$
Quinoline	$7.24x10^{-15}$	$3.45x10^{-13}$	$1.80x10^{-13}$	$7.43x10^{-14}$	$3.46x10^{-10}$	$4.12x10^{-10}$

4.3.3 Exposure to Infants

The exposure to an infant at the maximum POI location is calculated through the maternal milk pathway. The equations for infant exposure are provided in Attachment A. The infant is assumed to have a body weight of 10 kg and ingest 800 g breast milk/day. The concentration of benzo(a)pyrene in breast milk is calculated from the predicted daily ingestion of the chemical by the mother (Maximum POI adult). Benzo(a)pyrene is assessed, as it has a tendency to bio-accumulate and is therefore likely to transfer from the mother to nursing child. The dose of benzo(a)pyrene to the infant at the Maximum POI is calculated to be 9.2×10^{-8} (µg/kg-d) for operation at the 100 % C.F under the upper-bound operating scenario (OS3). This dose is insignificant and does not represent a level of concern. No further discussion on the maternal milk pathway is given in this assessment.

5.0 HAZARD ASSESSMENT

The hazard assessment involves the identification of the potentially toxic effects of chemicals, and the determination of the appropriate exposure limits for the various chemicals. The exposure limit or toxicity reference value (TRV) is defined as the amount of chemical exposure that can occur without any adverse health effects (for threshold or non-cancer causing chemicals), or that is associated with an acceptable level of risk (non-threshold or cancer-causing chemicals).

5.1 TOXICITY REFERENCE VALUES

For this assessment, the TRVs were obtained from reputable regulatory agencies such as Health Canada and the U.S. EPA. The majority of the TRVs used in this assessment are obtained from the U.S. EPA IRIS database. The quantity and quality of available information from human and animal studies varies widely from one contaminant to another, and it is sometimes only possible to present qualitative information on the toxicity of many of the chemicals to humans. In the hazard assessment, data would generally be obtained on:

- **Slope Factor (SF)** - (for carcinogens) comprises a plausible upper bound estimate of the probability of a response per unit intake of a contaminant over a lifetime. For carcinogens, no threshold is assumed to exist (i.e., every dose presents some risk); or
- **Reference Dose (RfD)** - (for non-carcinogens) comprises an estimate of the daily exposure level for a chemical for the entire population, including sensitive receptors, that is not anticipated to present an appreciable risk of an adverse effect during a portion of a lifetime.

Table 5.1-1 provides a summary of the exposure limits used in the assessment of risks associated with the coal-fired operation of the Brandon G.S. As seen from the table, all the identified COPC are non-threshold or cancer-causing chemicals since they all have slope factors associated with them.

For chemicals such as combustion gases, toxicity is dependent on the chemical concentration in the air rather than the total internal dose received by multiple exposure pathways. In general, the adverse effects are associated with irritation of the tissues of the eyes, and upper and lower respiratory systems. For combustion gases, TRVs are represented by air quality guidelines/objectives and are used as exposure limits to assess potential health effects. A summary of the exposure limits for combustion gases are presented in Table 5.1-2.

Table 5.1-1
Toxicity Reference Values for Human Receptors

COPC	SF oral		RfD oral		SF inhalation		RfD inhalation	
	1/(mg/kg-d)		(mg/kg-d)		1/(mg/kg-d)		(mg/kg-d)	
Arsenic	1.5	i	$3.00x10^{-4}$	i	$1.51x10^{1}$	i	n/a	
Beryllium	n/a		$2.00x10^{-3}$	i	8.40	i	$5.71x10^{-6}$	i
Cadmium	n/a		$5.00x10^{-4}$	i	6.30	i	n/a	
Chromium (assume total)	n/a		n/a		$4.20x10^{1}$	i	n/a	
Acetaldehyde	n/a		n/a		$7.70x10^{-3}$	i	$2.57x10^{-3}$	i
Benzene	$5.50x10^{-2}$	i	$4.00x10^{-3}$	i	$2.70x10^{-2}$	i	$8.60x10^{-3}$	i
Benzyl chloride	$1.70x10^{-1}$	i	n/a		$1.70x10^{-1}$	r		
Bis(2-ethylhexyl)phthalate (DEHP)	$1.40x10^{-2}$	i	$2.00x10^{-2}$	i	$1.40x10^{-2}$	r	$2.00x10^{-2}$	r
Bromoform	$7.90x10^{-3}$	i	$2.00x10^{-2}$	i	$3.85x10^{-3}$	i	$2.00x10^{-2}$	r
Chloroform	n/a		$1.00x10^{-2}$	i	$8.05x10^{-2}$	i	$1.40x10^{-2}$	n
Ethyl chloride	$2.90x10^{-3}$	n	$4.00x10^{-1}$	n	$2.90x10^{-3}$	r	2.86	i
Ethylene dibromide	2.00	i	$9.00x10^{-3}$	i	2.00	i	$2.60x10^{-3}$	i
Isophorone	$9.50x10^{-4}$	i	$2.00x10^{-1}$	i	$9.50x10^{-4}$	r	$2.00x10^{-1}$	r
Methyl hydrazine	1.10	h	n/a		1.10	r	n/a	
Methylene chloride (Dichloromethane)	$7.50x10^{-3}$	i	$6.00x10^{-2}$	i	$1.65x10^{-3}$	i	$8.57x10^{-1}$	h
Benzo(a)anthracene	$7.30x10^{-1}$	n	n/a		$7.30x10^{-1}$	r	n/a	
Benzo(a)pyrene	7.30	i	n/a		7.30	r	n/a	
Benzo(b)fluoranthene	$7.30x10^{-1}$	n	n/a		$7.30x10^{-1}$	r	n/a	
Chrysene	$7.30x10^{-3}$	n	n/a		$7.30x10^{-3}$	r	n/a	
Indeno(1,2,3-cd)pyrene	$7.30x10^{-1}$	n	n/a		$7.30x10^{-1}$	r	n/a	
Benzo(k)fluoranthene	0.073	n	n/a		0.073	r	n/a	
Dibenzo(a,h)anthracene	7.30	n	n/a		7.30	r	n/a	
Quinoline	3	i	n/a		3	r	n/a	
Total Dioxins (using 2,3,7,8-TCDD)	$1.50x10^{5}$	h	n/a		$1.50x10^{5}$	h	n/a	

Note: n/a- data not available
i- IRIS
n- NCEA
h- HEAST
r- route to route extrapolation

Table 5.1-2
Exposure Limits/Air Quality Guidelines for Combustion Gases

Combustion Gas		Concentration ($\mu g/m^3$)	Jurisdiction
CO	1-hr	30,000	WHO (2000)
	8-hr	10,000	WHO (2000)
	Annual	2,800	Calculated from Manitoba Guideline [a]
NO_2	1-hr	400	CCME / Manitoba (2005)
	1-hr	200	WHO (2005)
	24-hr	200	Manitoba (2005)
	Annual	40	WHO (2005)
SO_2	1-hr	350	WHO (2005)
	24-hr	125*	WHO (2000, 2005)
	24-hr	20	WHO (2005)
	Annual	n/a	WHO (2005)

Note: n/a – not available

a) The 1-hr guideline was divided by a factor of 12.5 to obtain an annual average value (U.S. EPA 1992)

* Interim guideline

Manitoba ambient air quality criteria are available for the combustion gases (see Air Quality Assessment Report - Appendix K); however, the World Health Organization (WHO) health-based guidelines for combustion gases were used to evaluate the potential for adverse health effects in this assessment.

Currently, there is no WHO 24-hr guideline for NO_2; therefore, the Manitoba air quality criterion was used for the exposure limit. As shown in Table 5.1-2, the 24-hour guideline for SO_2 has been updated in 2005 – the new guideline is about six times lower than the guideline set in 2000 and is based on epidemiological studies. The WHO realizes that this new guideline may be quite difficult to achieve in the short term, and has suggested a stepped approach using the interim value of 125 $\mu g/m^3$ shown in Table 5.1-2. It should be noted that these recommended guideline values for sulfur dioxide are not linked with guidelines for particles. WHO further noted in their *Air Quality Guidelines Global Update* (2005) that an annual guideline for SO_2 is not necessary since compliance with the 24-hour level will assure low levels for the annual average.

6.0 HUMAN HEALTH RISK ASSESSMENT

The final step in the risk assessment process is the characterization of health risks or impacts. In this step the predicted exposures are compared to the exposure limits for a given chemical in order to determine the risks associated with the various chemicals of concern.

For the current assessment, potential adverse effects and risks are calculated using deterministic (point estimate) risk estimates or concentration ratios. Concentration ratio values for short-term or long-term exposure to combustion gases are calculated by dividing the predicted concentration at the location of the maximum point of impingement by the appropriate reference concentration as shown in the following equation:

$$Concentration\,Ratio = \frac{Predicted\ Air\,Concentration\left(\mu g / m^3\right)}{Reference\,Concentration\left(\mu g / m^3\right)} \qquad (6\text{-}1)$$

A concentration ratio value below 1 implies that the health effects associated with the combustion gas are not significant.

In the case of the cancer-causing chemicals, the risk level is calculated by multiplying the predicted exposure (ingestion) by the cancer slope factor (SF), as shown in the following equation:

$$Risk = Predicted\ Exposure\left(\frac{mg}{kg\ \ d}\right) \times SF\left(\frac{mg}{kg\ \ d}\right)^{-1} \qquad (6\text{-}2)$$

For this assessment, an assumed lifetime risk level of 1×10^{-5} is used as the reference risk level where health impacts are considered to be insignificant. Health Canada considers that a reference lifetime risk of 1×10^{-5} represents an essentially negligible risk. The selection of an appropriate risk level can be weighed, as is the case with the U.S. EPA, against the size of populations exposed and what is reasonably achievable. Discussions around the U.S. EPA's Clean Air Act proposal for hazardous air pollutants include a risk range of 10^{-6} to 10^{-4} with a risk of 10^{-4} deemed to be safe (U.S. EPA 1990). Thus, a risk level of 1×10^{-5} represents a conservative value.

6.1 POTENTIAL SHORT-TERM AND LONG-TERM HEALTH RISKS ARISING FROM COMBUSTION GASES

As discussed previously, the health effects associated with combustion gases occur at the site of contact with the sensitive tissues of the eyes and respiratory system. Therefore, combustion

gases are assessed using concentration ratio values. Potential health effects from short-term exposures are determined by using 1-hour, 8-hour or 24-hour ground level air concentrations at the maximum point of impingement. Similarly, chronic health risks associated with the combustion gases are estimated using annual average air concentrations predicted at the maximum point of impingement for residential receptors.

Concentration ratio values less than 1 indicate that the predicted air concentrations are less than the reference concentrations, and as such it is not expected that any adverse health effects would occur. A concentration ratio value above 1 would indicate that the reference concentration is exceeded and that there is a possibility that an adverse health effect, namely irritation, may occur.

6.1.1 Potential Short-Term Human Health Risks Associated with Exposure to the Combustion Gases

The results of the assessment for short-term health effects associated with estimated exposure to the combustion gases produced by the coal-fired operation of the Brandon G.S. are presented in Table 6.1-1. The assessment was based on predicted incremental concentrations at the maximum point of impingement for current coal source that use different burner row combinations for OS1 and OS2 (See Table 1.1 for description of different operating scenarios). The predicted concentrations were also obtained from the maximum theoretical operating condition –100 % C.F which represents the maximum concentration that any receptor would be exposed to in the vicinity of the Brandon G.S. The use of an alternative coal source in the future only affects the SO_2 emissions. The CO and NO_2 concentrations for OS2 and OS3 are identical. Therefore, Table 6.1-1 also provides the values for SO_2 for the future scenario (OS3).

Table 6.1-1
Potential Short-Term Concentration Ratios for The Combustion Gases at Maximum Point of Impingement (Without Background)

Emission		Regulatory Jurisdiction	Maximum Concentration from Brandon G.S. ($\mu g/m^3$)	Regulatory Objectives ($\mu g/m^3$)	Concentration Ratio
OS1 - Current Coal Source with Burner Row Combination B,C,D					
CO	1-hr	Manitoba	16.1	35,000	0.0005
		WHO		30,000	0.0005
	8-hr	Manitoba	3.5	15,000	0.0002
		WHO		10,000	0.0004
NO_2*	1-hr	Manitoba	91	400	0.23
		WHO		200	0.46

Table 6.1-1 (Cont'd)
Potential Short-Term Concentration Ratios for The Combustion Gases at Maximum Point of Impingement (Without Background)

Emission		Regulatory Jurisdiction	Maximum Concentration from Brandon G.S. ($\mu g/m^3$)	Regulatory Objectives ($\mu g/m^3$)	Concentration Ratio
NO_2*	24-hr	Manitoba	7.9	200	0.04
		WHO		200	0.04
NO_2**	1-hr	Manitoba	243	400	0.61
		WHO		200	1.22
	24-hr	Manitoba	17.8	200	0.09
		WHO		200	0.09
SO_2	1-hr	Manitoba	190	900	0.21
		WHO		350	0.54
	24-hr	Manitoba	13.9	300	0.05
		WHO		125 (Interim)	0.11
		WHO		20	0.70
OS2 - Current Coal Source with Burner Row Combination A,B,C					
CO	1-hr	Manitoba	19.1	35,000	0.0005
		WHO		30,000	0.0006
	8-hr	Manitoba	4.2	15,000	0.0003
		WHO		10,000	0.0004
NO_2*	1-hr	Manitoba	119	400	0.30
		WHO		200	0.60
	24-hr	Manitoba	10.4	200	0.05
		WHO		200	0.05
NO_2**	1-hr	Manitoba	322	400	0.81
		WHO		200	1.61
	24-hr	Manitoba	23.6	200	0.12
		WHO		200	0.12
SO_2	1-hr	Manitoba	200.0	900	0.22
		WHO		350	0.57
	24-hr	Manitoba	14.6	300	0.05
		WHO		125 (Interim)	0.12
		WHO		20	0.73
OS3 - FutureCoal Source with Burner Row Combination A,B,C					
SO_2 – OS3 Scenario	1-hr	Manitoba	265.5	900	0.30
		WHO		350	0.76
	24-hr	Manitoba	19.5	300	0.07
		WHO		125 (Interim)	0.16
		WHO		20	0.98

Note: shaded value indicate concentration exceeding the critical value of 1
* Janssen method (see Appendix K). **assuming 100 % NO conversion to NO_2.

As seen in Table 6.1-1, all short-term concentration ratios in the current scenario are well below one, except for 1-hour NO_2 emissions when compared agains WHO guidelines. The concentration ratios are 1.2 for 1-hour emissions of NO_2 (assuming that 100 % of the NO emitted is immediately converted to NO_2) for OS1 and 1.6 for OS2. However, a more realistic estimate of NO_2 concentrations is based on the empirically-derived Janssen equation for the rate of conversion of NO to NO_2 in a power plant plume. The Janssen equation estimate produces a concentration ratio of 0.5 for OS1 and 0.6 for OS2.

As discussed in Section 5.1, the 2005 WHO air quality guideline updated the 24-hour SO_2 concentration to 20 $\mu g/m^3$ – about 15 times lower than the Manitoba air quality criteria (300 $\mu g/m^3$) (2005). The predicted maximum incremental SO_2 emissions from Brandon G.S. (at the maximum POI) for all scenarios are below this updated guideline value (Table 6.1-1).

Concentration ratios calculated using total maximum concentrations (i.e., including ambient background concentrations) of the combustion gases are provided in Tables 6.1-2a for scenarios OS1, OS2, and OS3. The background concentrations are based on results obtained from an air quality monitoring station in Winnipeg (Station 9118 at Scotia & Jefferson for CO) and Brandon (for NO_2). The average of the maxmum background concentrations from 2000-2004 for CO is 4517.6 and 2461 $\mu g/m^3$ for 1-hr and 8-hr period, respectively. The mean background concentration for NO_2 from 2000-2004 is 102 (see Air Quality Assessment Report – Appendix K) and 54 $\mu g/m^3$ for 1-hr and 24-hr period, respectively.

Most of the concentration ratios are below 1, as shown in the Table 6.1-2a, except for some concentration ratios of 1-hr NO_2 which exceeded the acceptable level of 1. In the case of 100 % conversion of NO to NO_2, the addition of background NO_2 measured at Brandon results in a ratio of 2.1 for OS2, but a ratio of 1.7 for the preferred mode of operation, OS1, using row burner combination B, C, D. However, since the assumption of 100 % conversion of NO to NO_2 is extremely conservative because such a rapid rate of conversion is not in fact possible, the exceedance of the 1.0 value in the former case is considered hypothetical. A more realistic estimate is considered to be that which is based on the Janssen equation, and the latter method indicates that the maximum predicted 1-hour average NO_2 concentrations plus background levels would come to a total NO_2 concentration of 221 $\mu g/m^3$ which is slightly above the WHO Health Guideline (200 $\mu g/m^3$), but below the Manitoba Maximum Acceptable Objective of 400 $\mu g/m^3$. It should be noted that the background concentration used in the calculation is the average of the maximum measured annual NO_2 concentration from 2000-2004. Therefore, the emissions from Unit #5 at the Brandon G.S. are unlikely to cause adverse health effects; however, it is advisable to continue operating practices that minimize NO_2 emissions.

Table 6.1-2a

Potential Short-Term Concentration Ratios for the Combustion Gases at the Maximum Point of Impingement (Including Background)

Emission		Regulatory Jurisdiction	Maximum Concentration from Brandon G.S. ($\mu g/m^3$)	Regulatory Objectives ($\mu g/m^3$)	Concentration Ratio
OS1 - Current Coal Source with Burner Row Combination B,C,D					
CO	1-hr	Manitoba	4534	35,000	0.13
		WHO		30,000	0.15
	8-hr	Manitoba	2465	15,000	0.16
		WHO		10,000	0.25
NO_2*	1-hr	Manitoba	193	400	0.48
		WHO		200	0.97
	24-hr	Manitoba	63	200	0.32
		WHO		200	0.32
NO_2**	1-hr	Manitoba	345	400	0.86
		WHO		200	1.73
	24-hr	Manitoba	62	200	0.31
		WHO		200	0.31
SO_2	1-hr	Manitoba	190	900	0.21
		WHO		350	0.54
	24-hr	Manitoba	13.9	300	0.05
		WHO		125 (Interim)	0.11
		WHO		20	0.70
OS2 - Current Coal Source with Burner Row Combination A,B,C					
CO	1-hr	Manitoba	4537	35,000	0.13
		WHO		30,000	0.15
	8-hr	Manitoba	2466	15,000	0.16
		WHO		10,000	0.25
NO_2*	1-hr	Manitoba	221	400	0.55
		WHO		200	1.11
	24-hr	Manitoba	64.4	200	0.32
		WHO		200	0.32
NO_2**	1-hr	Manitoba	424	400	1.06
		WHO		200	2.12
	24-hr	Manitoba	78	200	0.39
		WHO		200	0.39
SO_2	1-hr	Manitoba	200	900	0.22
		WHO		350	0.57
	24-hr	Manitoba	14.6	300	0.05
		WHO		125 (Interim)	0.12
		WHO		20	0.73

TABLE 6.1-2a (Con'td)
Potential Short-Term Concentration Ratios for the Combustion Gases at the Maximum Point of Impingement (Including Background)

OS3 - Future Coal Source with Burner Row Combination A,B,C					
SO_2	1-hr	Manitoba	265.5	900	0.30
		WHO		350	0.76
	24-hr	Manitoba	19.5	300	0.07
		WHO		125 (Interim)	0.16
		WHO		20	0.98

Note: - shaded value indicate concentration exceeding the critical value of 1
- average CO background concentration in Winnipeg over 5 years (1999 – 2003)
- 1-hour average background NO_2: maximum observed concentration over 5 years (2000-2004) minus maximum predicted 1-hour average concentration for emissions from Unit # 5 (see Appendix K) assuming 100 % NO to NO_2 conversion
- 24-hour average background NO_2: average of the maximum measured background NO_2 concentration in Brandon over 5 years (2000-2004)

* Janssen method (see Appendix K)
**assuming 100 % NO conversion to NO_2

It should be noted that there is currently no SO_2 monitoring in Brandon, Selkirk, or in Winnipeg. SO_2 monitoring was discontinued in Brandon in 1989, but readings prior to that were too low to be registered by the instrument (0.0 ppm). Therefore, it can reasonably be assumed that the emissions from the Brandon Generating Station are the largest source of SO_2 emission in the area, and that the SO_2 concentrations calculated for Brandon G.S. based on dispersion modelling can be directly compared with the air quality guidelines by WHO, ambient air quality objectives for Manitoba and/or the National Ambient Air Quality Objectives (NAAQO), without consideration of additional background SO_2 levels. Table 6.1-2 indicates that the predicted 24-hour average SO_2 concentrations are below the stringent WHO guideline for the current scenarios (OS1 and OS2) and the future scenario that uses an alternative coal source with higher sulphur content (OS3).

In addition, an assessment of the combined impacts from the operation of Unit #5 in conjunction with the two natural gas-fired combustion turbines (CTs – Units #6 and #7) were evaluated. Although Manitoba Hydro operates the CTs under a separate permit and the operation of the CTs is not part of this licence review for Unit #5, the potential for cumulative short-term impacts on air quality was considered an appropriate issue for consideration in this review. SO_2 was not evaluated since insignificant amounts are released from the two CTs. The combined concentrations of CO and NO_x shown in Table 6.1-2b were based on the maximum estimated off-site air concentrations at ground-level corresponding to the CTs operating at full capacity. However, a 25 % capacity scenario was used to predict maximum concentrations for CO since emissions of CO are significantly higher at lower turbine loads (see Section 5.6.2 in Air Quality

Assessment Report – Appendix K). It should be noted that the concentrations of CO and NO_2 are the same for Scenarios OS2 and OS3; therefore, only OS2 is presented in Table 6.1-2b.

Table 6.1-2b
Potential Short-Term Concentration Ratios for The Combustion Gases at the Maximum Point of Impingement (Including Background) – Combined Unit #5 And CT Operations

Emission		Regulatory Jurisdiction	Maximum Combined Concentration (Unit #5 and CTs) from Brandon G.S. ($\mu g/m^3$)	Regulatory Objectives ($\mu g/m^3$)	Concentration Ratio
OS2-Current Coal Source with Burner Row Combination A,B,C + CT Operations					
CO	1-hr	Manitoba	4710	35,000	0.13
		WHO		30,000	0.16
	8-hr	Manitoba	2525	15,000	0.17
		WHO		10,000	0.25
NO2*	1-hr	Manitoba	221	400	0.55
		WHO		200	1.11
	24-hr	Manitoba	64	200	0.32
		WHO		200	0.32
NO2**	1-hr	Manitoba	462	400	1.15
		WHO		200	2.31
	24-hr	Manitoba	78	200	0.39
		WHO		200	0.39

Note: - shaded value indicate concentration exceeding the critical value of 1
- average CO background concentration in Winnipeg over 5 years (1999 – 2003)
- 1-hour average background NO_2: maximum observed concentration over 5 years (2000-2004) minus maximum predicted 1-hour average concentration for emissions from Unit # 5 (see Air Quality Assessment Report – Appendix K) assuming 100 % NO to NO_2 conversion
- 24-hour average background NO_2: average of the maximum measured background NO_2 concentration in Brandon over 5 years (2000-2004)

* Janssen method. No direct NO_2 emission data were available for CTs. With use of the Janssen method, it was assumed that 20 % of the direct NO_x emissions are NO_2, with the remaining 80 % as NO (see Air Quality Assessment Report – Appendix K, Section 5.6)

**assuming 100 % NO conversion to NO_2

As shown in the Table 6.1-2b, most of the concentration ratios are below 1, except for 1-hr NO_2 which exceeded the acceptable level of 1. In the case of 100 % conversion of NO to NO_2, the addition of background NO_2 measured at Brandon results in a ratio of 2.3 for OS2. As discussed previously, the assumption of 100 % conversion of NO to NO_2 is extremely conservative because such a rapid rate of conversion is not in fact possible. A more realistic estimate is considered to be that which is based on the Janssen equation, and the latter method indicates that

the maximum predicted 1-hour average NO_2 concentrations plus background levels would result in a concentration of 221 µg/m^3 which is slightly above the WHO Health Guideline (200 µg/m^3), but below the Manitoba Maximum Acceptable Objective of 400 µg/m^3. It should be noted that the background concentration used in the calculation is the maximum measured 1-hour average NO_2 concentration at Assiniboine Community College in Brandon during 2000-2004, minus the maximum predicted 1-hour average NO_2 concentration for emissions from Unit #5 at the Assiniboine Community College station, assuming 100 % conversion of NO to NO_2 (see Air Quality Assessment Report – Appendix K). This evaluation indicates that the emissions from the combined operation of Unit #5 and the two CTs at the Brandon G.S. are unlikely to cause adverse health effects; however, it is advisable to continue operating practices that minimize NO_2 emissions.

In summary, these results indicate that no measurable short-term adverse health outcomes would be expected in sensitive individuals from the current operations of Unit #5 at the Brandon G.S. In the OS3 scenario, the SO_2 concentrations will not increase by more than 33 %. Although daily mortality in 12 Canadian Cities (Burnett *et al*. 2004) has been significantly associated with average daily SO_2 below those that have been predicted for the Brandon G.S., there remains *"considerable uncertainty as to whether sulfur dioxide is the pollutant responsible for the observed adverse effects or, rather, a surrogate for ultra-fine particles or some other correlated substance"* (WHO 2005).

6.1.2 Potential Long-Term Health Risks Associated with Exposure to Combustion Gases at the Maximum Point of Impingement for the Residential Locations

The results of the assessment for chronic health effects associated with estimated exposure to the combustion gases produced by Unit #5 of the Brandon G.S. in comparison to background are presented in Table 6.1-3. The results shown in Table 6.1-3 are based on OS3 that would result in the highest emission rates. The future operating scenario for SO_2 emissions assumes use of a coal with no more than 33 % higher sulphur content than for current operations.

Table 6.1-3
Potential Chronic Concentration Ratios for the Combustion Gases at The Maximum Point of Impingement –OS3 (Including Background)

	WHO Annual Guideline[a] (µg/m^3)	Maximum Predicted Annual Average Concentration (µg/m^3)	Average Background Concentration (µg/m^3)	Concentration Ratio [(Max Brandon + Background)/Criteria]
CO	2,800	0.05	442[b]	0.16
NO_2*	40	0.87	11[c]	0.30
SO_2	n/a	0.71	na	na

	WHO Annual Guideline[a] ($\mu g/m^3$)	**Maximum Predicted Annual Average Concentration ($\mu g/m^3$)**	**Average Background Concentration ($\mu g/m^3$)**	**Concentration Ratio [(Max Brandon + Background)/Criteria]**
SO_2 (Current Scenario-OS2)	n/a	0.54	na	na

Note: n/a – not available , na – not applicable

* assuming 100 % NO conversion to NO_2

a) see Table 5.1-2 for detail

b) average background concentration in Winnipeg over 5 years (1999 – 2003)

c) average background concentration in Brandon over 5 years (2000 – 2004)

As seen from the above tables, long-term concentration ratio values for CO and NO_2, including background levels measured in Winnipeg/Brandon, are below the acceptable value of one. These long-term concentrations are based on the maximum predicted annual average concentrations (at the maximum point of impingement) with the plant operating at the full load condition. The highest concentration ratio (0.30) shown in Table 6.1-3 is obtained for NO_2. It should be noted that the reference concentration values used to generate these ratios were derived by regulatory agencies to protect the most sensitive individuals within a population. The WHO in their recent guideline for SO_2 (2005) indicated that an annual value for SO_2 is not necessary since compliance with the 24-hour guideline will assume low annual values. The predicted 24-hour SO_2 concentrations are below the most stringent SO_2 guidelines which implies that there should be no long-term adverse effects from exposure to SO_2. Therefore, no measurable adverse health effects would occur from long-term exposure to the combustion gases emitted from Unit #5 of the Brandon G.S.

6.2 PARTICULATE MATTER

Particulate matter describes all airborne solid and liquid particles of microscopic size, with the exception of pure water. The suspended portion of particulate matter generally consists of particles less than 40 to 50 microns (μm) in diameter. These particles include a broad range of chemical species, such as elemental and organic carbon compounds, sulphates, nitrates and trace metals.

Many studies over the past few years have indicated that particulate matter (PM) in the air is associated with various adverse health effects in people who already have compromised respiratory systems such as asthma, chronic pneumonia and cardiovascular problems. The World Health Organization (WHO) in 2004 provided a summary of the effects relating to particulate matter. The WHO Working Group stated that in the absence of clearly defined thresholds in exposure-response relationships for both long-term and short-term health effects, and the fact that these exposure-response relationships had been established at currently observed particulate matter exposure ranges, it can be concluded that adverse health effects from

particulate matter exposure are occurring at the levels of exposure currently experienced in urban areas in Europe. Since the conclusions were based on multi-city studies in the U.S., Canada and Europe, it suggests that health impacts also occur at particulate matter levels commonly observed in Canada. The following table provides a summary of the health effects associated with particulate matter.

Table 6.2-1
Important Health Effects Associated with Exposure to Particulate Matter

Effects Related to Short-term Exposure	Effects Related to Long-term Exposure
Lung inflammatory reactions	Increase in lower respiratory symptoms
Respiratory symptoms	Reduction in lung function in children
Adverse effects on the cardiovascular system	Increase in chronic obstructive pulmonary disease
Increase in medication usage	Reduction in lung function in adults
Increase in hospital admissions	Reduction in life expectancy, owing mainly to cardiopulmonary mortality and probably to lung cancer
Increase in mortality	

Source: WHO (2004)

The United States Environmental Protection Agency in 2004 also completed a comprehensive review of epidemiological studies on the human health effects associated with particulate matter inhalation. The document, *Air Quality Criteria for Particulate Matter* (U.S. EPA 2004), provides a synthesis of the available information summarizing epidemiological and toxicological studies prior to 2004 and combines it with the previous reviews conducted by the U.S. EPA (1996). Some of the relevant conclusions include:

- A large majority of relevant mortality studies show a statistically positive correlation with concentration of PM_{10}. Based on several multi-city studies in the U.S., Canada and Europe, statistically significant associations have been developed for cardiovascular and respiratory mortality with effect estimates ranging from 1.0 to 3.5 % (per 50 $\mu g/m^3$ PM_{10} increment);
- A growing body of epidemiologic evidence that confirms short - and long - term exposure to $PM_{2.5}$ is associated with various mortality or morbidity endpoints effects. Cardiovascular and respiratory mortality risks show positive correlations; however, the respiratory risks are not statistically significant. For multi-city studies, there is a 1 to 3.5 % increased risk of mortality per 25 $\mu g/m^3$ $PM_{2.5}$ increment;
- There are positive statistical associations with hospitalization for cardiovascular and respiratory diseases with exposure to both PM_{10} and $PM_{2.5}$; and

- Evidence suggests that not only $PM_{2.5}$ but coarse thoracic particles (e.g., $PM_{10-2.5}$) may contribute in exacerbating various respiratory conditions (e.g., asthma). Furthermore, there is new evidence suggesting a likely increase in the occurrence of chronic bronchitis associated with particulate matter exposure, especially long-term particulate matter exposure.

A summary of some recent key studies is provided below.

Laden *et al*. (2000) provide supplementary evidence of an observed association between fine particulate matter from different sources and daily mortality based on heart disease, pneumonia and cardio-pulmonary disease (COPD) in six U.S. cities. Their analysis focused on the elemental composition of $PM_{2.5}$ to identify distinct source-related fractions. They determined that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ from mobile sources accounted for a 3.4 % increase in daily mortality. The fine particulate from coal combustion sources was associated with a 1.1 % increase in mortality for the same 10 $\mu g/m^3$ increase in $PM_{2.5}$. These results suggest that pollution emissions from mobile sources such as gasoline and diesel powered vehicles may have a larger impact on sensitive populations than do emissions from coal combustion sources.

Laden *et al.* (2006), extended the previous study conducted in 2000. The results of the analysis indicate that the city-specific adjusted all-cause mortality rate ratios decreased with decreasing $PM_{2.5}$ concentration. Furthermore, cardiovascular mortality, as well as lung cancer mortality, was positively associated with average $PM_{2.5}$ concentration. Respiratory mortality was positively associated with average $PM_{2.5}$ concentration; however, the association was not statistically significant. The study found that for each 10 $\mu g/m^3$ of $PM_{2.5}$ reduced there can be a decrease of 0.73 in the relative risk level specifically due to deaths associated with respiratory and cardiovascular health, but not lung cancer, as the latency period for lung cancer mortality is greater. The authors concluded that the reduced mortality risk for the study population was associated with reduction in $PM_{2.5}$ concentrations.

In a study of daily mortality rates in twenty U.S. cities, Samet *et al*. (2000) determined that a 10 $\mu g/m^3$ increase in PM_{10} resulted in an incremental increase in the mortality rate of 0.5 % for all causes of death. The authors also estimated that the relative rate of death from cardiovascular and respiratory causes was about 0.68 % per 10 $\mu g/m^3$ increase in PM_{10}. A slightly higher value of 0.7 % increase in daily mortality per 10 $\mu g/m^3$ increase has been suggested by Levy *et al.* (2000a). Levy *et al.* (1999, 2002a) estimated the impacts of power plants in the Boston and Chicago areas. The impacts of the known emissions of fine particles and secondary-pollutant-forming gases from the plants were estimated by dispersion modelling. In the Chicago area, the authors indicated a population-weighted (i.e., exposure-weighted) annual average impact from nine plants to be 0.3 $\mu g/m^3$ (total of primary and secondary PM). Given that the size of the population exposed at this level was 33 million, the incremental exposure estimated due to the

plants was 320 premature deaths per year. Levy *et al.* (2002b) applied a similar analysis to exposure of the demographically-characterized sub-populations of Washington, DC in relation to coal-fired power plants. They concluded that application of "Best Available Control Technology" to the five plants analysed would result in 240 fewer premature deaths per year in the greater Washington, DC area.

Some studies have also focused on morbidity issues relating to exposure to PM. For example, Zanobetti *et al*. (2000a) examined the effect of prior admission for respiratory disease on whether or not a patient turned up at an emergency hospital facility during a high pollution event. They determined that the risk associated with PM_{10} for hospitalization of elderly patients (>65 years old) with cardiovascular disease was approximately twice as high for patients with concurrent respiratory infection. The evidence for pre-existing heart disease modifies the risk of chronic obstructive pulmonary disease (COPD) admissions on high pollution days. The study also found that evidence of a previous heart failure increased the risk for admission on high PM days. Hospital visits also increased for elderly patients with acute respiratory infections when the PM concentrations were high. However, while the study found that greater PM concentrations exacerbated existing respiratory conditions, it did not find that high PM concentrations were the cause of these conditions.

Zanobetti *et al.* (2000b) also performed a multi-city analysis of the relationship between levels of PM_{10} and hospital admissions for heart and lung disease. They found that for each increase of 10 $\mu g/m^3$ of PM_{10}, COPD hospitalization rates increased by 2.5 %, pneumonia rates increased by 1.95 % and cardiovascular disease rates increased by 1.27 %. The authors were not able to determine whether the public health impacts were dominated more by a few high pollution days or whether such impacts persist at concentrations generally observed in urban areas on most days.

Riediker *et al.* (2004) examined the cardiovascular effects of particulate matter exposure in cars by healthy young men. The study measured the exposure associated with 10 male highway patrol troopers working a 3 pm to midnight shift across 4 consecutive workdays. The results of the study indicate that in-vehicle $PM_{2.5}$ (per 10 $\mu g/m^3$) was associated with pathophysiologic changes that are associated with inflammation, coagulation and cardiac rhythm.

Recent studies have demonstrated that the human health effects associated with fine particulate matter may have been previously underestimated. One such study was conducted by Jerrett *et al.* (2005), where a subpopulation of approximately 23,000 southern Californians was followed between 1982 to 2000. Of the subpopulation, a total of 5,856 deaths were accounted for during this period. In addition, 44 individual confounders including lifestyle, diet, demographics, occupation and education, were taken into account. The results indicate that the relative risk calculated for $PM_{2.5}$ exposure was approximately 3 times greater than previous models reported

in literature. Furthermore, the study found a stronger association between air pollution and ischemic heart disease than with more general measures of cardiopulmonary deaths or all-cause mortality. In terms of the relative risk levels, the study indicates that all-cause mortality had a relative risk of 1.17 for an increase of 10 $\mu g/m^3$ $PM_{2.5}$. The relative risk for mortality resulting from ischemic heart disease and lung cancer deaths were elevated, in the range of 1.24-1.6 for an increase 10 $\mu g/m^3$ $PM_{2.5}$, depending on the model used. For cancers, the relative risks for lung cancers were higher than for digestive cancers or other cancers as expected. However, attempts to replicate these results in other cities have not yet been successful.

Dominici *et al.* (2006) examined the risks of cardiovascular and respiratory effects based upon hospital admissions associated with short-term exposure to $PM_{2.5}$. The results of the study indicate a short-term increase in hospital admission rates associated with $PM_{2.5}$ for all of the health outcomes (i.e., cerebrovascular disease, peripheral vascular disease, ischemic heart disease, heart rhythm, heart failure, respiratory tract infection, and chronic obstructive pulmonary disease) except injuries. The largest association was for heart failure, which had a 1.28 % increase in risk per 10 $\mu g/m^3$ increase in same-day $PM_{2.5}$ concentration.

For Unit #5 emissions, the maximum point of impingement for 24-hour average concentrations occurs at the facility property line near the northwest corner of the plant boundary. A secondary point of elevated concentration occurs within the City of Brandon. For the fugitive dust emissions from the coal storage area, the maximum 24-hour average and annual average concentrations occur at the facility property line on the south side of the plant, while those for the ash storage area occur at the property line along the north boundary of the plant, near the Assiniboine River.

The current regulatory limits for particulate matter are as follows:

- Canada-Wide Standard (CWS) target level for 24-hour concentrations of $PM_{2.5}$ of 30 $\mu g/m^3$ by the year 2010[2]. The Canadian Environmental Protection Act/Federal Provincial Advisory Committee Working Group on Air Quality Objectives and Guidelines (CEPA/FPAC WGAQOG) recommends a 24-hour average $PM_{2.5}$ health reference level of 15 $\mu g/m^3$ below which statistically significant health effects cannot be determined.
- Manitoba guideline for PM_{10} of 50 $\mu g/m^3$ (24-hour average) and $PM_{2.5}$ of 30 $\mu g/m^3$ (24-hour average).
- Manitoba 24-hour maximum acceptable objective for suspended particulate matter (SPM) of 120 $\mu g/m^3$ (24-hour average) and 70 $\mu g/m^3$ (annual geometric mean).

The CWS of 30 $\mu g/m^3$ for $PM_{2.5}$, which was formally adopted in June 2000, is considered to be a reasonably achievable target level for particulate matter to be achieved nationally by 2010.

[2] Attainment of the CWS is based on the 98th percentile ambient measurement annually, averaged over three consecutive years.

Manitoba has adopted this value in 2005. Maximum $PM_{2.5}$ concentration measured at the Assiniboine Community College in Brandon over the period 2001-2004 was 26 $\mu g/m^3$, while the 98th percentile values in any given year range from 15 $\mu g/m^3$ to 18 $\mu g/m^3$. The maximum predicted concentration of $PM_{2.5}$ is below the Manitoba guideline of 30 $\mu g/m^3$. The CWS parameter (98th percentile averaged over 3 consecutive years) for $PM_{2.5}$ was 17 $\mu g/m^3$ in 2003 and 16 $\mu g/m^3$ in 2004. Therefore, the measured $PM_{2.5}$ concentrations in Brandon are well below the CWS level of 30 $\mu g/m^3$.

At the maximum point of impingement, the highest predicted 24-hour average $PM_{2.5}$ concentration due to emissions from the Brandon G.S. Unit #5 stack is 0.5 $\mu g/m^3$. Therefore, the Unit #5 stack emissions contribute a relatively minor amount of material to the $PM_{2.5}$ levels in the Brandon area. The maximum predicted concentrations for fugitive dust from coal and ash storage are conservatively estimated at 15 $\mu g/m^3$ and 1 $\mu g/m^3$, respectively. At the point of maximum predicted concentration, the 98th percentile 24-hour average concentration for fugitive coal dust is only 1.7 $\mu g/m^3$, while that for ash is much less than 1 $\mu g/m^3$. Although the maximum point of impingement for the Unit #5 stack emissions and fugitive coal/ash emissions do not occur at the same location, the CWS in the area would not be exceeded even if they did coincide and were added to the 98th percentile levels measured at the Assiniboine Community College in Brandon.

The maximum measured 24-hour average PM_{10} concentrations at the Assiniboine Community College in Brandon over the period 1998-2004 consistently exceeded the Manitoba guideline value of 50 $\mu g/m^3$. Maximum 24-hour average PM_{10} concentrations ranged from 127 $\mu g/m^3$ in 1998 to 229 $\mu g/m^3$ in 2002. Annual average PM_{10} concentrations over the same period ranged from 20 $\mu g/m^3$ to 23 $\mu g/m^3$. The primary cause for the high levels of SPM and PM_{10} in the Brandon area is believed to be fugitive dust emissions from agricultural activity, as well as possibly seasonal burning of agricultural waste and stubble in fields.

For PM_{10} emissions from the Unit #5 combustion stack, the maximum predicted 24-hour incremental concentration is 0.8 $\mu g/m^3$. This value is well below the measurement accuracy of a PM_{10} monitor. Therefore, the contributions of particulate matter emissions from the Unit #5 combustion stack do not significantly contribute to the exceedances of the PM_{10} guideline of 50 $\mu g/m^3$ recorded at the PM_{10} monitor in Brandon.

The maximum predicted PM_{10} concentrations for fugitive dust emissions are conservatively estimated at 26 $\mu g/m^3$ for coal dust and 7 $\mu g/m^3$ for ash from the ash storage area. Ninety-nine percent (99 %) of the time, the maximum contribution of fugitive coal dust to ambient PM_{10} levels anywhere in the area would be less than 15 $\mu g/m^3$. Therefore, fugitive emissions from the Brandon G.S. alone would not be sufficient to cause the high PM_{10} concentrations measured in Brandon. Moreover, as indicated in Table 6.2-2, the maximum predicted PM_{10} concentrations

are negligible at the Riverview Elementary School (i.e., in the closest residential area west of the Brandon G.S. and near the air quality monitoring station at the Assiniboine Community College), as well as at the nearest residence east of the plant.

Table 6.2-2
Maximum Off-Site Air Concentrations (μg/m^3) of Particulate Matter Due to Fugitive Dust Releases

PM Mass Fraction	Coal Storage and Handling		Ash Lagoon	
	Maximum 24-hour	Annual Average	Maximum 24-hour	Annual Average
	Maximum Point of Impingement			
SPM	105	0.5	8	1.3
PM_{10}	26	0.2	7	0.3
$PM_{2.5}$	15	0.1	1	0.1
	Receptor R2 – Riverview Elementary			
SPM	0.09	0.0033	0.15	0.0077
PM_{10}	0.07	0.0025	0.14	0.0074
$PM_{2.5}$	0.009	0.00039	0.014	0.00074
	Receptor R3 – nearest residence east of plant			
SPM	0.52	0.0079	0.29	0.017
PM_{10}	0.40	0.0054	0.28	0.016
$PM_{2.5}$	0.047	0.00083	0.025	0.015

Background SPM concentrations in the Brandon area are not measured. However, since the maximum measured 24-hour average ambient PM_{10} levels in Brandon have been reported to be as high as 229 μg/m^3, it is reasonable to assume that maximum 24-hour background SPM concentrations greater than 200 μg/m^3 are not uncommon. Therefore, it is likely that the Maximum Acceptable ambient air quality objectives for SPM noted above are exceeded every year in the Brandon area. However, these objectives relate to nuisance impacts from plume visibility and soiling. No significant health impacts are expected to occur at the Maximum Acceptable level of 120 μg/m^3 (Government of Canada 1991). Increased sensitivity of patients with asthma and bronchitis may occur only when SPM levels exceed the Maximum Tolerable level of 400 μg/m^3 (24-hour average). There is no evidence to suggest that the Maximum Tolerable levels are exceeded in the Brandon area.

The maximum 24-hour average incremental SPM concentrations of 0.8 μg/m^3 due to Unit #5 combustion stack emissions is predicted to occur near the northwest corner of the Brandon G.S. property line**.** Maximum predicted SPM concentrations for fugitive dust from the coal and ash storage areas are 105 μg/m^3 and 8 μg/m^3, respectively. As discussed in the air quality assessment report (Appendix K), the estimate of fugitive coal dust contributions to ambient SPM concentrations is considered to be conservative, in that the estimated SPM emission rates from

the coal storage area likely overstate actual emission rates. Even so, conservatively predicted concentrations would be less than 80 $\mu g/m^3$ on all but one day per year. Ninety-nine percent (99 %) of the time (i.e., 361 of 365 days per year), the maximum predicted contribution to ambient SPM levels due to fugitive coal dust would be less than 27 $\mu g/m^3$. Furthermore, as indicated in Table 6.2-1, the predicted SPM concentrations due to fugitive dust from coal and ash storage are negligible at the nearest residential areas east and west of the plant. Therefore, fugitive dust emissions from the Brandon G.S. alone would not be sufficient to account for the high PM_{10} (and by extension, SPM) concentrations that have been measured in Brandon.

Overall, the results of the dispersion modelling analysis indicate that maximum predicted impacts of $PM_{2.5}$ from coal-fired operations at the Brandon G.S. are negligible (i.e., below the measurement capability of $PM_{2.5}$ monitors). While the fugitive coal dust emissions potentially could exceed the PM_{10} health reference level of 25 $\mu g/m^3$ on perhaps one day per year, background PM_{10} levels in the area due to other sources are much more significant contributors to observed PM_{10} levels. Similarly, conservatively estimated SPM emissions from coal-fired operations at the Brandon G.S. on their own would not exceed levels that might exacerbate asthma or bronchitis in sensitive individuals. SPM levels in the area around Brandon are already elevated due to other emission sources, and the emission of fugitive coal dust from the Brandon G.S. represents only one of many contributing sources. In the residential areas of Brandon as well as at the nearest residential neighbours east of the plant, the contribution of fugitive dust to ambient levels of PM_{10} and SPM is below the detection capability of PM_{10} and SPM monitors.

6.3 POTENTIAL LONG-TERM HUMAN HEALTH RISKS ASSOCIATED WITH EXPOSURE TO CHEMICALS OTHER THAN COMBUSTION GASES

Long-term risks for the COPC are calculated by multiplying the predicted exposure by the carcinogenic slope factor as described in Section 5.0. In this assessment, inhalation risks are calculated separately to ingestion risks and finally the total risk to the human receptor is obtained by summing these two risks together. As discussed previously a cancer risk level of one-in-one hundred thousand (1×10^{-5}) was considered acceptable and at a level where health risks are insignificant. Tables 6.3-1 presents the potential chronic health risks for the future scenario (OS3). Background exposures are not considered in these calculations.

As seen from Table 6.3-1, the cancer risks for long-term exposure to metals, VOCs and PAHs are several orders of magnitude lower than the Health Canada acceptable risk level of one-in-one hundred thousand. Additionally, it has been assumed that a residence is located at the maximum POI, which is not the case. Furthermore, these risk levels represent the exposure of people in the community to the upper-bound emissions scenario from the operation at the Brandon G.S. Therefore, it can be concluded that no measurable adverse health effects would be expected for

people in the community from the current coal source and long-term adverse health effects even if the coal source were changed at the Brandon G.S.

Additionally, these risk levels are well below those associated with exposures to background concentrations of the same chemicals (in the order of 10^{-4} to 10^{-6}). Thus, no measurable increase over background in long-term adverse health effects are predicted from exposure of people in the community to potential emissions from the current and future operation at the Brandon G.S.

Table 6.3-1
Total Risk Levels for Predicted Exposures to Chemicals of Concern From the Brandon G.S. Stack & Fugitive Emissions – OS3

(mg/kg-d)	Receptor 1	Receptor 2		Receptor 3	Receptor 4		Receptor 5
	Industrial Worker	Resident		School	Resident		School
COPC	Adult	Adult	Composite	Adult	Adult	Composite	Adult
Arsenic	5.20×10^{-9}	8.21×10^{-9}	8.79×10^{-9}	3.17×10^{-9}	1.08×10^{-8}	1.15×10^{-8}	3.49×10^{-9}
Beryllium	3.89×10^{-10}	6.37×10^{-10}	6.81×10^{-10}	2.78×10^{-10}	7.54×10^{-10}	8.06×10^{-10}	2.79×10^{-10}
Cadmium	2.10×10^{-10}	3.19×10^{-10}	3.41×10^{-10}	1.22×10^{-10}	3.88×10^{-10}	4.15×10^{-10}	1.38×10^{-10}
Chromium (Total)	5.48×10^{-8}	8.79×10^{-8}	9.40×10^{-8}	3.70×10^{-8}	1.05×10^{-7}	1.12×10^{-7}	3.84×10^{-8}
Acetaldehyde	1.95×10^{-11}	2.89×10^{-11}	3.09×10^{-11}	1.05×10^{-11}	3.54×10^{-11}	3.78×10^{-11}	1.25×10^{-11}
Benzene	1.56×10^{-10}	2.31×10^{-10}	2.47×10^{-10}	8.41×10^{-11}	2.83×10^{-10}	3.03×10^{-10}	1.00×10^{-10}
Benzyl chloride	5.28×10^{-10}	7.84×10^{-10}	8.38×10^{-10}	2.85×10^{-10}	9.60×10^{-10}	1.03×10^{-9}	3.39×10^{-10}
di-(2-Ethylhexyl) phthalate	6.91×10^{-12}	3.07×10^{-10}	3.54×10^{-10}	4.24×10^{-12}	3.81×10^{-10}	4.40×10^{-10}	4.79×10^{-12}
Bromoform	6.66×10^{-13}	9.89×10^{-13}	1.06×10^{-12}	3.60×10^{-13}	1.21×10^{-12}	1.29×10^{-12}	4.28×10^{-13}
Chloroform	2.11×10^{-11}	3.13×10^{-11}	3.35×10^{-11}	1.14×10^{-11}	3.83×10^{-11}	4.09×10^{-11}	1.35×10^{-11}
Ethyl Chloride	5.40×10^{-13}	8.02×10^{-13}	8.58×10^{-13}	2.92×10^{-13}	9.82×10^{-13}	1.05×10^{-12}	3.47×10^{-13}
Ethylene Dibromide	1.06×10^{-11}	1.58×10^{-11}	1.69×10^{-11}	5.75×10^{-12}	1.94×10^{-11}	2.07×10^{-11}	6.85×10^{-12}
Formaldehyde	4.90×10^{-11}	7.27×10^{-11}	7.78×10^{-11}	2.65×10^{-11}	8.90×10^{-11}	9.52×10^{-11}	3.15×10^{-11}
Isophorone	2.44×10^{-12}	3.66×10^{-12}	3.91×10^{-12}	1.32×10^{-12}	4.48×10^{-12}	4.80×10^{-12}	1.57×10^{-12}
Methyl Hydrazine	8.30×10^{-10}	1.48×10^{-8}	1.80×10^{-8}	4.49×10^{-10}	2.12×10^{-8}	2.57×10^{-8}	5.34×10^{-10}
Dichloromethane	2.12×10^{-12}	3.15×10^{-12}	3.37×10^{-12}	1.15×10^{-12}	3.86×10^{-12}	4.13×10^{-12}	1.36×10^{-12}
Benzo(a)pyrene	3.46×10^{-12}	1.57×10^{-11}	1.88×10^{-11}	3.95×10^{-12}	8.11×10^{-11}	9.84×10^{-11}	4.78×10^{-12}
Benzo(b)fluoranthene	2.73×10^{-13}	1.30×10^{-12}	1.50×10^{-12}	2.32×10^{-13}	6.36×10^{-12}	7.49×10^{-12}	2.79×10^{-13}
Chrysene	9.32×10^{-15}	4.60×10^{-14}	5.46×10^{-14}	1.00×10^{-14}	2.38×10^{-13}	2.87×10^{-13}	1.21×10^{-14}
Indeno(1,2,3-cd)pyrene	1.85×10^{-13}	6.75×10^{-13}	8.03×10^{-13}	2.11×10^{-13}	3.34×10^{-12}	4.06×10^{-12}	2.56×10^{-13}
TCDD, 2,3,7,8-	5.37×10^{-10}	2.09×10^{-9}	2.38×10^{-9}	4.37×10^{-10}	9.26×10^{-9}	1.08×10^{-8}	5.00×10^{-10}
benz(a)anthracene	2.30×10^{-13}	1.13×10^{-12}	1.35×10^{-12}	2.54×10^{-13}	5.86×10^{-12}	7.10×10^{-12}	3.08×10^{-13}
benzo(k)fluoranthene	4.55×10^{-14}	1.99×10^{-13}	2.37×10^{-13}	5.21×10^{-14}	1.02×10^{-12}	1.24×10^{-12}	6.30×10^{-14}
dibenzo(a,h)anthracene	1.95×10^{-13}	7.48×10^{-13}	8.89×10^{-13}	2.24×10^{-13}	3.73×10^{-12}	4.53×10^{-12}	2.71×10^{-13}
Quinoline	9.08×10^{-13}	2.02×10^{-11}	2.40×10^{-11}	1.04×10^{-12}	1.18×10^{-10}	1.40×10^{-10}	1.25×10^{-12}

Table 6.3-1 (Con'td)
Total Risk Levels for Predicted Exposures to Chemicals of Concern From the Brandon G.S. Stack & Fugitive Emissions – OS3

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Industrial Worker	Resident	School	Resident	School	
COPC	Adult	Adult	Adult	Adult	Adult	Composite
Arsenic	$7.85x10^{-9}$	$3.42x10^{-9}$	$2.57x10^{-9}$	$1.56x10^{-9}$	$1.34x10^{-7}$	$1.43x10^{-7}$
Beryllium	$6.10x10^{-10}$	$2.51x10^{-10}$	$1.90x10^{-10}$	$1.31x10^{-10}$	$2.19x10^{-8}$	$2.34x10^{-8}$
Cadmium	$3.14x10^{-10}$	$1.39x10^{-10}$	$1.04x10^{-10}$	$6.07x10^{-11}$	$3.17x10^{-9}$	$3.39x10^{-9}$
Chromium (Total)	$8.47x10^{-8}$	$3.57x10^{-8}$	$2.70x10^{-8}$	$1.77x10^{-8}$	$2.41x10^{-6}$	$2.58x10^{-6}$
Acetaldehyde	$2.87x10^{-11}$	$1.33x10^{-11}$	$9.81x10^{-12}$	$5.41x10^{-12}$	$7.79x10^{-11}$	$8.33x10^{-11}$
Benzene	$2.30x10^{-10}$	$1.06x10^{-10}$	$7.85x10^{-11}$	$4.33x10^{-11}$	$6.23x10^{-10}$	$6.66x10^{-10}$
Benzyl chloride	$7.79x10^{-10}$	$3.60x10^{-10}$	$2.66x10^{-10}$	$1.47x10^{-10}$	$2.11x10^{-9}$	$2.26x10^{-9}$
di-(2-Ethylhexyl) phthalate	$6.73x10^{-12}$	$5.23x10^{-12}$	$3.54x10^{-12}$	$2.02x10^{-12}$	$9.63x10^{-10}$	$1.13x10^{-9}$
Bromoform	$9.82x10^{-13}$	$4.54x10^{-13}$	$3.36x10^{-13}$	$1.85x10^{-13}$	$2.66x10^{-12}$	$2.85x10^{-12}$
Chloroform	$3.11x10^{-11}$	$1.44x10^{-11}$	$1.06x10^{-11}$	$5.85x10^{-12}$	$8.42x10^{-11}$	$9.01x10^{-11}$
Ethyl Chloride	$7.97x10^{-13}$	$3.68x10^{-13}$	$2.72x10^{-13}$	$1.50x10^{-13}$	$2.16x10^{-12}$	$2.31x10^{-12}$
Ethylene Dibromide	$1.57x10^{-11}$	$7.26x10^{-12}$	$5.37x10^{-12}$	$2.96x10^{-12}$	$4.26x10^{-11}$	$4.55x10^{-11}$
Formaldehyde	$7.22x10^{-11}$	$3.34x10^{-11}$	$2.47x10^{-11}$	$1.36x10^{-11}$	$1.96x10^{-10}$	$2.09x10^{-10}$
Isophorone	$3.61x10^{-12}$	$1.67x10^{-12}$	$1.23x10^{-12}$	$6.79x10^{-13}$	$1.00x10^{-11}$	$1.07x10^{-11}$
Methyl Hydrazine	$1.22x10^{-9}$	$5.66x10^{-10}$	$4.18x10^{-10}$	$2.31x10^{-10}$	$1.21x10^{-7}$	$1.48x10^{-7}$
Dichloromethane	$3.13x10^{-12}$	$1.45x10^{-12}$	$1.07x10^{-12}$	$5.90x10^{-13}$	$8.49x10^{-12}$	$9.08x10^{-12}$
Benzo(a)pyrene	$2.99x10^{-12}$	$5.26x10^{-12}$	$3.03x10^{-12}$	$1.39x10^{-12}$	$6.02x10^{-10}$	$7.39x10^{-10}$
Benzo(b)fluoranthene	$3.15x10^{-13}$	$3.02x10^{-13}$	$1.89x10^{-13}$	$9.32x10^{-14}$	$4.42x10^{-11}$	$5.25x10^{-11}$
Chrysene	$8.67x10^{-15}$	$1.33x10^{-14}$	$7.77x10^{-15}$	$3.62x10^{-15}$	$1.75x10^{-12}$	$2.13x10^{-12}$
Indeno(1,2,3-cd)pyrene	$1.59x10^{-13}$	$2.82x10^{-13}$	$1.62x10^{-13}$	$7.45x10^{-14}$	$2.45x10^{-11}$	$3.02x10^{-11}$
TCDD, 2,3,7,8-	$6.88x10^{-10}$	$5.19x10^{-10}$	$3.39x10^{-10}$	$1.83x10^{-10}$	$7.11x10^{-8}$	$8.27x10^{-8}$
benz(a)anthracene	$2.06x10^{-13}$	$3.39x10^{-13}$	$1.96x10^{-13}$	$9.10x10^{-14}$	$4.35x10^{-11}$	$5.33x10^{-11}$
benzo(k)fluoranthene	$3.91x10^{-14}$	$6.94x10^{-14}$	$4.00x10^{-14}$	$1.84x10^{-14}$	$7.56x10^{-12}$	$9.30x10^{-12}$
dibenzo(a,h)anthracene	$1.68x10^{-13}$	$2.98x10^{-13}$	$1.72x10^{-13}$	$7.89x10^{-14}$	$2.74x10^{-11}$	$3.38x10^{-11}$
Quinoline	$7.83x10^{-13}$	$1.38x10^{-12}$	$7.95x10^{-13}$	$3.66x10^{-13}$	$1.04x10^{-9}$	$1.24x10^{-9}$

6.4 RADIONUCLIDES

Coal and coal ash contain trace quantities of naturally occurring radionuclides such as uranium and thorium, and the corresponding members of their respective decay series (a total of 14 radionuclides in the U-238 series and 10 in the Th-232 series). During combustion, some of these radionuclides are released into the atmosphere with flue gases, and the remainder are retained in fly and bottom ash. Also, some of the radioactivity in coal ash can be re-suspended into the air by wind and mechanical manipulation of stockpiles.

Atmospheric dispersion modelling is used to estimate the annual average airborne concentrations of uranium and thorium at selected locations around the facility that are attributable to releases from the Brandon station (see Appendix K) the highest incremental annual average

concentrations of uranium and thorium in air (from both stack and fugitive emission) for the future scenario (OS3) are predicted to be 5.1×10^{-5} μg/m^3 and 5.4×10^{-6} μg/m^3, respectively.

Natural background uranium concentrations in air have been measured at only a few locations in Canada and in the United States. Tracy and Prantl (1985) reported the ambient concentration of uranium in air in southern Ontario at 1×10^{-4} μg/m^3, and Ahier and Tracy (1993) reported the mean concentration in Oshawa, Ontario at approximately 6×10^{-4} μg/m^3, ranging from 2×10^{-4} to 12×10^{-4} μg/m^3 (see Table 6.4-1). Airborne concentrations of uranium were reported at selected centres in the United States by the U.S. National Council on Radiation Protection and Measurements (NCRP 1975) and are also shown in Table 6.4-1. Table 6.4-2 provides typical natural background concentrations of thorium in air. Background thorium concentrations can range from 1.3 to 4.0×10^{-4} μg/m^3.

Table 6.4-1
Natural Background Levels of Uranium in Air

Mean ($\times 10^{-4}$ μg/m^3)	Range ($\times 10^{-4}$ μg/m^3)	Location	Reference
1	n/a	Southern Ontario	Tracy and Prantl (1985)
6	2 to 12	Oshawa, Ontario	Ahier and Tracy (1993)
1.8	1 to 2.6	Argonne National Laboratory, USA	NCRP (1975)
3.8		New York City	NCRP (1975)
6		New York State	NCRP (1975)
0.4		global	UNSCEAR (1993)

Table 6.4-2
Natural Background Levels of Thorium in Air

Mean ($\times 10^{-4}$ μg/m^3)	Range ($\times 10^{-4}$ μg/m^3)	Location	Reference
2.7	1.3 to 4.0	Argonne National Laboratory, USA	NCRP (1975)
2.5		global	UNSCEAR (1993)

Based on the modelling predictions, the incremental airborne concentrations of uranium and thorium that are attributable to releases from the coal-fired operations of the Brandon G.S. are expected to be a small fraction of the natural background concentrations of uranium and thorium in air. In addition, the incremental concentrations are also a small fraction of the variability in the natural background concentrations. Therefore, the impacts from inhalation and deposition to soil and vegetation of releases from naturally occurring radioactivity in the coal and coal ash are expected to be insignificant, and indistinguishable from the impacts of naturally occurring uranium and thorium in the air.

6.5 UNCERTAINTY ANALYSIS

Many areas of uncertainty attend a risk assessment. This is due to the fact that assumptions have to be made throughout the assessment either due to data gaps, environmental fate complexities or in the generalization of receptor characteristics. To be able to place a level of confidence in the results, an accounting of the uncertainty, the magnitude and type of which are important in determining the significance of the results, must be completed. In recognition of these uncertainties, generally conservative assumptions are used throughout the assessment to ensure that the potential for an adverse effect would not be underestimated. Several of the major assumptions are outlined below. Overall, it is considered that the assumptions used in this assessment tend to overestimate the risks and that efforts made to reduce the uncertainty would result in reduced risks and increase the confidence in the conclusions that there is no potential for adverse health effects from emissions of the coal-fired operations of the Brandon Generating Station.

6.5.1 Uncertainties in the Assessment

Selection of the assessed chemicals are based on emissions data as well as emission factors obtained from the U.S. EPA's AP-42 for coal-fired operations. The emission factors used in this assessment are also based on these factors. Emission factors are average values, which introduce uncertainty to the estimate of exposure.

The predicted air concentrations are based on maximum sustained generation rates, and in practice, the station is not operated at the maximum sustained generation rate for a long period of time, and most certainly not 100 % of the time, as assumed for this assessment. Therefore, the actual impacts of combustion gas and particulate emissions will be much lower than indicated by the predicted concentrations used for this upper-bound scenario.

The receptors and their characteristics are selected in order to overestimate potential exposures. For example, it is assumed that an adult residential receptor is assumed to live at their house 24 hours/day, 365 days/year for 70 years with no time away from the site for vacations, working off-site, etc. Additionally, the inhalation and ingestion rates are also chosen to be conservative.

For the pathways modelling, where data are lacking (e.g., physical characteristics of the soil, soil density etc.) upper-bound values are generally assumed. For example, soil bulk density can range from 1.2 to 1.5 g/cm^3 (Perry and Chilton 1973) and a value of 1.5 g/cm^3 is assumed for the calculations in this assessment. A higher soil bulk density predicts a higher soil concentration; therefore, the upper-bound value for soil bulk density is 1.5 g/cm^3. Similarly, the most conservative values for chemical parameters such as soil-to-plant transfer factors are used. Since these transfer factors may vary by several orders of magnitude, this introduces a considerable

level of conservatism. The uncertainty in these assumptions could be reduced by using site-specific data gathered in Manitoba. Given that the health impacts are predicted to be insignificant, these changes would not result in any changes to the overall conclusion of the assessment.

The assessment also considers a hypothetical receptor living at the maximum point of impingement when in fact no such individual lives at this location.

It was assumed that residential receptors in the area would consume 5 % of milk and meat from study area. Some studies indicate that this value could be as high as 44 % (U.S. DOE 2001). A sensitivity analysis was conducted to explore the effect of increasing the milk and meat intake from study area. The analysis showed that ingestion dose did not demonstrate significant differences in the total risk level. Thus, the results of the assessment would be unchanged. The sensitivity analysis for a residential receptor at the maximum POI location is provided in Attachment D.

Another area of uncertainty is the use of a single value for toxicity. The slope factors are selected to be very protective. The factors used in the subject assessment represent risks from upper bound (95th percentile) dose-response estimates. No adjustments are made for bioavailability of chemicals in soils and air, which can result in either an over estimation of exposure and thus leads to uncertainty in the risk assessment. The toxicity assays used to generate these slope factors are not generally conducted for humans, thus toxicological data from the most sensitive laboratory species, generally rats or mice are extrapolated to humans and used in the assessment. Based on the current state of toxicology, these are the best values available and tend to overestimate risks.

Thus, it is currently not feasible to develop approaches to evaluate the validity of the above assumptions on the overall assessment. As improvements occur in the toxicological/human health research and assessments, the uncertainties may be reduced. However, given that the risk levels associated with the metals, VOCs and PAHs are so small, it is not anticipated that these improvements would change the overall conclusion of the assessment.

7.0 ECOLOGICAL RISK ASSESSMENT

A screening level ecological risk assessment was carried out to assess the need for a more detailed assessment for plant and animal species. The screening level assessment confirmed that the emissions from the generating station are not expected to be significant enough to warrant further investigation of the impacts on ecological receptors in the area.

The Canadian Council of Ministers of the Environment (CCME) has provided general guidance concerning their views on what constitutes an ecological risk assessment, ERA (CCME 1996, 1997). The framework provided is similar to that proposed by Environment Canada (Environment Canada 1997). The CCME proposes three levels of investigation:

1) Screening level assessment (SLA): essentially a qualitative assessment of potential risks to important ecological receptors.
2) Preliminary quantitative risk assessment (PQRA): focuses on filling gaps identified at the screening level.
3) Detailed quantitative risk assessment (DQRA): includes more detailed data and modelling.

Each level of the assessment includes the following elements:

- **Receptor characterization** – At this phase of the assessment the potential receptors are identified and the pathways of exposure defined.
- **Exposure assessment** – The purpose of this stage is to quantify the contact between the receptor and the contaminant of concern.
- **Hazard assessment** – This phase of the ERA examines the potential effects of a contaminant to a receptor using toxicity reference values (TRVs).
- **Risk characterization** – The risk characterization stage combines the information collected in the exposure assessment and the hazard assessment and the potential for adverse ecological effects is estimated.

Assessing the potential risks of unacceptable mortality, decreased growth, or reproductive impairment for populations exposed to chemicals requires the accurate integration of estimates or measures of exposure and dose with TRV concentrations known to produce toxic responses. Such assessments can be performed effectively and economically using a sequential approach to ecological risk assessment. This iterative analysis is consistent with the approach outlined by the Canadian Council of Ministers of the Environment (CCME 1996).

The rigour of the risk assessment adopted for a particular situation should be commensurate with the degree and extent of potential harm and may progress to a more stringent level (i.e., from

SLA to PQRA or from PQRA to DQRA) depending on the findings at each level. Each level in this tiered approach has the same structure and builds upon the data, information, knowledge and decisions generated from the preceding level. Thus, each level is progressively more rigorous and complex.

In many cases, site conditions dictate selection of a *screening level assessment or SLA* (which is referred to as Tier 1) to identify the likelihood of ecological risks posed by the presence of identified chemicals. Qualitative and/or comparative methods are used in the assessment. Screening indices are often used in an initial screening assessment to facilitate comparisons. The screening index value is defined as the ratio of the modelled exposure or dose to laboratory toxicity data. Screening assessments, often completed at a species level, involve assumptions that bias estimates of exposure and toxicity towards predicting an ecological impact (i.e., overestimating exposure or dose and underestimating the concentration required to produce a toxic response). If, under these conservative assumptions, a site passes the screening assessment, then reasonable conclusions of minimal ecological risk are supported. The propagation of uncertainty throughout the analysis provides a quantitative measure of the reliability of the assessment and may be useful in identifying major sources of uncertainty for which further refinement of the assessment may be warranted, for example in a site-specific detailed assessment.

A *preliminary quantitative risk assessment, or PQRA,* (which is referred to as Tier 2) follows an SLA and can be used to further evaluate combinations of species and chemicals that fail the screening level assessment. In the preliminary quantitative assessment, the pessimistic assumptions of the screening level calculations are examined more closely to produce more realistic values of exposure, dose and TRV. A combination of field measurements, laboratory experiments, data analysis and ecological modelling might prove useful for increasing the accuracy and precision in estimating exposure and response for the species and chemicals of concern. The results of the preliminary quantitative assessment provide more realistic comparisons of expected exposure or dose with more realistic toxicity data. The results of the preliminary quantitative assessment either support the conclusion of minimal ecological risk, indicate an unacceptable risk, or suggest a more detailed quantitative ecological risk assessment.

The *detailed quantitative risk assessment, or DQRA,* (which is referred to as Tier 3) attempts to introduce as much realism and site-specific detail into the risk assessment as supported by current ecological and toxicological understanding. Sophisticated contaminant transport models and high resolution ecological models can be combined with rigorously defined spatial-temporal sampling and monitoring programs to produce the most scientifically defensible estimates of ecological risk. The results of the detailed assessment give estimates of ecological risk based on state-of-the-art quantitative systems analysis and modelling using the best available data, or in some instances, requiring new data to be collected.

It is important to recognize that there is a fundamental difference between a human health risk assessment (HHRA) and an ecological risk assessment. While a HHRA is concerned with estimating the effects on individuals, an ERA is concerned with estimating effects on populations, communities and ecosystems (multi-species). Environment Canada also suggests a weight of evidence approach, using the results of the ecological risk assessment along with monitoring data and test work to determine the potential for effects (Environment Canada 1997).

A screening level ecological risk assessment is carried out for the coal-fired operations at the Brandon G.S. to determine if any risks exist to ecological receptors in the vicinity of the station and whether a more detailed analysis is warranted. The screening level assessment is carried out using the four basic elements as described above.

As discussed in Section 2.3.2, in total, ecological TRVs for vegetation and CCME soil guidelines exist for 40 of the potential chemicals emitted from the Brandon G.S. (aluminum, antimony, arsenic, barium, beryllium, boron, cadmium, chromium, cobalt, copper, lead, lithium, manganese, mercury, molybdenum, nickel, selenium, silver, thallium, tin, uranium, vanadium, zinc, benzene, cyanide, ethyl benzene, phenol, tetrachloroethylene, toluene, styrene, m-xylene, benzo(a)pyrene, benzo(b)fluoranthene, chrysene, indeno(1,2,3-cd)pyrene, naphthalene, benzo(a)anthrancene, benzo(k)fluoranthene, biphenyl, and dibenzo(a,h)anthracene). As a result of the screening process discussed in Section 2, only dioxin is carried through the ecological assessment since dioxins are generally biomagnified up the food chain.

7.1 RECEPTOR CHARACTERIZATION

In the receptor characterization phase of an ecological assessment, ecological receptors of potential concern are identified. The choice of receptor is dependent on the presence in the study area, the potential pathways of exposure (exposure assessment) and the toxicity of the chemicals present at the site (hazard assessment).

7.1.1 Selection of Receptors

Figure 7.1-1 indicates the various receptors that are selected for this assessment. These receptors are chosen to represent a wide range of exposure.

Terrestrial Vegetation, and Trees

Terrestrial plants and crops comprise one of the most potentially exposed populations since these receptors reside in the soil and are therefore continuously exposed to contaminated soil. Because these receptors are not mobile or have limited mobility, they would be exposed to the contamination in place over a lifetime.

The terrestrial vegetation receptors chosen for this assessment comprise a generic terrestrial plant species which represents grasses, shrubs and trees and crops., since there is a general lack of toxicity data in this regard. The selection of this generic receptor is a typical assumption in ecological risk assessments.

Terrestrial Invertebrates

Terrestrial invertebrates or soil dwelling organisms also comprise a potentially highly exposed population since these receptors reside in the soil and are therefore continually exposed to contaminated soil.

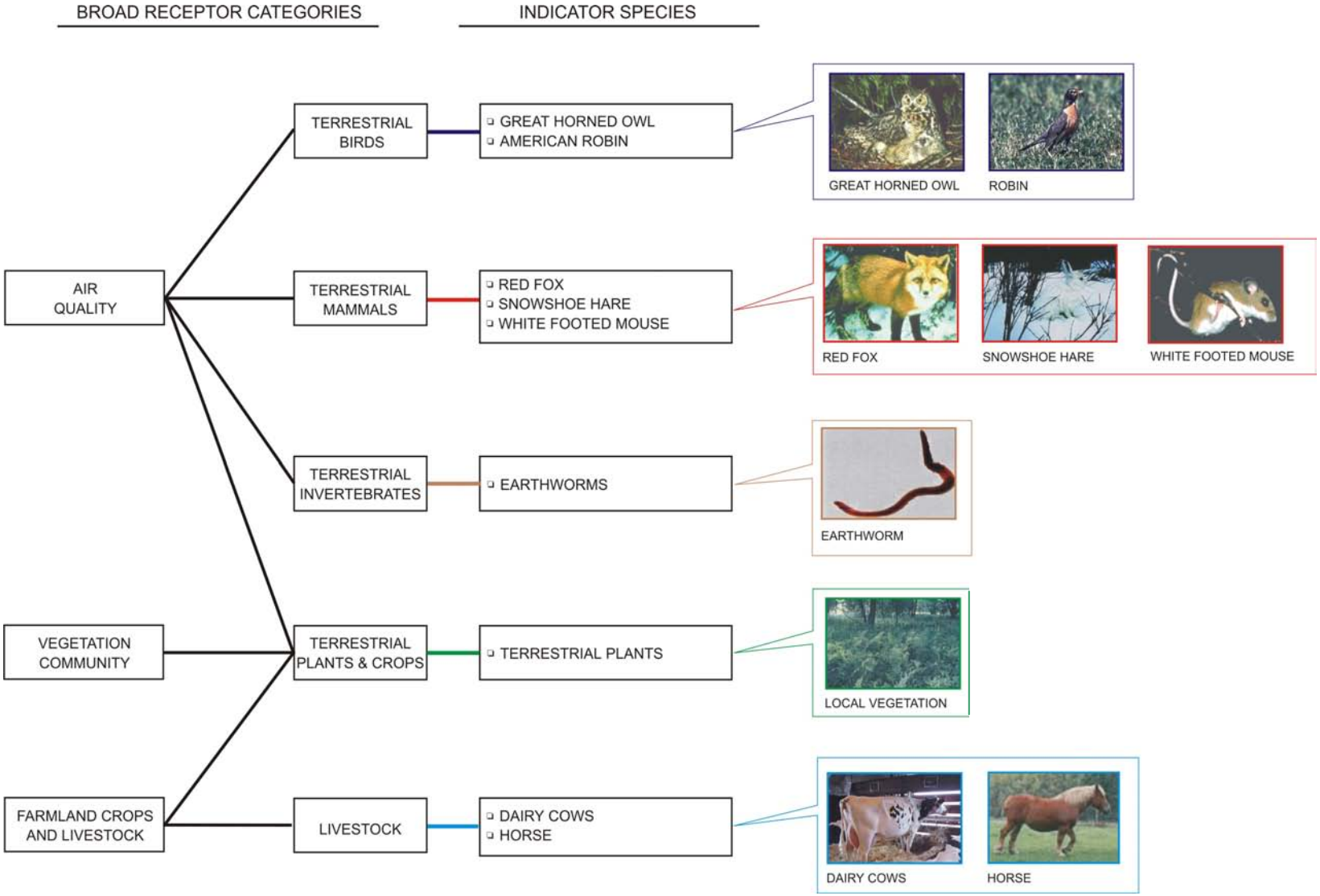
These soil dwelling organisms will act as a surrogate for effects on all soil dwelling organisms due to the fact that the most comprehensive toxicity data is available for the earthworm.

Terrestrial Mammals and Livestock

The vegetation that will be at the receptor location may provide a food source for terrestrial receptors. Small mammals such as mice will most likely inhabit the study area. In general, these small mammals are potentially exposed by consuming vegetation from the study area as well as through direct contact with the soil. These receptors generally have a limited home range in which they reside; however, due to their mobility within this range these receptors may

Figure 7.1-1

Ecological Receptors Used in the Assessment of Risks From the Coal-Fired Operation of the Brandon G.S.



experience a wide variation in their exposure. Larger, more mobile animals such as rabbits and foxes may also inhabit the ecological area surrounding the Brandon G.S. It is expected that for many of these receptors exposure to COPC at the site will be limited as these species are highly mobile and forage for food in many places.

For this assessment, the white-footed mouse is used since this receptor may come in contact with contaminated soil and will consume vegetation in the area. Rabbits and foxes are also considered. In addition, since there are farms in the study area, cows and horses are used as representative species for the farm as they are potentially exposed by grazing and ingestion of soil. Pigs and chickens were not considered as they consume food that is not grown in the study area.

The assessment endpoint for these species at the population level is the reproductive success of the selected receptors. The measurement endpoints or TRVs are No Observable Adverse Effects Levels (NOAELs) obtained from laboratory toxicity studies on surrogate mammals (such as rats).

Terrestrial Birds

Birds are also expected to be in the study area, thus both a robin, which consumes earthworms and vegetation, and a predatory owl is considered in the assessment. These two species are considered to represent a range of exposures experienced by terrestrial birds which encompasses the maximum potential exposure.

Receptor Characteristics

The characteristics for the receptors described above are provided in Table 7.1-1. These characteristics are obtained from reputable literature sources, such as the U.S. EPA (1993) *Wild Life Exposures Factor Handbook*. The entire dietary characteristics of some of the species are not accounted for since those portions of the diet are not influenced by the emissions from the Brandon G.S.

Table 7.1-1
Ecological Receptors Characteristics

	Units	Mouse	Rabbit	Fox	Cow	Horse	Owl	Robin
Body weight	kg	0.02	1.4	4.5	750	700	1.5	0.077
Vegetation ingestion	g dw/day	0.24	100	9.3	13,200	7,000		7.5
Grain ingestion	g dw/day	0.6			3,000	7,000		
Silage ingestion	g dw/day	0.36			4,100			
Earthworm ingestion	g dw/day							7.5
Mice ingestion	g dw/day						18.8	
Bird ingestion	g dw/day			18.6			18.8	

	Units	Mouse	Rabbit	Fox	Cow	Horse	Owl	Robin
Rabbit ingestion	g dw/day			65.1			37.5	
Soil ingestion	g dw/day	0.02	5.7	2.6	500	280	3.8	1.5

Note: g dw/day – grams dry weight per day

7.2 EXPOSURE ASSESSMENT

The exposure assessment considered the exposure of the various receptors to dioxins. For the assessment of potential effects on terrestrial plants and soil dwelling organisms, the TRV for dioxin in soil is compared directly to measured soil concentrations, and as such, exposure values are not calculated. For all other terrestrial animals, exposure data is needed. The dioxin emission used in this assessment was for the upper-bound emission scenario, namely OS3 – Future Scenario.

In considering the appropriate concentrations to use in the risk assessment, Receptor Locations 9, 10 and 11 were examined. Receptor Location 9 is near agricultural land for a research station, northwest of the plant, Receptor Location 10 is the Brandon Hills which are the largest tract of forested land near the site and is surrounded by different habitats such as prairie, parkland, and boreal forest and Receptor Location 11 is the location of a marsh area (Douglas Marsh) which has a large wetland area that is habitat to a large number of aquatic birds. Therefore, ducks were considered in this area. There was inadequate information available to calculate the incremental concentrations of dioxins in the sediments in the Douglas Marsh. Therefore, only the water pathway were considered. It should be noted that even though the sediment pathway of exposure was not considered in the assessment, the emissions of dioxins from the facility were so low that it is unlikely that they can be discerned to be different from background.

The predicted incremental dioxin soil concentrations for Receptor Locations 9 to 11 are provided in Table 7.2-1. The incremental soil concentrations were predicted for the maximum theoretical plant operating condition (i.e., with plant operating at 100 % C.F.). The equations used to calculate these concentrations are provided in Attachment A. As seen from the table, the incremental soil concentrations due to dioxin emissions from the Brandon G.S. are extremely low.

Table 7.2-2 provides a summary of the predicted exposures to dioxins for the various ecological receptors selected in this assessment at Locations 9 to 11. The equations used to calculate these exposures are provided in Attachment A. It should be noted that the specific ecological receptors may not be found at all locations (for example, cows and horses in the Douglas Marsh); however, they are considered representative ecological receptors for animals with similar diets.

Table 7.2-1
Predicted Incremental Dioxin in Soil Concentrations at Ecological Receptor Locations

	Soil Concentration (mg/kg) – 2,3,7,8-TCDD
Receptor Location	**OS3 - Future Scenario**
R9	$8.9x10^{-10}$
R10	$7.4x10^{-10}$
R11	$9.5x10^{-10}$

Table 7.2-2
Predicted Exposure Values (mg/kg-d) to Dioxins for the Ecological Receptors at Receptor Locations 9,10,11

Receptor Location	Mouse	Rabbit	Fox	Dairy Cow	Horse	Owl	Robin
OS3-Future Scenario							
R9	$1.53x10^{-11}$	$4.13x10^{-12}$	$5.33x10^{-13}$	$3.00x10^{-12}$	$4.49x10^{-12}$	$2.26x10^{-12}$	$7.47x10^{-10}$
R10	$1.33x10^{-11}$	$3.46x10^{-12}$	$4.45x10^{-13}$	$2.58x10^{-12}$	$3.88x10^{-12}$	$1.89x10^{-12}$	$6.23x10^{-10}$
R11	$1.68x10^{-11}$	$4.42x10^{-12}$	$5.69x10^{-13}$	$3.28x10^{-12}$	$4.92x10^{-12}$	$2.42x10^{-12}$	$7.98x10^{-10}$

As seen from the above table, the robin and mouse are predicted to have the highest exposures to dioxins emitted from the Brandon G.S.

7.3 HAZARD ASSESSMENT

An examination of various literature sources was conducted for the hazard assessment to determine the appropriate toxicity reference values for exposure to dioxins.

The objective of an ecological risk assessment is to evaluate the potential effects of a chemical on a population. Typically in risk assessments, an effects concentration value (EC_{20}) is used as a TRV. An EC_{20} value is the dose under laboratory conditions that causes an effect in 20 % of the population. For quickly reproducing populations, such as plants and earthworms, this is an acceptable TRV. For slower reproducing and less dense populations, such as larger mammals, a decrease of 20 % in population may not be acceptable. Thus, the TRV for these receptors is generally a NOAEL or No Observable Adverse Effects Level.

7.3.1 Terrestrial Plants and Soil-dwelling Organisms

Toxicity of dioxins (2,3,7,8-TCDD) to terrestrial plants and soil dwelling organisms were based on the toxicity reference value provided in the U.S. EPA Region 6 Guidance Document (1999). The toxicity reference value (TRV) for plants was not available.

Table 7.3-1 provides the TRVs for dioxins for plants and soil dwelling organisms used in this assessment. The TRVs are directly comparable to soil concentrations.

Table 7.3-1
Toxicity Reference Values for Dioxin Exposure for Plants and Earthworms

EC_{20} (mg/kg)	
Plant	**Earthworm[a]**
n/a	0.5

Note:
a) Data obtained from U.S. EPA Region 6 Guidance Document, Appendix K (1999)
n/a not available

7.3.2 Terrestrial Mammals and Livestock

For this assessment, a report produced by Sample *et al*. (1996) from the Oak Ridge National Laboratory (ORNL) was used as the primary data source. Sample *et al*. (1996) examined data from different studies and selected an appropriate toxicity value based on studies in which reproductive and developmental endpoints were considered (endpoints that may be directly related to potential population-level effects), multiple exposure levels were investigated, and the reported results were evaluated statistically to identify any significant differences from control values. The toxicity values from Sample *et al*. (1996) are generally scaled for the different ecological species based on their relative body weights (allometric scaling). This is the generally accepted approach for ecological risk assessments and provides the best foundation to obtain TRVs.

A NOAEL of 1×10^{-6} mg/kg-d for exposure to 2,3,7,8-TCDD was provided by Sample *et al*. (1996) based on reproductive effects in rats. The TRVs for selected wildlife were estimated by scaling by body weight from the test species, using the following accepted equation (5-2) taken from Sample and Arenal (1999):

$$Tox_{Biota} = Tox_{TestSpecies} \times \left[\frac{BW_{TestSpecies}}{BW_{Biota}} \right]^{1-b} \qquad (7\text{-}1)$$

The generic factor for "b" of 0.94 for mammals, provided by Sample and Arenal (1999), was used. Table 7.3-2 provides the TRVs for the terrestrial mammals and livestock derived using this methodology.

Table 7.3-2
Toxicity Reference Values for Dioxin Exposures for Terrestrial Mammals

No Observable Adverse Effects Level (NOAEL) (mg/kg-d)				
Mouse	**Rabbit**	**Fox**	**Dairy Cow**	**Horse**
1.19×10^{-6}	9.2×10^{-7}	8.58×10^{-7}	6.31×10^{-7}	6.34×10^{-7}

7.3.3 Terrestrial Birds

Toxicity reference values for birds and ducks exposed to dioxin are also scaled using the above equation. The TRV for birds are based on ring-necked pheasant (test species for birds and ducks) obtained from Sample *et al.* (1996). For birds, the generic factor "b" is 1.2. The TRVs for birds derived using this methodology are shown in Table 7.3-3.

Table 7.3-3
Dioxin Toxicity Reference Values for Terrestrial Birds

No Observable Adverse Effects Level (NOAEL) (mg/kg-d)		
Robin	**Owl**	**Duck**
8.38×10^{-6}	1.52×10^{-5}	1.40×10^{-5}

7.4 RISK CHARACTERIZATION

The risk characterization step compares the TRVs from the hazard assessment (Tables 7.3-2 and 7.3–3) to the predicted exposure for the terrestrial mammals and birds. For plants and soil dwelling organisms, the TRVs (Table 7.3-1) are compared to the maximum predicted soil concentrations. As described previously, there are no dioxin TRVs for plants and therefore they are not quantatively evaluated.

A screening index value is used to determine whether there is a potential for adverse impacts in any ecological species. The screening index is defined as the ratio of the estimated exposure or soil concentration to the ecological TRV. Due to the conservative nature of this assessment, it has been assumed that a screening index value below 0.2 indicates there is no potential for an ecological effect and a more detailed assessment is not necessary.

Tables 7.4-1 and 7.4-2 provide the screening index values for the soil dwelling organisms and the terrestrial mammals and birds, respectively for the three receptor locations (R9, R10, R11). As seen from the tables, the screening index values are orders of magnitude below 0.2. Even though, direct comparisons to ecological TRVs could not be made for plants, the exposures are very low, in addition, these effects are captured within the food chain effects for the terrestrial mammals, such as the mouse, fox and rabbit. Thus, it can safely be stated that ecological receptors will not demonstrate adverse effects from emissions arising from the coal-fired operation of the Brandon G.S. and a more detailed ecological risk assessment is not warranted.

Table 7.4-1
Screening Index Values for Plants and Earthworms Exposed to Dioxins at Receptor Location 9,10,11

Receptor Location	Plant	Earthworm
OS3 - Future Scenario		
R9	n/a	$1.8x10^{-9}$
R10	n/a	$1.5x10^{-9}$
R11	n/a	$1.9x10^{-9}$

Note: n/a - toxicity data not available

Table 7.4-2
Screening Index Values for the Terrestrial Animals and Birds Exposed to Dioxins at Receptor Locations 9,10,11

Receptor Location	Mouse	Rabbit	Fox	Dairy Cow	Horse	Owl	Robin
OS3 - Future Scenario							
R9	$1.29x10^{-5}$	$4.49x10^{-6}$	$6.21x10^{-7}$	$4.76x10^{-6}$	$7.09x10^{-6}$	$1.49x10^{-7}$	$8.91x10^{-5}$
R10	$1.12x10^{-5}$	$3.76x10^{-6}$	$5.18x10^{-7}$	$4.09x10^{-6}$	$6.12x10^{-6}$	$1.24x10^{-7}$	$7.43x10^{-5}$
R11	$1.42x10^{-5}$	$4.81x10^{-6}$	$6.64x10^{-7}$	$5.20x10^{-6}$	$7.77x10^{-6}$	$1.59x10^{-7}$	$9.52x10^{-5}$

Douglas Marsh (Receptor Location 11)

Since the Douglas Marsh area is located approximately 15 km east of Brandon area, the potential effects on duck species from exposure to dioxins emitted from the Brandon G.S. are also examined. Due to the lack of adequate information to determine sediment concentration, it is assumed that the major exposure pathway for ducks is ingestion of water. Table 7.4-3 shows the results for ducks. From the table, it can be seen that the screening index values are all well below 0.2 and as such, the coal-fired operations of the Brandon G.S. have no adverse effects on ducks at the Douglas Marsh, even if the sediment pathway was considered in the assessment.

Table 7.4-3
Screening Index Values for the Duck Receptor Exposed to Dioxins at Douglas Marsh

Dioxin Concentration	Dose from Ingestion of Water	Toxicity Reference Value*	Screening Index
(mg/L)	**(mg/kg-d)**	**(mg/kg-d)**	**(-)**
Future Scenario			
2.44×10^{-14}	1.56×10^{-15}	0.000014	1.11×10^{-10}

Note: *based on Sample *et al.* (1996)

Summary

The results of the ecological risk assessment demonstrates that there are no adverse effects on ecological receptors associated with emissions from the Brandon G.S.

7.5 SOURCES OF UNCERTAINTY

As with the human health assessment, there are also many areas of uncertainty involved in the ecological risk assessment. Several of the major assumptions used in the ecological risk assessment are outlined below.

Maximum predicted concentrations of dioxins were used to assess ecological impacts. In addition, it was assumed that all of the ecological species were present at each ecological receptor location. In all likelihood, these receptors would not all be found at all locations and may also move in the study area (for example birds and foxes) and thus may not always be exposed to the maximum concentrations of dioxin. Given that the screening level ecological assessment demonstrated that impacts are predicted to be insignificant, these changes would not result in any changes to the overall conclusion of the assessment, which states that there will be no ecological impacts from the coal-fired operation of the Brandon G.S.

No adequate toxicological database is available that determines the concentrations of dioxins that impact all ecological species. Thus, in choosing ecological receptors, general species of plants and earthworms were chosen in order to find available data. In this assessment, no TRVs were available for plants. The SI values for earthworms and other ecological receptors were all orders of magnitude below 0.2 indicating that the lack of plant toxicity data would not change the overall conclusions of the assessment.

Another area of uncertainty in the ecological risk assessment is the effect of multiple chemicals. When dealing with toxic chemicals, there is potential interaction with other chemicals that may be found at the same location. It is well established that synergism, potentiation, antagonism or additivity of toxic effects occurs in the environment. A quantitative assessment of these

interactions is outside the scope of this study and, in any event, would be constrained, as there is not an adequate base of toxicological evidence to quantify these interactions.

Since the screening level ecological risk assessment showed very small screening index values (less than 0.2), it is not anticipated that the reduction in the uncertainty surrounding this assessment will change the overall conclusion.

REFERENCES

Allied Technology Group Inc. (ATG) 1999. *Preliminary Risk Assessment: Mixed Waste Facility RCRA/TSCA Permit Application*. ATG Inc., Richland, Washington.

Ahier, B.A. and B.L. Tracy 1993. *Uranium emissions in Port Hope, Ontario.* Report to the Atomic Energy Control Board.

Agency for Toxic Substances and Disease Registry (ATSDR) 2001. *Toxicological Profile for Colbalt.*

Baes, G.F., R.D. Sharp, A.L. Sjoreen, R.W. Shaw 1984. *A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides in Agriculture.* Oak Ridge National Laboratory, Oak Ridge, TN (ORNL-5786).

Burnett, R.T., *et al.* 2004. *Associations between short-term changes in nitrogen dioxide and mortality in Canadian cities.* Archives of Environmental Health 59: 228-236, as cited in WHO (2005).

Boermans, Dr. H. 2000. *Personal Communication*. Department of Biomedical Sciences, University of Guelph.

Burton, J. 1997. *North Carolina Protocol for Performing Indirect Risk Assessments for Hazardous Waste Combustion Units*. Division of Waste Management, 92D-6726-000.

Canadian Council of Ministers of the Environment (CCME) 2003. *Canadian Environmental Quality Guidelines.* Includes updates to the Original 1999 Version.

Canadian Council of Ministers of the Environment (CCME) 1997. *A Framework for Ecological Risk Assessment: Technical Appendices.* March.

Canadian Council of Ministers of the Environment (CCME) 1996. *A Framework for Ecological Risk Assessment: General Guidance.* March.

Canadian Environmental Protection Act/Federal Provincial Advisory Committee Working Group on Air Quality Objectives and Guidelines (CEPA/FPAC WGAQOG), 1999. *National Ambient Air Quality Objectives for Particulate Matter, Part 1: Science Assessment Documents*. Health Canada, Environment Canada

Dann, T. 2001. *Trends in Ambient Levels of CWS Pollutants*. Environment Canada, EPS, Ottawa, Ontario. Presented at the Air & Waste management Specialty Conference on Implementation of Canada-Wide Standards, March 7-8, 2001, Toronto, Ontario.

Dominici, F., R.D. Peng, M.L. Bell, L. Pham, A. McDermott, S. Zeger and J. Samet. 2006. *Fine Particulate Air Pollution and Hospital Admission for Cardiovascular and Respiratory Diseases.* JAMA, 295(10), 1127-1134

Efroymson, R.A., B.E. Sample, M.S. Aplin, G.W. Suter II, C.J.E. Welsh 1997. *Methods and Tools for Estimation of the Exposure of Terrestrial Wildlife to Contaminants*. Environmental Sciences Division Publication No. 4650, Prepared for United States Department of Energy by Oak Ridge National Laboratory, ORNL/TM-13391, October.

Environment Canada 1997. *Environmental Assessments of Priority Substances Under the Canadian Environmental Protection Act*. Guidance Manual Version 1.0 – March 1997. EPS 2/CC/3E.

Environment Canada 1999. *Ambient Particulate Matter: An Overview*. From National Ambient Air Quality Objectives for Particulate Matter: Part 1: Science Assessment Document by the Federal Provincial Working Group on Air Quality Objectives and Guidelines.

Government of Canada 1991. *The State of Canada's Environment*. Ministry of Supply and Services Canada, Ottawa, Catalogue No. EN21-54/1991E.

Guidotti, T. 1996. *Ambient Air Quality and Human Health: Current Concepts, Part 2*. Can. Respir. J. 3(1): 29 – 39.

Health Canada 2004. *Federal Contaminated Site Risk Assessment in Canada – Part II: Health Canada Toxicological Reference Values (TRVs).*

Health Canada/Environment Canada 1999. *National Ambient Air Quality Objectives for Particulate Matter. Part 1: Science Assessment Document*. A report by the CEPA/FPAC Working Group on Air Quality Objectives and Guidelines. Bureau of Chemical Hazards, Environmental Health Directorate, Health Canada, Ottawa, ON/Science Assessment and Policy Integration Division, Atmospheric Environment Services, Environment Canada, Toronto, ON.

Environment Canada and Health Canada 1994. *Preparing the Second Priority Substances List Under the Canadian Environmental Protection Act (CEPA).* Response to Stakeholder's Comments on the Federal Government Proposals.

Jerrett, M., R.T. Burnett, R. Ma, C.A. Pope III, D. Krewski, K.B. Newbold, G. Thurston, Y. Shi, N. Finkelstein, E.E. Calle and M.J. Thun 2005. *Spatial Analysis of Air Pollution and Mortality in Los Angeles.* Epidemiology, 16(6), 727-736.

Krewski, D., R.T. Burnett, M.S. Goldberg *et al*. 2000. *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality*. Health Effects Research Special Report, Parts 1 and 2, July 2000. Health Effects Institute, Cambridge, MA.Laden, F., L.M. Neas, D.M. Dockery and J. Schwartz 2000. *Association of Fine Particulate Matter from Different Sources with Daily Mortality in Six U.S. Cities*. Environ. Health Perspectives 108: 941-947.

Laden, F., J. Schwartz, F.E. Speizer and D.W. Dockery 2006. *Reduction in Fine Particulate Air Pollution and Mortality.* Am. J. Respir. Crit. Care Med., 173, 667-672.

Laden F; Neas LM; Dockery DW; Schwartz J. 2000. *Association of Fine Particulate Matter from Different Sources with Daily Mortality in Six U.S. Cities.* Environ Health Perspect 108:941-947.

Levy, J.I., J.K. Hammett and J.D. Spengler 2000. *Estimating the Mortality Impacts of Particulate Matter: What Can be Learned from Between Study Variability*. Environ. Health Perspectives 108(2): 107 – 117.

Lippmann, M., K. Ito, A. Nadas and R.T. Burnett 2000. *Association of Particulate Matter Components with Daily Mortality and Morbidity in Urban Populations*. Health Effects Institute Research Report Number 95. Health Effects Institute, Cambridge, MA.

ORTECH Environmental, 2005. Comprehensive Stack Gas Emission Testing Program at Manitoba Hydro, Brandon Generating Station, Unit No. 5. Prepared for Manitoba Hydro, Winnipeg, MB.

Perry, R.H. and C.H. Chilton 1973. *Chemical Engineer's Handbook*. 5th Edition. McGraw Hill Book Company.

Puls, R. 1994. *Mineral Levels in Animal Health: Diagnostic Data*. 2nd Edition. Sherpa International. ISBN 0969342926.

Riediker, M., W.E. Cascio, T.R. Griggs, M.C. Herbst, P.A. Bromberg, L. Neas, R.W. Williams and R.B. Devlin 2004. *Particulate Matter Exposure in Cars Is Associated With Cardiovascular Effects In Healthy Young Men.* Am. J. Respir. Crit. Care Med., 169, 934-940.

Richardson, G.M. 1997. *Compendium of Canadian Human Exposure Factors for Risk Assessment*. O'Connor Associates Environmental Inc.

Samet, J.M., F. Dominici, F.C. Curriero, I. Coursac and S.L. Zeger 2000. *Fine Particulate Air Pollution and Mortality in 20 U.S. Cities*. The New England Journal of Medicine, 343(24): 1742-1749.

Sample, B.E., D.M. Opresko, G.W. Suter II 1996. *Toxicological Benchmarks for Wildlife: 1996 Revision*. June 1996.

Sample, B.E. and C.A. Arenal 1999. *Allometric models for interspecies extrapolation of wildlife toxicity data.* Bulletin of Environmental Contamination and Toxicology. 62:656-663.

SENES Consultants Limited 1992. *Environmental Impact Assessment for Sub-Bituminous Coal-Fired Generation Brandon Generating Station*. Prepared for Manitoba Hydro.

Suter, G.W. II and C.L. Tsao 1996. *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision.* Prepared by Risk Assessment Program, Lockheed Martin Energy Systems, Inc., Oak Ridge, Tennessee. Prepared for the U.S. Department of Energy.

Tracy, B.L. and F.A. Prantl 1985. *Radiological impact of coal-fired power generation*. J. Environ. Radioactivity, Vol. 2 pp. 145-160.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) 1993. *Sources and Effects of Ionizing Radiation*.

United States Department of Energy (U.S. DOE) 2001. *User's Manual for RESRAD*. Version 6. July.

United States Environmental Protection Agency (U.S. EPA). 2005a. Integrated Risk Information System (IRIS). Values updated monthly on line at http://www.epa.gov/iris/. Values presented are current as of January 2006.

United States Environmental Protection Agency (U.S. EPA.) 2005b. *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities*. Final. EPA Region 6, Office of Solid Waste. September.

U.S. Environmental Protection Agency (U.S. EPA.) 2004. *Air Quality Criteria for Particulate Matter. Volume I of II.* Washington D.C. U.S. EPA, Office of Research and Development Publication No. EPA /600/P-99/002aF.

United States Environmental Protection Agency (U.S. EPA) 2001. *National Center for Environmental Assessment – NCEA.*

United States Environmental Protection Agency (U.S. EPA.) 1999. *Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities*. Peer Review Draft. EPA Region 6, Office of Solid Waste. August.

United States Environmental Protection Agency (U.S. EPA) 1998. *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities*. Peer Review Draft. EPA Region 6, Office of Solid Waste, July.

U.S. Environmental Protection Agency (U.S. EPA) 1997. *Health Effects Assessment Summary Tables (HEAST): Annual Update*, FY 1997. National Center For Environmental Assessment (NCEA), Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (U.S. EPA) 1996. *Air Quality Criteria for Particulate Matter.* Washington D.C. U.S. EPA, Office of Research and Development Publication No. EPA /600/P-95/001cF.

United States Environmental Protection Agency (U.S. EPA) 1995. *Compilation of Air Pollutant Emission Factors (AP-42) Volume I: Stationary Point and Area Sources*. Chapter 1.3 – Fuel Oil Combustion.

United States Environmental Protection Agency (U.S. EPA) 1993. *Wildlife Exposure Factors Handbook*. (Volumes I and II) Washington, D.C., Office of Research and Development, EPA/600/R-93/187a and EPA/600/R-93/187b.

United States Environmental Protection Agency (U.S. EPA) 1992. *Screening Procedures for Estimating the Air Quality Impact of Stationary Sources, Revised*. Office of Air and Radiation, Office of Air Quality Planning and Standards. EPA/454/R-92-019. Research Triangle Park, North Carolina.

United States Environmental Protection Agency (U.S. EPA). 1991. *Risk Assessment Guidance for Superfund Volume 1: Human Health Evaluation Manual (Part B, Development of Risk-Based Preliminary Remediation Goals).* Publication 9285.7-01B. Office of Emergency and Remedial Response, Washington, DC. NTIS PB92-963333.

United States Environmental Protection Agency (U.S. EPA) 1990. *Clean Air Act Amendments of 1990.*

United States Environmental Protection Agency (U.S. EPA) 1989. *Risk Assessment Guidance for Superfund: Volume I. Human Health Evaluation Manual (Part A).* OERR. Washington, D.C. OERR 9200 6-303-894.

United States National Council on Radiation Protection and Measurements (NCRP) 1975. *Natural Background Radiation in the United States*. NCRP Report No. 45.

Weinhold, B. 2000. *A Clearer Look at PM_{10}*. Environ. Health Perspectives, 108: A520.

Will, M.E. and G.W. Suter II 1995. *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision*.

World Health Organization (WHO) 2005. *WHO Air Quality Guidelines Global Update.* Report on a Working Group Meeting, Bonn, Germany, 18-20 October 2005.

World Health Organization (WHO) 2004. *Health Aspects of Air Pollution: Results from the WHO Project "Systematic Review of Health Aspects of Air Pollution in Europe".* WHO Regional Office for Europe, Copenhagen, Denmark.

World Health Organization (WHO) 2000. *Air Quality Guidelines for Europe*. Second Edition. WHO Regional Publications, European Series, No.91. Regional Office for Europe, Copenhagen.

Zanobetti, A., J. Schwartz, D. Gold 2000a. *Are There Sensitive Subgroups for the Effects of Airborne Particles?* Environ. Health Perspect. 108: 841-845.

Zanobetti, A., J. Schwartz, D. Gold 2000b. *Airborne Particles Are A Risk Factor For Hospital Admissions For Heart And Lung Disease*. Environ. Health Perspect. 108: 1071–1077.

ATTACHMENT A

Pathways Calculations

ATTACHMENT A

The equations listed here follow the human health risk assessment protocol by U.S. EPA (1998).

A.1 INHALATION PATHWAY

The dose due to inhalation (mg/kg-d) is:

$$Dose = \frac{R_{inh} C_a}{B_w} \times \frac{1}{1000} \quad \text{(A-1)}$$

where:

R_{inh}	=	inhalation rate (m^3/day) [receptor-specific];
C_a	=	concentration of constituent in air ($\mu g/m^3$) [calculated (A-2)];
B_w	=	body weight of receptor (kg) [receptor-specific];
1/1000	=	unit conversion (mg/µg).

The concentration of constituent in air (C_a) is calculated following:

$$C_a = F_v \times C_{av} + (1 - F_v) \times C_{ap} \quad \text{(A-2)}$$

where:

F_v	=	fraction of chemical in vapour phase (-) [chemical-specific];
C_{av}	=	vapour concentration ($\mu g/m^3$) [calculated (A-3)];
C_{ap}	=	particle concentration ($\mu g/m^3$) [calculated (A-4)].

The vapour concentration (C_{av}) is calculated as:

$$C_{av} = Q \times C_{v-source} \quad \text{(A-3)}$$

where:

Q	=	emission rate (g/s) [chemical-specific];
$C_{v\text{-source}}$	=	modelled vapour source concentration (($\mu g/m^3$)/(g/s)) [receptor-specific].

And the particle concentration (C_{ap}) is calculated as:

$$C_{ap} = Q \times C_{p-source} \quad \text{(A-4)}$$

where:

Q	=	emission rate (g/s) [chemical-specific];
$C_{p\text{-source}}$	=	modelled particle source concentration (($\mu g/m^3$)/(g/s)) [receptor-specific].

A.2 INGESTION PATHWAY

The general form of the equation used to determine the dose due to the ingestion of constituents in dust, soil, vegetation, beef, or milk is:

$$Dose = \frac{R_{ing} C_x}{B_w} \times F_{location} \times \frac{1}{units\_conv} \quad \text{(A-5)}$$

where:

R_{ing} = ingestion rate (g/d) [receptor-specific];
B_w = body weight of receptor (kg) [receptor-specific];
C_x = concentration of constituent ∀ x, such that:
  C_{soil} – (mg/kg) [calculated in (A-6)];
  C_{dust} – (mg/kg) [assumed to = C_{soil}];
  C_{veg} – (g/kg) [calculated in (A-20)];
  C_{beef} – (g/kg) [calculated in (A-24)];
  C_{milk} – (g/kg) [calculated in (A-25)];
$F_{location}$ = fraction of day at location for soil and dust ingestion and fraction from local source for veg, beef, and milk ingestion (-) [receptor-specific];
units_conv= 1000 g/kg for veg, beef, and milk and 1000000 mg/kg for soil and dust.

The concentration of the constituent in soil is calculated for carcinogens following (A-6).

$$C_{soil} = \frac{\left(\frac{Ds \times Tc - Sc_{TC}}{ks}\right) + \left(\frac{Sc_{TC}}{ks} \times \left[1 - e^{(-ks(T_2 - Tc)}\right]\right)}{(T_2 - T_1)} \quad \text{(A-6)}$$

where:

Ds = deposition term (mg/(kg yr)) [calculated (A-7)];
Tc = time period over which deposition occurs (yr) [assumed to be 8];
Sc_{TC} = soil concentration at time Tc (mg/kg) [calculated (A-12)];
ks = soil loss constant (1/yr) [calculated (A-13)];
T_2 = length of exposure duration (yr) [receptor-specific];
T_1 = time at beginning of exposure period (yr) [adult = 0, child = Tc-6 = 2].

The deposition term (Ds) is calculated as:

$$Ds = \frac{100}{z \times BD} \times \left[F_v (D_{dv} + D_{wv}) + (1 - F_v)(D_{dp} + D_{wp})\right] \quad \text{(A-7)}$$

where:

100 = units conversion factor ((mg m^2)/(kg cm^2));
z = soil mixing depth (cm) [tilled = 20, forage = 1];

BD = soil bulk density (g/cm^3) [assumed to be 1.5];
F_v = fraction of chemical in vapour phase (-) [chemical-specific];
D_{dv} = dry deposition from vapour phase ($g/(m^2\ yr)$) [calculated (A-8)];
D_{wv} = wet deposition from vapour phase ($g/(m^2\ yr)$) [calculated (A-9)];
D_{dp} = dry deposition from particle phase ($g/(m^2\ yr)$) [calculated (A-10)];
D_{wp} = wet deposition from particle phase ($g/(m^2\ yr)$) [calculated (A-11)].

The soil mixing depth (z) changes depending on the type of exposure being calculated. For soil ingestion (both human and cow) and root uptake for forage vegetation, the soil concentration calculated with z for forage was used. This was a conservative assumption, since soil concentrations for forage soils are generally higher than soil concentrations for tilled soils because the constituent is dispersed through a smaller region (1 cm vs. 20 cm) and therefore is found in greater concentrations. The tilled soil concentration was used for root uptake by above-ground vegetables and silage because these vegetation types are grown on tilled soil.

The dry deposition from the vapour phase (D_{dv}) is calculated following:

$$D_{dv} = Q \times C_{d\ dep\ v} \quad \text{(A-8)}$$

where:

Q = emission rate (g/s) [chemical-specific];
$C_{d\ dep\ v}$ = modelled vapour dry deposition ($(g/(m^2\ yr))/(g/s)$) [receptor-specific].

The wet deposition from the vapour phase (D_{wv}) is calculated following:

$$D_{wv} = Q \times C_{w\ dep\ v} \quad \text{(A-9)}$$

where:

Q = emission rate (g/s) [chemical-specific];
$C_{w\ dep\ v}$ = modelled vapour wet deposition ($(g/(m^2\ yr))/(g/s)$) [receptor-specific].

The dry deposition from the particle phase (D_{dp}) is calculated following:

$$D_{dp} = Q \times C_{d\ dep\ p} \quad \text{(A-10)}$$

where:

Q = emission rate (g/s) [chemical-specific];
$C_{d\ dep\ p}$ = modelled particle dry deposition ($(g/(m^2\ yr))/(g/s)$) [receptor-specific].

The wet deposition from the particle phase (D_{wp}) is calculated following:

$$D_{wp} = Q \times C_{w\ dep\ p} \quad \text{(A-11)}$$

where:

Q = emission rate (g/s) [chemical-specific];
$C_{w\ dep\ p}$ = modelled particle wet deposition ($(g/(m^2\ yr))/(g/s)$) [receptor-specific].

The soil concentration at time Tc (Sc_{Tc}) is calculated following:

$$Sc_{Tc} = \frac{Ds \times \left(1 - e^{(-ks \times Tc)}\right)}{ks} \quad \text{(A-12)}$$

where:

Ds	=	deposition term (mg/(kg yr)) [calculated (A-7)];
ks	=	soil loss constant (1/yr) [calculated (A-13)];
Tc	=	time period over which deposition occurs (yr) [assumed to be 8].

The soil loss constant (ks) accounts for the loss of contaminant from soil by several mechanisms and is calculated following:

$$ks = ksl + kse + ksr + ksg + ksv \quad \text{(A-13)}$$

where:

ksl	=	loss constant due to leaching (1/yr) [calculated (A-14)];
kse	=	loss constant due to soil erosion (1/yr) [calculated (A-15)];
ksr	=	loss constant due to surface runoff (1/yr) [calculated (A-18)];
ksg	=	loss constant due to degradation (1/yr) [assumed to be 0];
ksv	=	loss constant due to volatilization (1/yr) [calculated (A-19)].

The loss constant due to leaching (ksl) is calculated following:

$$ksl = \frac{q}{\Theta_s \times z \times \left[1 + \left(\frac{BD \times Kd_s}{\Theta_s}\right)\right]} \quad \text{(A-14)}$$

where:

q	=	average annual recharge (cm/yr) [assumed to be 5];
Θ_s	=	soil volumetric water content (mL/cm^3) [assumed to be 0.2];
z	=	soil mixing depth (cm) [tilled = 20, forage = 1];
Kd_s	=	soil-water partition coefficient (cm^3/g) [chemical-specific];
BD	=	soil bulk density (g/cm^3) [assumed to be 1.5].

The loss constant due to soil erosion (kse) is calculated following:

$$kse = \frac{0.1 \times X_e \times SD \times ER}{BD \times z} \times \left(\frac{Kd_s \times BD}{\Theta_s + (Kd_s \times BD)}\right) \quad \text{(A-15)}$$

where:

X_e	=	unit soil loss ($kg/(m^2\ yr)$) [calculated (A-16)];
SD	=	sediment delivery ratio (-) [calculated (A-17)];
ER	=	contaminant enrichment ratio (-) [assumed to be 3];
BD	=	soil bulk density (g/cm^3) [assumed to be 1.5];
z	=	soil mixing depth (cm) [tilled = 20, forage = 1];

Kd_s = soil-water partition coefficient (cm^3/g) [chemical-specific];
Θ_s = soil volumetric water content (mL/cm^3) [assumed to be 0.2].

The unit soil loss (X_e) is calculated using the Universal Soil Loss Equation for the soil loss rate from the watershed. The result is used in the soil erosion load equation.

$$X_e = RF \times K \times LS \times C \times P \times \frac{907.18}{4047} \quad \text{(A-16)}$$

where:

RF = USLE rainfall (or erosivity) factor (1/yr) [assumed to be 55.5];
K = USLE erodibility factor (ton/acre) [assumed to be 0.36];
LS = USLE length-slope factor (-) [assumed to be 1.5];
C = USLE cover management factor (-) [assumed to be 0.1];
P = USLE supporting practice factor (-) [assumed to be 1];
907.18 = conversion factor (kg/ton);
4047 = conversion factor (m^2/acre).

The sediment delivery ratio for the watershed is used in the soil erosion load equation and is calculated as follows:

$$SD = a \times \left(WA_L\right)^{-b} \quad \text{(A-17)}$$

where:

WA_L = watershed area receiving fallout (m^2) [assumed to be 10000];
b = empirical slope coefficient (-) [assumed to be 0.125];
a = empirical intercept coefficient (-) [assumed to be 1.4].

The contaminant loss constant due to runoff from soil (ksr) is calculated as follows:

$$ksr = \frac{R}{\Theta_s \times z} \times \left(\frac{1}{1 + \left(\frac{BD \times Kd_s}{\Theta_s} \right)} \right) \quad \text{(A-18)}$$

where:

R = average annual runoff (cm/yr) [assumed to be 2.5];
Θ_s = soil volumetric water content (mL/cm^3) [assumed to be 0.2];
z = soil mixing depth (cm) [tilled = 20, forage = 1];
BD = soil bulk density (g/cm^3) [assumed to be 1.5];
Kd_s = soil-water partition coefficient (cm^3/g) [chemical-specific].

The contaminant loss constant due to volatilization from soil (ksv) is calculated following:

$$ksv = \left(\frac{3.1536 \times 10^7 \times H}{z \times Kd_s \times R \times T \times BD} \right) \times \left(0.482 \times u^{0.78} \times \left(\frac{\mu_a}{\rho_a \times D_a} \right)^{-0.67} \times \left(\sqrt{\frac{4 \times A}{\pi}} \right)^{-0.11} \right) \quad \text{(A-19)}$$

where:

3.15×10^7	=	conversion constant (s/yr);
H	=	Henry's Law constant (atm m^3/mol) [chemical-specific];
z	=	soil mixing depth (cm) [tilled = 20, forage = 1];
BD	=	soil bulk density (g/cm^3) [assumed to be 1.5];
Kd_s	=	soil-water partition coefficient (cm^3/g) [chemical-specific];
R	=	universal gas constant ((atm m^3)/(mol K)) [assumed to be 8.205×10^{-5}];
T	=	ambient air temperature (K) [assumed to be 285.15];
u	=	average annual wind speed (m/s) [assumed to be 3.16];
μ_a	=	viscosity of air (g/(cm s)) [assumed to be 1.81×10^{-4}];
ρ_a	=	density of air (g/cm^3) [assumed to be 1.2×10^{-3}];
D_a	=	diffusivity of chemicals in air (cm^2/s) [chemical-specific];
A	=	surface area of contaminanted area (m^2) [assumed to be 1×10^8].

The contaminant concentration in vegetation (C_{veg}) is calculated following (A-20) and includes the uptake of chemicals by roots, the direct deposition of chemicals from the air to vegetation surfaces, and the direct uptake by plant leaves of vapour phase chemicals in the air.

$$C_{veg} = C_r + C_d + C_v \quad \text{(A-20)}$$

where:

C_r	=	concentration in plant from root uptake (mg/kg DW) [calculated (A-21)];
C_d	=	concentration in plant from direct deposition (mg/kg DW) [calc (A-22)];
C_v	=	concentration in plant from air-to-plant transfer (mg/kg DW) [calc (A-23)].

The contaminant concentration in above ground vegetation due to direct uptake of chemicals from soil (C_r) is calculated as shown in (A-21). For vegetation, the tilled soil concentration was used with the Br for leafy vegetation. For forage, the forage soil concentration was used with the Br for forage and for silage, the tilled soil concentration was used with the Br for forage.

$$C_r = C_{soil} \times Br \quad \text{(A-21)}$$

where:

C_{soil}	=	contaminant concentration in soil (mg/kg) [calculated (A-6)];
Br	=	plant-soil bioconc. factor for veg ((µg/g DW)/(µg/g soil)) [chem.-specific].

The contaminant concentration in above-ground vegetation due to wet and dry deposition of contaminant to the plant surface (C_d) is calculated as follows:

$$C_d = \frac{1000 \times (1 - F_v) \times \left(D_{dp} + \left(Fw \times D_{wp}\right)\right) \times Rp \times \left[\left(1 - e^{(-kp \times Tp)}\right)\right]}{Yp \times kp} \qquad \text{(A-22)}$$

where:

1000	=	units conversion factor (mg/g);
F_v	=	fraction of chemical in vapour phase (-) [chemical-specific];
D_{dp}	=	dry deposition from particle phase (g/(m^2 yr)) [calculated (A-10)];
D_{wp}	=	wet deposition from particle phase (g/(m^2 yr)) [calculated (A-11)];
Fw	=	fraction of wet deposition that adheres to plant (-) [assumed 0.6];
Rp	=	interception fraction edible portion [veg =0.04, forage = 0.5, silage = 0.46];
kp	=	plant surface loss coefficient (1/yr) [assumed to be 18];
Tp	=	length of plant exposure to deposition of edible portion of plant (yrs); [veg = 0.16, forage = 0.12, silage = 0.16];
Yp	=	yield or standing crop biomass of the edible portion of plant (kg DW/m^2); [veg = 1.6, forage = 0.24, silage = 0.8].

The contaminant concentration in aboveground vegetation due to the direct uptake of vapour phase chemicals into the plant leaves (C_v) is calculated as follows:

$$C_v = F_v \times \frac{C_{av} \times Bv \times VG_{ag}}{\rho_s} \qquad \text{(A-23)}$$

where:

F_v	=	fraction of chemical in vapour phase (-) [chemical-specific];
C_{av}	=	concentration in vapour phase (µg/m^3) [calculated (A-3)];
Bv	=	air-to-plant biotransfer factor ((mg/kg plant DW)/(µg/g air)) [chem-spf];
VG_{ag}	=	empirical correction (-) [assumed to be 1];
ρ_s	=	density of air (g/m^3) [1.2 x 10^3].

The concentration in beef (C_{beef}) (mg chemical/kg FW) due to plant and soil ingestion is calculated following:

$$C_{beef} = \left(F_i\left((Qp_f \times P_f) + (Qp_s \times P_s) + (Qp_g \times P_g)\right) + (Q_{soil} \times C_{soil})\right) \times BA_{beef} \qquad \text{(A-24)}$$

where:

F_i	=	fraction of plant grown on local soil, eaten by animal (-) [assumed to be 1];
Qp_f	=	quantity of forage eaten by animal (kg plant DW/day) [assumed to be 8.8];
P_f	=	total concentration of pollutant in forage (mg/kg DW) [calculated (A-20)];
Qp_s	=	quantity of silage eaten by animal (kg plant DW/day) [assumed to be 2.5];

P_s = total concentration of pollutant in silage (mg/kg DW) [calculated (A-20)];
Qp_g = quantity of grain eaten by animal (kg plant DW/day) [assumed to be 0.47];
P_g = total concentration of pollutant in grain (mg/kg DW) [calculated (A-20)];
Q_{soil} = quantity of soil eaten by animal (kg soil/day) [assumed to be 0.5];
C_{soil} = soil concentration (mg/kg) [calculated (A-6)];
BA_{beef} = biotransfer factor for beef (d/kg) [chemical specific].

The concentration in cow's milk (C_{milk}) (mg chemical/kg FW) due to plant and soil ingestion is calculated as follows:

$$C_{milk} = \left(F_i\left((Qp_f \times P_f) + (Qp_s \times P_s) + (Qp_g \times P_g)\right) + (Q_{soil} \times C_{soil})\right) \times BA_{milk} \qquad \text{(A-25)}$$

where:

F_i = fraction of plant grown on local soil, eaten by animal (-) [assumed to be 1];
Qp_f = qty of forage eaten by animal (kg plant DW/day) [assumed to be 13.2];
P_f = total concentration of pollutant in forage (mg/kg DW) [calculated (A-20)];
Qp_s = quantity of silage eaten by animal (kg plant DW/day) [assumed to be 4.1];
P_s = total concentration of pollutant in silage (mg/kg DW) [calculated (A-20)];
Qp_g = quantity of grain eaten by animal (kg plant DW/day) [assumed to be 3.0];
P_g = total concentration of pollutant in grain (mg/kg DW) [calculated (A-20)];
Q_{soil} = quantity of soil eaten by animal (kg soil/day) [assumed to be 0.5];
C_{soil} = soil concentration (mg/kg) [calculated (A-6)];
BA_{milk} = biotransfer factor for milk (d/kg) [chemical specific].

A.3 INGESTION FOR INFANT

The calculation of exposure to infants of benzo(a)pyrene (mg/kg-d) is calculated as follows:

$$Dose_{infant} = \frac{C_{breast\ milk} \times I_{breast\ milk} \times B_{oral}}{BW_{infant}} \qquad \text{(A-26)}$$

where:

$C_{breast\ milk}$ = concentration in breast milk (mg/g milk) [calculated (A-27)];
$I_{breast\ milk}$ = infant ingestion rate of breast milk (g/day) [assumed to be 800];
B_{oral} = oral bioavailability (-) [assumed to be 1];
BW_{infant} = body weight of infant (kg) [assumed to be 10].

The concentration of benzo(a)pyrene in breast milk is related to the mother's ingestion as follows:

$$C_{breast\ milk} = \frac{Dose_{mother} \times BW_{mother} \times TF_{bm}}{1000} \qquad \text{(A-27)}$$

where:

$Dose_{mother}$ = ingestion dose (A-5) + inhalation dose (A-1) for b(a)p (mg/kg-day);
BW_{mother} = body weight of mother (kg) [assumed to be 70];
TF_{bm} = breast milk bio-transfer factor (mg/kg milk)/(mg/day) [calculated (A-28)];
1000 = unit conversion factor (g/kg).

The breast milk bio-transfer factor (mg/kg milk)/(mg/day) is calculated following:

$$TF_{bm} = 2 \times 10^{-7} \times K_{ow} \quad \text{(A-28)}$$

where:

K_{ow} = octanol-water partition coefficient for b(a)p [assumed to be 1.35×10^{6}].

A.4 ECOLOGICAL SCREENING RISK ASSESSMENT CALCULATIONS

The assessment for toxicity to animals uses the estimated contaminant concentrations in soil, vegetation or flesh to calculate screening benchmark concentrations for terrestrial species. Exposures (mg/kg-d) are calculated as follows:

$$Exposure = \frac{C \times I}{BW} \quad \text{(A-29)}$$

where:

C = cont conc in plant, soil, flesh (mg/kg) [calculated (A-30), (A-6), (A-31)];
I = intake of plant, soil, flesh (kg/day) [receptor-specific];
BW = body weight (kg) [receptor-specific].

The calculation of the contaminant concentration in plants for the ecological screening assessment is less detailed than the calculation used for human health risk assessments (see (A-20) to (A-23)). The estimated contaminant concentration in plants for the ecological screening assessment is calculated as follows:

$$C_{plant} = C_{soil} \times TF_{soil-plant} \quad \text{(A-30)}$$

where:

C_{soil} = contaminant concentration in soil (mg cont/kg soil) [calculated (A-6)];
$TF_{soil\text{-}plant}$ = soil-plant transfer factor (mg cont/kg plant)/(mg cont/kg soil) [chem-specific].

Contaminant concentrations in flesh for terrestrial animals are obtained in much the same way. The contaminant ingested by a terrestrial animal is factored to estimate the transfer of a contaminant from the feed ingested to the flesh of the animal, which is subsequently eaten by another animal. This calculation is shown in (A-31).

$$C_{flesh} = C_{feed} \times I \times TF_{feed-flesh} \quad \text{(A-31)}$$

where:

C_{feed} = cont concentration in feed component (mg/kg feed) [calculated (A-30)];
I = intake of feed (kg feed/day) [receptor-specific];
$TF_{feed\text{-}flesh}$ = transfer factor (day/kg DW) [chemical-specific].

Transfer factors are available for feed-to-beef or feed-to-chicken. Values for feed-to-beef are assumed the same for all terrestrial animals (e.g., rabbit), and feed-to-chicken are used for all poultry and birds (e.g., robin). When feed-to-chicken factors are not available, they are assumed to be equivalent to the feed-to-beef factor.

The concentration of chemicals in earthworms is calculated using equation (A-32).

$$C_{worm} = C_{soil} \times K_{sw} \quad \text{(A-32)}$$

where:

C_{soil} = contaminant concentration in soil (mg cont/kg soil) [calculated (A-6)];
K_{sw} = worm-soil partitioning coefficient (kg soil/kg worm) [chem.-spc/calc].

Where K_{sw} were not available in the literature, they were calculated using the formula (A-33) from Sample *et al*. (1997).

$$K_{sw} = L\left(0.66 \times f_{oc}\right)^{-1} \quad \text{(A-33)}$$

where:

L = proportion of lipid in worm (-) [assumed to be 0.14, avg of values in Sample *et al.* (1997)];
f_{oc} = fraction of organic carbon in soil [assumed to be 0.01].

ATTACHMENT B

Results of Risk Assessment Calculations

ATTACHMENT B

This attachment provides all the results for the pathways modelling and risk assessment for the coal-fired operation of the Brandon G.S. Section B.1 provides the ingestion dose from soil, vegetation, beef and milk. Section B.2 provides the results of the risk calculations at all nine receptor locations. Section B.3 gives the calculated maximum air concentrations at all nine receptor locations.

B.1 CALCULATED DOSES

Table B.1-1
Calculated Soil Ingestion Dose (mg/kg-d) – Scenario OS3

(mg/kg-d)	Receptor 1	Receptor 2		Receptor 3	Receptor 4		Receptor 5
	Industrial Worker	Resident		School	Resident		School
COPC	Adult	Adult	Composite	Adult	Adult	Composite	Adult
Arsenic	3.11E-18	1.53E-18	2.26E-18	6.01E-18	9.21E-18	1.36E-17	7.34E-18
Beryllium	5.42E-16	4.16E-16	5.19E-16	1.05E-15	2.50E-15	3.11E-15	1.28E-15
Cadmium	1.98E-17	1.52E-17	1.89E-17	3.81E-17	9.09E-17	1.13E-16	4.66E-17
Chromium (Total)	4.00E-18	3.07E-18	3.83E-18	7.73E-18	1.84E-17	2.30E-17	9.44E-18
Acetaldehyde	2.45E-17	3.43E-17	4.27E-17	1.85E-17	4.96E-17	6.18E-17	1.93E-17
Benzene	1.94E-17	1.74E-17	2.57E-17	1.46E-17	2.52E-17	3.72E-17	1.53E-17
Benzyl chloride	2.35E-15	2.11E-15	3.11E-15	1.77E-15	3.06E-15	4.50E-15	1.85E-15
di-(2-Ethylhexyl) phthalate	1.70E-10	2.16E-10	3.17E-10	1.28E-10	3.12E-10	4.59E-10	1.34E-10
Bromoform	1.72E-20	1.55E-20	2.28E-20	1.30E-20	2.24E-20	3.30E-20	1.36E-20
Chloroform	8.14E-19	1.14E-18	1.42E-18	6.15E-19	1.65E-18	2.06E-18	6.41E-19
Ethyl Chloride	4.71E-20	4.24E-20	6.24E-20	3.56E-20	6.13E-20	9.03E-20	3.71E-20
Ethylene Dibromide	1.56E-18	1.41E-18	2.07E-18	1.18E-18	2.04E-18	3.00E-18	1.23E-18
Formaldehyde	6.03E-15	8.44E-15	1.05E-14	4.55E-15	1.22E-14	1.52E-14	4.75E-15
Isophorone	3.38E-14	3.05E-14	4.48E-14	2.55E-14	4.41E-14	6.49E-14	2.66E-14
Methyl Hydrazine	4.31E-13	3.88E-13	5.72E-13	3.26E-13	5.62E-13	8.27E-13	3.40E-13
Dichloromethane	2.02E-18	1.82E-18	2.68E-18	1.52E-18	2.63E-18	3.87E-18	1.59E-18
Benzo(a)pyrene	2.04E-13	1.41E-13	2.08E-13	3.94E-13	8.49E-13	1.25E-12	4.81E-13
Benzo(b)fluoranthene	8.30E-14	4.55E-14	6.71E-14	1.60E-13	2.73E-13	4.03E-13	1.96E-13
Chrysene	4.87E-13	3.15E-13	4.63E-13	9.42E-13	1.89E-12	2.78E-12	1.15E-12
Indeno(1,2,3-cd)pyrene	1.10E-13	7.64E-14	1.12E-13	2.12E-13	4.59E-13	6.74E-13	2.59E-13
TCDD, 2,3,7,8-	5.53E-16	2.92E-16	4.30E-16	1.07E-15	1.75E-15	2.58E-15	1.31E-15
benz(a)anthracene	1.28E-13	8.54E-14	1.26E-13	2.47E-13	5.13E-13	7.55E-13	3.02E-13
benzo(k)fluoranthene	2.70E-13	1.88E-13	2.76E-13	5.22E-13	1.13E-12	1.66E-12	6.37E-13
dibenzo(a,h)anthracene	1.16E-14	8.09E-15	1.19E-14	2.24E-14	4.86E-14	7.13E-14	2.74E-14
Quinoline	1.30E-13	9.03E-14	1.33E-13	2.52E-13	5.42E-13	7.97E-13	3.07E-13

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
COPC	Adult	Adult	Adult	Adult	Adult	Composite
Arsenic	1.23E-19	8.25E-18	4.29E-18	1.77E-18	8.18E-17	1.20E-16
Beryllium	3.33E-17	1.44E-15	7.47E-16	3.09E-16	2.22E-14	2.76E-14

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
Cadmium	1.21E-18	5.23E-17	2.72E-17	1.13E-17	8.25E-16	1.03E-15
Chromium (Total)	2.46E-19	1.06E-17	5.52E-18	2.28E-18	1.64E-16	2.04E-16
Acetaldehyde	4.83E-19	2.20E-17	1.30E-17	7.85E-18	2.98E-16	3.71E-16
Benzene	2.46E-19	1.75E-17	1.03E-17	6.21E-18	1.52E-16	2.23E-16
Benzyl chloride	2.98E-17	2.12E-15	1.24E-15	7.53E-16	1.84E-14	2.70E-14
di-(2-Ethylhexyl) phthalate	3.04E-12	1.53E-10	8.98E-11	5.44E-11	1.87E-09	2.75E-09
Bromoform	2.18E-22	1.55E-20	9.11E-21	5.52E-21	1.35E-19	1.98E-19
Chloroform	1.61E-20	7.34E-19	4.31E-19	2.61E-19	9.91E-18	1.24E-17
Ethyl Chloride	5.98E-22	4.24E-20	2.49E-20	1.51E-20	3.69E-19	5.42E-19
Ethylene Dibromide	1.99E-20	1.41E-18	8.29E-19	5.02E-19	1.22E-17	1.80E-17
Formaldehyde	1.19E-16	5.43E-15	3.19E-15	1.93E-15	7.34E-14	9.15E-14
Isophorone	4.29E-16	3.05E-14	1.79E-14	1.08E-14	2.65E-13	3.90E-13
Methyl Hydrazine	5.47E-15	3.88E-13	2.28E-13	1.38E-13	3.38E-12	4.97E-12
Dichloromethane	2.56E-20	1.82E-18	1.07E-18	6.48E-19	1.58E-17	2.33E-17
Benzo(a)pyrene	1.13E-14	5.41E-13	2.81E-13	1.16E-13	7.54E-12	1.11E-11
Benzo(b)fluoranthene	3.65E-15	2.20E-13	1.14E-13	4.73E-14	2.43E-12	3.57E-12
Chrysene	2.52E-14	1.29E-12	6.73E-13	2.78E-13	1.68E-11	2.47E-11
Indeno(1,2,3-cd)pyrene	6.12E-15	2.91E-13	1.51E-13	6.25E-14	4.07E-12	5.98E-12
TCDD, 2,3,7,8-	2.34E-17	1.47E-15	7.64E-16	3.16E-16	1.56E-14	2.29E-14
benz(a)anthracene	6.85E-15	3.39E-13	1.76E-13	7.30E-14	4.55E-12	6.70E-12
benzo(k)fluoranthene	1.51E-14	7.16E-13	3.72E-13	1.54E-13	1.00E-11	1.47E-11
dibenzo(a,h)anthracene	6.48E-16	3.08E-14	1.60E-14	6.62E-15	4.31E-13	6.33E-13
Quinoline	7.24E-15	3.45E-13	1.80E-13	7.43E-14	4.81E-12	7.07E-12

Table B.1-2
Calculated Vegetation Ingestion Dose (mg/kg-d) – Scenario OS3

(mg/kg-d)	Receptor 1	Receptor 2		Receptor 3	Receptor 4		Receptor 5
	Industrial Worker	Resident		School	Resident		School
COPC	Adult	Adult	Composite	Adult	Adult	Composite	Adult
Arsenic	0	6.34E-11	6.91E-11	0	3.86E-10	4.21E-10	0
Beryllium	0	7.90E-12	8.61E-12	0	4.81E-11	5.24E-11	0
Cadmium	0	6.27E-12	6.83E-12	0	3.82E-11	4.16E-11	0
Chromium (Total)	0	2.30E-10	2.50E-10	0	1.40E-09	1.52E-09	0
Acetaldehyde	0	6.09E-14	6.64E-14	0	8.81E-14	9.61E-14	0
Benzene	0	9.79E-15	1.19E-14	0	1.39E-14	1.69E-14	0
Benzyl chloride	0	8.23E-13	1.01E-12	0	1.19E-12	1.46E-12	0
di-(2-Ethylhexyl) phthalate	0	5.41E-09	5.93E-09	0	6.69E-09	7.33E-09	0
Bromoform	0	5.47E-18	6.71E-18	0	7.90E-18	9.69E-18	0
Chloroform	0	7.08E-16	7.71E-16	0	1.01E-15	1.10E-15	0
Ethyl Chloride	0	2.55E-16	2.85E-16	0	3.24E-16	3.63E-16	0
Ethylene Dibromide	0	8.12E-16	9.95E-16	0	1.17E-15	1.44E-15	0
Formaldehyde	0	1.50E-11	1.64E-11	0	2.17E-11	2.37E-11	0
Isophorone	0	2.72E-11	3.34E-11	0	3.94E-11	4.83E-11	0
Methyl Hydrazine	0	1.23E-08	1.52E-08	0	1.79E-08	2.19E-08	0
Dichloromethane	0	2.73E-15	3.34E-15	0	3.93E-15	4.81E-15	0
Benzo(a)pyrene	0	1.08E-13	1.24E-13	0	6.55E-13	7.47E-13	0
Benzo(b)fluoranthene	0	9.85E-14	1.12E-13	0	5.96E-13	6.77E-13	0
Chrysene	0	3.98E-13	4.59E-13	0	2.40E-12	2.77E-12	0
Indeno(1,2,3-cd)pyrene	0	4.12E-14	4.62E-14	0	2.50E-13	2.80E-13	0
TCDD, 2,3,7,8-	0	7.12E-16	7.94E-16	0	4.32E-15	4.82E-15	0
benz(a)anthracene	0	9.52E-14	1.10E-13	0	5.75E-13	6.61E-13	0
benzo(k)fluoranthene	0	1.36E-13	1.55E-13	0	8.23E-13	9.37E-13	0
dibenzo(a,h)anthracene	0	4.58E-15	5.15E-15	0	2.77E-14	3.12E-14	0
Quinoline	0	6.40E-12	7.60E-12	0	3.84E-11	4.56E-11	0

Note: Human Receptors at location 1, 3, 5, 6, 7, 8, 9 are assumed to consume no vegetation from each respective location

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
COPC	Adult	Adult	Adult	Adult	Adult	Composite
Arsenic	0	0	0	0	2.68E-09	2.92E-09
Beryllium	0	0	0	0	3.34E-10	3.64E-10
Cadmium	0	0	0	0	2.73E-10	2.98E-10
Chromium (Total)	0	0	0	0	9.70E-09	1.06E-08
Acetaldehyde	0	0	0	0	5.29E-13	5.77E-13
Benzene	0	0	0	0	7.89E-14	9.65E-14
Benzyl chloride	0	0	0	0	7.08E-12	8.69E-12
di-(2-Ethylhexyl) phthalate	0	0	0	0	1.63E-08	1.80E-08
Bromoform	0	0	0	0	4.72E-17	5.79E-17
Chloroform	0	0	0	0	5.82E-15	6.34E-15
Ethyl Chloride	0	0	0	0	1.00E-15	1.16E-15

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
Ethylene Dibromide	0	0	0	0	7.02E-15	8.61E-15
Formaldehyde	0	0	0	0	1.30E-10	1.42E-10
Isophorone	0	0	0	0	2.36E-10	2.90E-10
Methyl Hydrazine	0	0	0	0	1.07E-07	1.32E-07
Dichloromethane	0	0	0	0	2.32E-14	2.84E-14
Benzo(a)pyrene	0	0	0	0	5.20E-12	5.96E-12
Benzo(b)fluoranthene	0	0	0	0	4.63E-12	5.29E-12
Chrysene	0	0	0	0	1.96E-11	2.26E-11
Indeno(1,2,3-cd)pyrene	0	0	0	0	1.89E-12	2.13E-12
TCDD, 2,3,7,8-	0	0	0	0	3.18E-14	3.56E-14
benz(a)anthracene	0	0	0	0	4.68E-12	5.41E-12
benzo(k)fluoranthene	0	0	0	0	6.50E-12	7.44E-12
dibenzo(a,h)anthracene	0	0	0	0	2.12E-13	2.39E-13
Quinoline	0	0	0	0	3.41E-10	4.04E-10

Note: Human Receptors at location 1, 3, 5, 6, 7, 8, 9 are assumed to consume no vegetation from each respective location

Table B.1-3
Calculated Beef Ingestion Dose (mg/kg-d) – Scenario OS3

(mg/kg-d)	Receptor 1	Receptor 2		Receptor 3	Receptor 4		Receptor 5
	Industrial Worker	Resident		School	Resident		School
COPC	Adult	Adult	Composite	Adult	Adult	Composite	Adult
Arsenic	0	5.07E-11	5.39E-11	0	3.09E-10	3.28E-10	0
Beryllium	0	3.16E-12	3.36E-12	0	1.93E-11	2.04E-11	0
Cadmium	0	3.01E-13	3.19E-13	0	1.83E-12	1.94E-12	0
Chromium (Total)	0	5.06E-10	5.37E-10	0	3.08E-09	3.27E-09	0
Acetaldehyde	0	1.18E-17	1.25E-17	0	1.70E-17	1.80E-17	0
Benzene	0	3.81E-16	4.28E-16	0	5.09E-16	5.74E-16	0
Benzyl chloride	0	2.66E-14	3.11E-14	0	3.78E-14	4.43E-14	0
di-(2-Ethylhexyl) phthalate	0	1.20E-08	1.28E-08	0	1.49E-08	1.60E-08	0
Bromoform	0	1.90E-19	2.23E-19	0	2.71E-19	3.19E-19	0
Chloroform	0	2.08E-17	2.21E-17	0	2.81E-17	2.99E-17	0
Ethyl Chloride	0	7.10E-21	7.56E-21	0	8.74E-21	9.32E-21	0
Ethylene Dibromide	0	2.19E-21	2.58E-21	0	3.14E-21	3.70E-21	0
Formaldehyde	0	1.17E-14	1.24E-14	0	1.69E-14	1.79E-14	0
Isophorone	0	3.07E-13	3.61E-13	0	4.41E-13	5.20E-13	0
Methyl Hydrazine	0	1.72E-16	2.03E-16	0	2.48E-16	2.94E-16	0
Dichloromethane	0	1.88E-17	2.17E-17	0	2.63E-17	3.05E-17	0
Benzo(a)pyrene	0	1.17E-12	1.29E-12	0	7.09E-12	7.80E-12	0
Benzo(b)fluoranthene	0	9.20E-13	9.90E-13	0	5.59E-12	6.02E-12	0
Chrysene	0	3.41E-12	3.74E-12	0	2.07E-11	2.27E-11	0
Indeno(1,2,3-cd)pyrene	0	4.60E-13	5.05E-13	0	2.79E-12	3.06E-12	0
TCDD, 2,3,7,8-	0	6.27E-15	6.71E-15	0	3.81E-14	4.08E-14	0
benz(a)anthracene	0	8.42E-13	9.26E-13	0	5.11E-12	5.61E-12	0
benzo(k)fluoranthene	0	1.45E-12	1.59E-12	0	8.79E-12	9.66E-12	0
dibenzo(a,h)anthracene	0	5.22E-14	5.74E-14	0	3.17E-13	3.48E-13	0
Quinoline	0	7.33E-16	8.60E-16	0	4.41E-15	5.17E-15	0

Note: Human Receptors at location 1, 3, 5, 6, 7, 8, 9 are assumed to consume no beef from each respective location

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
COPC	Adult	Adult	Adult	Adult	Adult	Composite
Arsenic	0	0	0	0	2.14E-09	2.27E-09
Beryllium	0	0	0	0	1.33E-10	1.42E-10
Cadmium	0	0	0	0	1.31E-11	1.39E-11
Chromium (Total)	0	0	0	0	2.13E-08	2.27E-08
Acetaldehyde	0	0	0	0	1.01E-16	1.07E-16
Benzene	0	0	0	0	2.18E-15	2.51E-15
Benzyl chloride	0	0	0	0	2.13E-13	2.51E-13
di-(2-Ethylhexyl) phthalate	0	0	0	0	3.77E-08	4.09E-08
Bromoform	0	0	0	0	1.56E-18	1.85E-18
Chloroform	0	0	0	0	1.29E-16	1.37E-16
Ethyl Chloride	0	0	0	0	2.04E-20	2.19E-20
Ethylene Dibromide	0	0	0	0	1.83E-20	2.16E-20

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
Formaldehyde	0	0	0	0	1.00E-13	1.06E-13
Isophorone	0	0	0	0	2.59E-12	3.06E-12
Methyl Hydrazine	0	0	0	0	1.49E-15	1.76E-15
Dichloromethane	0	0	0	0	1.39E-16	1.63E-16
Benzo(a)pyrene	0	0	0	0	5.36E-11	5.93E-11
Benzo(b)fluoranthene	0	0	0	0	4.01E-11	4.33E-11
Chrysene	0	0	0	0	1.55E-10	1.71E-10
Indeno(1,2,3-cd)pyrene	0	0	0	0	2.10E-11	2.31E-11
TCDD, 2,3,7,8-	0	0	0	0	2.70E-13	2.90E-13
benz(a)anthracene	0	0	0	0	3.85E-11	4.26E-11
benzo(k)fluoranthene	0	0	0	0	6.64E-11	7.35E-11
dibenzo(a,h)anthracene	0	0	0	0	2.38E-12	2.63E-12
Quinoline	0	0	0	0	3.86E-14	4.54E-14

Note: Human Receptors at location 1, 3, 5, 6, 7, 8, 9 are assumed to consume no beef from each respective location

Table B.1-4
Calculated Milk Ingestion Dose (mg/kg-d) – Scenario OS3

(mg/kg-d)	Receptor 1	Receptor 2		Receptor 3	Receptor 4		Receptor 5
	Industrial Worker	Resident		School	Resident		School
COPC	Adult	Adult	Composite	Adult	Adult	Composite	Adult
Arsenic	0	2.29E-12	3.38E-12	0	1.40E-11	2.06E-11	0
Beryllium	0	4.28E-15	6.31E-15	0	2.61E-14	3.84E-14	0
Cadmium	0	2.45E-14	3.62E-14	0	1.49E-13	2.20E-13	0
Chromium (Total)	0	2.08E-10	3.06E-10	0	1.26E-09	1.86E-09	0
Acetaldehyde	0	4.22E-18	6.22E-18	0	6.10E-18	8.99E-18	0
Benzene	0	1.29E-16	2.14E-16	0	1.73E-16	2.89E-16	0
Benzyl chloride	0	9.44E-15	1.69E-14	0	1.34E-14	2.41E-14	0
di-(2-Ethylhexyl) phthalate	0	3.81E-09	5.69E-09	0	4.71E-09	7.06E-09	0
Bromoform	0	7.03E-20	1.27E-19	0	1.01E-19	1.82E-19	0
Chloroform	0	7.13E-18	1.05E-17	0	9.70E-18	1.43E-17	0
Ethyl Chloride	0	3.47E-21	5.15E-21	0	4.27E-21	6.35E-21	0
Ethylene Dibromide	0	1.21E-21	2.18E-21	0	1.73E-21	3.13E-21	0
Formaldehyde	0	4.16E-15	6.14E-15	0	6.00E-15	8.85E-15	0
Isophorone	0	1.10E-13	2.00E-13	0	1.59E-13	2.88E-13	0
Methyl Hydrazine	0	9.37E-17	1.71E-16	0	1.36E-16	2.47E-16	0
Dichloromethane	0	6.43E-18	1.13E-17	0	9.02E-18	1.59E-17	0
Benzo(a)pyrene	0	3.34E-13	5.22E-13	0	2.03E-12	3.17E-12	0
Benzo(b)fluoranthene	0	2.83E-13	4.26E-13	0	1.72E-12	2.59E-12	0
Chrysene	0	1.00E-12	1.56E-12	0	6.10E-12	9.48E-12	0
Indeno(1,2,3-cd)pyrene	0	1.34E-13	2.09E-13	0	8.15E-13	1.27E-12	0
TCDD, 2,3,7,8-	0	1.95E-15	2.91E-15	0	1.19E-14	1.77E-14	0
benz(a)anthracene	0	2.46E-13	3.85E-13	0	1.49E-12	2.33E-12	0
benzo(k)fluoranthene	0	4.25E-13	6.64E-13	0	2.58E-12	4.03E-12	0
dibenzo(a,h)anthracene	0	1.50E-14	2.33E-14	0	9.09E-14	1.41E-13	0
Quinoline	0	3.56E-16	6.39E-16	0	2.14E-15	3.84E-15	0

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
COPC	Adult	Adult	Adult	Adult	Adult	Composite
Arsenic	0	0	0	0	9.68E-11	1.43E-10
Beryllium	0	0	0	0	1.81E-13	2.67E-13
Cadmium	0	0	0	0	1.07E-12	1.58E-12
Chromium (Total)	0	0	0	0	8.77E-09	1.29E-08
Acetaldehyde	0	0	0	0	3.64E-17	5.36E-17
Benzene	0	0	0	0	7.55E-16	1.32E-15
Benzyl chloride	0	0	0	0	7.62E-14	1.38E-13
di-(2-Ethylhexyl) phthalate	0	0	0	0	1.16E-08	1.78E-08
Bromoform	0	0	0	0	5.83E-19	1.06E-18
Chloroform	0	0	0	0	4.53E-17	6.68E-17
Ethyl Chloride	0	0	0	0	1.00E-20	1.51E-20
Ethylene Dibromide	0	0	0	0	1.01E-20	1.84E-20
Formaldehyde	0	0	0	0	3.56E-14	5.25E-14

(mg/kg-d)	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
Isophorone	0	0	0	0	9.36E-13	1.70E-12
Methyl Hydrazine	0	0	0	0	8.14E-16	1.49E-15
Dichloromethane	0	0	0	0	4.83E-17	8.70E-17
Benzo(a)pyrene	0	0	0	0	1.51E-11	2.38E-11
Benzo(b)fluoranthene	0	0	0	0	1.22E-11	1.85E-11
Chrysene	0	0	0	0	4.50E-11	7.08E-11
Indeno(1,2,3-cd)pyrene	0	0	0	0	6.02E-12	9.44E-12
TCDD, 2,3,7,8-	0	0	0	0	8.36E-14	1.25E-13
benz(a)anthracene	0	0	0	0	1.11E-11	1.75E-11
benzo(k)fluoranthene	0	0	0	0	1.92E-11	3.03E-11
dibenzo(a,h)anthracene	0	0	0	0	6.72E-13	1.06E-12
Quinoline	0	0	0	0	1.87E-14	3.37E-14

B.2 CALCULATED RISKS

Table B.2-1
Calculated Risk From Inhalation – Scenario OS3

	Receptor 1	Receptor 2		Receptor 3	Receptor 4		Receptor 5
	Industrial Worker	Resident		School	Resident		School
COPC	Adult	Adult	Composite	Adult	Adult	Composite	Adult
Arsenic	$1.56x10^{-9}$	$8.04x10^{-9}$	$8.60x10^{-9}$	3.17E-09	9.70E-09	1.04E-08	3.49E-09
Beryllium	$1.31x10^{-10}$	$6.37x10^{-10}$	$6.81x10^{-10}$	2.78E-10	7.54E-10	8.06E-10	2.79E-10
Cadmium	$6.07x10^{-11}$	$3.19x10^{-10}$	$3.41x10^{-10}$	1.22E-10	3.88E-10	4.15E-10	1.38E-10
Chromium (Total)	$1.22x10^{-7}$	$6.07x10^{-7}$	$6.49x10^{-7}$	3.70E-08	1.05E-07	1.12E-07	3.84E-08
Acetaldehyde	$5.41x10^{-12}$	$2.89x10^{-11}$	$3.09x10^{-11}$	1.05E-11	3.54E-11	3.78E-11	1.25E-11
Benzene	$4.33x10^{-11}$	$2.31x10^{-10}$	$2.47x10^{-10}$	8.41E-11	2.83E-10	3.03E-10	1.00E-10
Benzyl chloride	$1.47x10^{-10}$	$7.84x10^{-10}$	$8.38x10^{-10}$	2.85E-10	9.59E-10	1.03E-09	3.39E-10
di-(2-Ethylhexyl) phthalate	$1.26x10^{-12}$	$6.73x10^{-12}$	$7.20x10^{-12}$	2.45E-12	8.24E-12	8.81E-12	2.92E-12
Bromoform	$1.85x10^{-13}$	$9.89x10^{-13}$	$1.06x10^{-12}$	3.60E-13	1.21E-12	1.29E-12	4.28E-13
Chloroform	$5.85x10^{-12}$	$3.13x10^{-11}$	$3.35x10^{-11}$	1.14E-11	3.83E-11	4.09E-11	1.35E-11
Ethyl Chloride	$1.50x10^{-13}$	$8.02x10^{-13}$	$8.58x10^{-13}$	2.92E-13	9.82E-13	1.05E-12	3.47E-13
Ethylene Dibromide	$2.96x10^{-12}$	$1.58x10^{-11}$	$1.69x10^{-11}$	5.75E-12	1.94E-11	2.07E-11	6.85E-12
Formaldehyde	$1.36x10^{-11}$	$7.27x10^{-11}$	$7.78x10^{-11}$	2.65E-11	8.90E-11	9.52E-11	3.15E-11
Isophorone	$6.79x10^{-13}$	$3.63x10^{-12}$	$3.88x10^{-12}$	1.32E-12	4.44E-12	4.75E-12	1.57E-12
Methyl Hydrazine	$2.31x10^{-10}$	$1.23x10^{-9}$	$1.32x10^{-9}$	4.48E-10	1.51E-09	1.61E-09	5.33E-10
Dichloromethane	$5.90x10^{-13}$	$3.15x10^{-12}$	$3.37x10^{-12}$	1.15E-12	3.86E-12	4.13E-12	1.36E-12
Benzo(a)pyrene	$5.77x10^{-13}$	$3.10x10^{-12}$	$3.32x10^{-12}$	1.07E-12	3.59E-12	3.83E-12	1.26E-12
Benzo(b)fluoranthene	$6.21x10^{-14}$	$3.34x10^{-13}$	$3.57x10^{-13}$	1.15E-13	3.86E-13	4.13E-13	1.36E-13
Chrysene	$1.69x10^{-15}$	$9.07x10^{-15}$	$9.70x10^{-15}$	3.12E-15	1.05E-14	1.12E-14	3.70E-15
Indeno(1,2,3-cd)pyrene	$3.06x10^{-14}$	$1.65x10^{-13}$	$1.76x10^{-13}$	5.67E-14	1.90E-13	2.03E-13	6.71E-14
TCDD, 2,3,7,8-	$1.43x10^{-10}$	$7.40x10^{-10}$	$7.91x10^{-10}$	2.76E-10	8.46E-10	9.05E-10	3.04E-10
benz(a)anthracene	$3.99x10^{-14}$	$2.15x10^{-13}$	$2.30x10^{-13}$	7.40E-14	2.48E-13	2.65E-13	8.75E-14
benzo(k)fluoranthene	$7.54x10^{-15}$	$4.06x10^{-14}$	$4.34x10^{-14}$	1.40E-14	4.69E-14	5.01E-14	1.65E-14
dibenzo(a,h)anthracene	$3.24x10^{-14}$	$1.74x10^{-13}$	$1.86x10^{-13}$	6.00E-14	2.01E-13	2.15E-13	7.10E-14
Quinoline	$1.51x10^{-13}$	$8.14x10^{-13}$	$8.71x10^{-13}$	2.80E-13	9.41E-13	1.01E-12	3.32E-13

	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
COPC	Adult	Adult	Adult	Adult	Adult	Composite
Arsenic	7.85E-09	3.42E-09	2.57E-09	1.56E-09	1.26E-07	1.35E-07
Beryllium	6.10E-10	2.51E-10	1.90E-10	1.31E-10	2.19E-08	2.34E-08
Cadmium	3.14E-10	1.39E-10	1.04E-10	6.07E-11	3.17E-09	3.39E-09
Chromium (Total)	8.47E-08	3.57E-08	2.70E-08	1.77E-08	2.41E-06	2.58E-06
Acetaldehyde	2.87E-11	1.33E-11	9.81E-12	5.41E-12	7.79E-11	8.33E-11
Benzene	2.30E-10	1.06E-10	7.85E-11	4.33E-11	6.23E-10	6.66E-10
Benzyl chloride	7.79E-10	3.60E-10	2.66E-10	1.47E-10	2.11E-09	2.26E-09
di-(2-Ethylhexyl) phthalate	6.69E-12	3.09E-12	2.28E-12	1.26E-12	1.81E-11	1.94E-11
Bromoform	9.82E-13	4.54E-13	3.36E-13	1.85E-13	2.66E-12	2.85E-12

	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
Chloroform	3.11E-11	1.44E-11	1.06E-11	5.85E-12	8.42E-11	9.01E-11
Ethyl Chloride	7.97E-13	3.68E-13	2.72E-13	1.50E-13	2.16E-12	2.31E-12
Ethylene Dibromide	1.57E-11	7.26E-12	5.37E-12	2.96E-12	4.26E-11	4.55E-11
Formaldehyde	7.22E-11	3.34E-11	2.47E-11	1.36E-11	1.96E-10	2.09E-10
Isophorone	3.61E-12	1.67E-12	1.23E-12	6.79E-13	9.77E-12	1.05E-11
Methyl Hydrazine	1.22E-09	5.66E-10	4.18E-10	2.31E-10	3.32E-09	3.55E-09
Dichloromethane	3.13E-12	1.45E-12	1.07E-12	5.90E-13	8.49E-12	9.08E-12
Benzo(a)pyrene	2.90E-12	1.31E-12	9.78E-13	5.45E-13	7.92E-12	8.47E-12
Benzo(b)fluoranthene	3.13E-13	1.41E-13	1.05E-13	5.87E-14	8.53E-13	9.12E-13
Chrysene	8.48E-15	3.84E-15	2.86E-15	1.59E-15	2.32E-14	2.48E-14
Indeno(1,2,3-cd)pyrene	1.54E-13	6.97E-14	5.19E-14	2.89E-14	4.20E-13	4.50E-13
TCDD, 2,3,7,8-	6.85E-10	2.99E-10	2.24E-10	1.36E-10	1.10E-08	1.17E-08
benz(a)anthracene	2.01E-13	9.09E-14	6.77E-14	3.77E-14	5.48E-13	5.86E-13
benzo(k)fluoranthene	3.80E-14	1.72E-14	1.28E-14	7.12E-15	1.04E-13	1.11E-13
dibenzo(a,h)anthracene	1.63E-13	7.38E-14	5.49E-14	3.06E-14	4.45E-13	4.76E-13
Quinoline	7.62E-13	3.45E-13	2.57E-13	1.43E-13	2.08E-12	2.22E-12

Table B.2-2
Calculated Total Risk From Ingestion – Scenario OS3

	Receptor 1	Receptor 2		Receptor 3	Receptor 4		Receptor 5
	Industrial Worker	Resident		School	Resident		School
COPC	Adult	Adult	Composite	Adult	Adult	Composite	Adult
Arsenic	4.66E-18	1.75E-10	1.90E-10	9.01E-18	1.06E-09	1.15E-09	1.10E-17
Beryllium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chromium (Total)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Acetaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Benzene	1.07E-18	5.67E-16	6.90E-16	8.05E-19	8.05E-16	9.81E-16	8.39E-19
Benzyl chloride	3.99E-16	1.46E-13	1.80E-13	3.01E-16	2.11E-13	2.60E-13	3.14E-16
di-(2-Ethylhexyl) phthalate	2.37E-12	3.00E-10	3.47E-10	1.79E-12	3.72E-10	4.31E-10	1.87E-12
Bromoform	1.36E-22	4.54E-20	5.59E-20	1.03E-22	6.55E-20	8.08E-20	1.07E-22
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Ethyl Chloride	1.37E-22	7.39E-19	8.27E-19	1.03E-22	9.39E-19	1.05E-18	1.08E-22
Ethylene Dibromide	3.13E-18	1.63E-15	1.99E-15	2.36E-18	2.35E-15	2.88E-15	2.46E-18
Formaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Isophorone	3.21E-17	2.63E-14	3.23E-14	2.43E-17	3.80E-14	4.67E-14	2.53E-17
Methyl Hydrazine	4.74E-13	1.36E-08	1.67E-08	3.58E-13	1.96E-08	2.41E-08	3.74E-13
Dichloromethane	1.51E-20	2.07E-17	2.53E-17	1.14E-20	2.97E-17	3.64E-17	1.19E-20
Benzo(a)pyrene	1.49E-12	1.28E-11	1.56E-11	2.88E-12	7.75E-11	9.46E-11	3.51E-12
Benzo(b)fluoranthene	6.06E-14	9.83E-13	1.16E-12	1.17E-13	5.97E-12	7.07E-12	1.43E-13
Chrysene	3.56E-15	3.75E-14	4.55E-14	6.88E-15	2.27E-13	2.75E-13	8.40E-15
Indeno(1,2,3-cd)pyrene	8.00E-14	5.20E-13	6.37E-13	1.55E-13	3.15E-12	3.85E-12	1.89E-13
TCDD, 2,3,7,8-	8.30E-11	1.38E-09	1.63E-09	1.61E-10	8.42E-09	9.89E-09	1.96E-10
benz(a)anthracene	9.33E-14	9.26E-13	1.13E-12	1.80E-13	5.61E-12	6.84E-12	2.20E-13
benzo(k)fluoranthene	1.97E-14	1.60E-13	1.96E-13	3.81E-14	9.72E-13	1.19E-12	4.65E-14
dibenzo(a,h)anthracene	8.47E-14	5.83E-13	7.13E-13	1.64E-13	3.53E-12	4.32E-12	2.00E-13
Quinoline	3.91E-13	1.95E-11	2.32E-11	7.55E-13	1.17E-10	1.39E-10	9.22E-13

	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
COPC	Adult	Adult	Adult	Adult	Adult	Composite
Arsenic	1.84E-19	1.24E-17	6.43E-18	2.66E-18	7.37E-09	8.00E-09
Beryllium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chromium (Total)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Acetaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Benzene	1.35E-20	9.60E-19	5.64E-19	3.42E-19	4.51E-15	5.53E-15
Benzyl chloride	5.07E-18	3.60E-16	2.11E-16	1.28E-16	1.26E-12	1.55E-12
di-(2-Ethylhexyl) phthalate	4.26E-14	2.14E-12	1.26E-12	7.62E-13	9.45E-10	1.11E-09
Bromoform	1.72E-24	1.22E-22	7.20E-23	4.36E-23	3.91E-19	4.82E-19
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Ethyl Chloride	1.73E-24	1.23E-22	7.23E-23	4.38E-23	2.91E-18	3.36E-18
Ethylene Dibromide	3.97E-20	2.82E-18	1.66E-18	1.00E-18	1.41E-14	1.73E-14
Formaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00

	Receptor 6	Receptor 7	Receptor 8	Receptor 9	Max POI	
	Hospital	School	School	Resident	Hypothetical Residence	
Isophorone	4.08E-19	2.89E-17	1.70E-17	1.03E-17	2.28E-13	2.80E-13
Methyl Hydrazine	6.02E-15	4.27E-13	2.51E-13	1.52E-13	1.18E-07	1.45E-07
Dichloromethane	1.92E-22	1.36E-20	8.02E-21	4.86E-21	1.76E-16	2.15E-16
Benzo(a)pyrene	8.28E-14	3.95E-12	2.05E-12	8.49E-13	5.94E-10	7.31E-10
Benzo(b)fluoranthene	2.66E-15	1.61E-13	8.36E-14	3.46E-14	4.34E-11	5.16E-11
Chrysene	1.84E-16	9.44E-15	4.91E-15	2.03E-15	1.73E-12	2.11E-12
Indeno(1,2,3-cd)pyrene	4.47E-15	2.12E-13	1.10E-13	4.56E-14	2.40E-11	2.97E-11
TCDD, 2,3,7,8-	3.51E-12	2.20E-10	1.15E-10	4.74E-11	6.01E-08	7.10E-08
benz(a)anthracene	5.00E-15	2.48E-13	1.29E-13	5.33E-14	4.30E-11	5.27E-11
benzo(k)fluoranthene	1.10E-15	5.23E-14	2.72E-14	1.12E-14	7.45E-12	9.19E-12
dibenzo(a,h)anthracene	4.73E-15	2.25E-13	1.17E-13	4.83E-14	2.70E-11	3.33E-11
Quinoline	2.17E-14	1.04E-12	5.39E-13	2.23E-13	1.04E-09	1.23E-09

B.3 CALCULATED INCREMENTAL AIR CONCENTRATIONS

Table B.3-1
Calculated Incremental Air Concentrations (µg/m^3) – Scenario OS3

	Receptor Location									
Chemical	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**Max POI**
Arsenic	3.62E-06	1.86E-06	2.20E-06	2.25E-06	2.43E-06	2.60E-06	2.38E-06	1.79E-06	1.08E-06	2.93E-05
Beryllium	4.86E-07	2.66E-07	3.48E-07	3.14E-07	3.49E-07	3.63E-07	3.14E-07	2.38E-07	1.64E-07	9.14E-06
Cadmium	3.51E-07	1.77E-07	2.03E-07	2.15E-07	2.30E-07	2.49E-07	2.32E-07	1.73E-07	1.01E-07	1.76E-06
Chromium (Total)	1.37E-05	7.33E-06	9.25E-06	8.73E-06	9.59E-06	1.01E-05	8.92E-06	6.74E-06	4.42E-06	2.01E-04
Acetaldehyde	2.66E-05	1.31E-05	1.43E-05	1.61E-05	1.71E-05	1.86E-05	1.81E-05	1.34E-05	7.38E-06	3.54E-05
Benzene	6.06E-05	3.00E-05	3.27E-05	3.67E-05	3.89E-05	4.25E-05	4.13E-05	3.05E-05	1.68E-05	8.07E-05
Benzyl chloride	3.26E-05	1.61E-05	1.76E-05	1.98E-05	2.10E-05	2.29E-05	2.22E-05	1.64E-05	9.06E-06	4.35E-05
di-(2-Ethylhexyl) phthalate	3.40E-06	1.68E-06	1.84E-06	2.06E-06	2.19E-06	2.39E-06	2.32E-06	1.71E-06	9.45E-07	4.53E-06
Bromoform	1.82E-06	8.99E-07	9.81E-07	1.10E-06	1.17E-06	1.28E-06	1.24E-06	9.15E-07	5.05E-07	2.42E-06
Chloroform	2.75E-06	1.36E-06	1.48E-06	1.66E-06	1.77E-06	1.93E-06	1.87E-06	1.38E-06	7.64E-07	3.66E-06
Ethyl Chloride	1.96E-06	9.68E-07	1.06E-06	1.19E-06	1.26E-06	1.37E-06	1.33E-06	9.86E-07	5.44E-07	2.61E-06
Ethylene Dibromide	5.59E-08	2.77E-08	3.02E-08	3.39E-08	3.59E-08	3.93E-08	3.81E-08	2.82E-08	1.55E-08	7.45E-08
Formaldehyde	1.12E-05	5.53E-06	6.04E-06	6.77E-06	7.19E-06	7.85E-06	7.62E-06	5.63E-06	3.11E-06	1.49E-05
Isophorone	2.70E-05	1.34E-05	1.46E-05	1.64E-05	1.74E-05	1.90E-05	1.84E-05	1.36E-05	7.51E-06	3.60E-05
Methyl Hydrazine	7.92E-06	3.92E-06	4.28E-06	4.80E-06	5.09E-06	5.56E-06	5.40E-06	3.99E-06	2.20E-06	1.06E-05
Dichloromethane	1.35E-05	6.69E-06	7.30E-06	8.18E-06	8.69E-06	9.49E-06	9.21E-06	6.81E-06	3.75E-06	1.80E-05
Benzo(a)pyrene	2.84E-09	1.40E-09	1.54E-09	1.72E-09	1.82E-09	1.99E-09	1.89E-09	1.41E-09	7.83E-10	3.80E-09
Benzo(b)fluoranthene	3.05E-09	1.51E-09	1.66E-09	1.85E-09	1.96E-09	2.14E-09	2.03E-09	1.51E-09	8.44E-10	4.09E-09
Chrysene	8.29E-09	4.10E-09	4.49E-09	5.02E-09	5.32E-09	5.81E-09	5.52E-09	4.11E-09	2.29E-09	1.11E-08
Indeno(1,2,3-cd)pyrene	1.50E-09	7.45E-10	8.16E-10	9.12E-10	9.65E-10	1.06E-09	1.00E-09	7.46E-10	4.16E-10	2.02E-09
TCDD, 2,3,7,8-	3.17E-11	1.64E-11	1.93E-11	1.97E-11	2.13E-11	2.28E-11	2.09E-11	1.57E-11	9.52E-12	2.56E-10
benz(a)anthracene	1.96E-09	9.72E-10	1.06E-09	1.19E-09	1.26E-09	1.38E-09	1.31E-09	9.74E-10	5.42E-10	2.63E-09
benzo(k)fluoranthene	3.71E-09	1.84E-09	2.01E-09	2.25E-09	2.38E-09	2.60E-09	2.47E-09	1.84E-09	1.02E-09	4.97E-09
dibenzo(a,h)anthracene	1.59E-10	7.89E-11	8.63E-11	9.65E-11	1.02E-10	1.12E-10	1.06E-10	7.90E-11	4.40E-11	2.13E-10
Quinoline	1.81E-09	8.97E-10	9.82E-10	1.10E-09	1.16E-09	1.27E-09	1.21E-09	8.98E-10	5.00E-10	2.42E-09

ATTACHMENT C

Sample Calculation and Screening Results

ATTACHMENT C

This attachment includes a worked calculation for one chemical at one of the receptor locations. The equations in Attachment A are the basis for these calculations. Also included in this attachment are the screening calculations for the human health risk assessment.

C.1 SAMPLE CALCULATION

Following is a worked calculation for risks associated with arsenic exposure for an adult resident receptor at the maximum point of impingement for a hypothetical residence. The fixed values and assumptions used in the calculations are summarized in Table C.1-1.

Table C.1-1
Starting Values and Assumptions

	Parameter	Symbol	Value	Units	Source
Receptor Characteristics	Breathing Rate	R_{inh}	20	m^3/d	Richardson 1997
	Soil Ingestion Rate	$R_{ing\text{-}soil}$	80	mg/d	Richardson 1997
	Beef Ingestion Rate	$R_{ing\text{-}beef}$	182	g/d	Richardson 1997
	Milk Ingestion Rate	$R_{ing\text{-}milk}$	181	g/d	Richardson 1997
	Vegetation Ingestion Rate	$R_{ing\text{-}veg}$	339	g/d	Richardson 1997
	Body Weight	B_w	70	kg	Richardson 1997
	Fraction of soil and air from site	$F_{location}$	1.0	-	Assumed
	Fraction of meat, milk, and veg from site	$F_{location}$	0.05	-	Assumed
	Time at site		24	hours/day	Assumed
	Time at site		365	days/year	Assumed
	Duration of exposure		70	year	Assumed
Chemical Specific	Soil-water partition coefficient	Kd_s	29	cm^3/g	Burton 1997
	Diffusivity in air	D_a	0.077	cm^2/s	U.S. EPA Region 6 2005
	Fraction of air in the vapour phase	F_v	0	-	Burton 1997
	Henry's Law constant	H	0.77	$(atm\ m^3)/mol$	U.S. EPA Region 6 2005
	Air-to-plant biotransfer factor	B_v	n/a		-
	Soil-to-plant biotransfer factor (leafy)	$B_{r\ leafy}$	$6.30x10^{-3}$	(µg/g plant)/(µg/g soil)	U.S. EPA Region 6 2005
	Soil-to-plant biotransfer factor (forage)	$B_{r\ forage}$	$3.60x10^{-2}$		
	Biotransfer factor for beef	Ba_{beef}	$2.00x10^{-3}$	d/kg	Baes *et al.* 1984
	Biotransfer factor for milk	Ba_{milk}	$6.00x10^{-5}$	d/kg	U.S. EPA Region 6 2005
	Average daily emission rate from stack	Q	$6.25x10^{-4}$	g/s	Predicted
Location Specific	Source Particle Concentration (from stack)	$C_{p\text{-}source}$	$7.42x10^{-3}$	$(\mu g/m^3)/(g/s)$	Predicted
	Particle Dry Deposition	$C_{d\ dep\ p}$	$1.3x10^{-10}$	$(g/m^2\ yr)/(g/s)$	Predicted
	Particle Wet Deposition	$C_{w\ dep\ p}$	$4.97x10^{-10}$	$(g/m^2\ yr)/(g/s)$	Predicted
	Source Vapour Concentration	$C_{v\text{-}source}$	$7.45x10^{-3}$	$(\mu g/m^3)/(g/s)$	Predicted
	Vapour Dry Deposition	$C_{d\ dep\ v}$	$3.73x10^{-11}$	$(g/m^2\ yr)/(g/s)$	Predicted
	Vapour Wet Deposition	$C_{w\ dep\ v}$	$9.15x10^{-11}$	$(g/m^2\ yr)/(g/s)$	Predicted

Table C.1-1 (Cont'd)

Starting Values and Assumptions

	Parameter	Symbol	Value	Units	Source
Tox Data	Slope Factor – oral	SF_o	1.5	1/(mg/kg-d)	IRIS 2005
	Slope Factor – inhalation	SF_i	15.1	1/(mg/kg-d)	IRIS 2005

Note: - data not available

Table C.1-2 shows the calculations used to obtain the inhalation dose and risk due to inhalation.

Table C.1-2
Air Concentration, Inhalation Dose and Risk Calculation

Equation #	Parameter	Equation	Value	Units
(A-4)	C_{ap}	$Q \times C_{p-source}$	2.9×10^{-5}	µg/m^3
(A-3)	C_{av}	$Q \times C_{v-source}$	0	µg/m^3
(A-2)	C_a	$F_v \times C_{av} + (1 - F_v) \times C_{ap}$	2.9×10^{-5}	µg/m^3
(A-1)	$Dose_{inhalation}$	$\frac{R_{inh} C_a}{B_w} \times \frac{1}{1000}$	8.0×10^{-9}	mg/kg-d
(5-2)	$Risk_{inhalation}$	$Dose_{inhalation} \times SF_i$	1.26×10^{-7}	-

The soil concentrations are obtained from the equations shown in Table C.1-3. Default values for the parameters are provided in the equations in Attachment A.

Table C.1-3
Soil Concentration Calculation

Eqn #	Para-meter	Equation	Value	Units
(A-19)	ksv	$\left(\frac{3.1536\times10^{7}\times H}{z\times Kd_s\times R\times T\times BD}\right)\times\left(0.482\times u^{0.78}\times\left(\frac{\mu_a}{\rho_a\times D_a}\right)^{-0.67}\times\left(\sqrt{\frac{4\times A}{\pi}}\right)^{-0.11}\right)$	$7.4\text{x}10^{6}$ (forage), $3.7\text{x}10^{5}$ (tilled)	1/yr
(A-18)	ksr	$\frac{R}{\Theta_s\times z}\times\left(\frac{1}{1+\left(\frac{BD\times Kd_s}{\Theta_s}\right)}\right)$	0.27 (forage), 0.013 (tilled)	1/yr
(A-17)	SD	$a\times\left(WA_L\right)^{-b}$	0.44	-
(A-16)	Xe	$RF\times K\times LS\times C\times P\times\frac{907.18}{4047}$	0.67	kg/(m^2 yr)
(A-15)	kse	$\frac{0.1\times X_e\times SD\times ER}{BD\times z}\times\left(\frac{Kd_s\times BD}{\Theta_s+\left(Kd_s\times BD\right)}\right)$	0.06 (forage), 0.003 (tilled)	1/yr
(A-14)	ksl	$\frac{q}{\Theta_s\times z\times\left[1+\left(\frac{BD\times Kd_s}{\Theta_s}\right)\right]}$	0.11 (forage), 0.006 (tilled)	1/yr
(A-13)	ks	$ksl+kse+ksr+ksg+ksv$	$7.4\text{x}10^{6}$ (forage), $3.7\text{x}10^{5}$ (tilled)	1/yr
(A-11)	D_{wp}	$Q\times C_{w\,dep\,p}$	$9.7\text{x}10^{-6}$	g/(m^2 yr)
(A-10)	D_{dp}	$Q\times C_{d\,dep\,p}$	$2.6\text{x}10^{-6}$	g/(m^2 yr)
(A-9)	D_{wv}	$Q\times C_{w\,dep\,v}$	0	g/(m^2 yr)
(A-8)	D_{dv}	$Q\times C_{d\,dep\,v}$	0	g/(m^2 yr)
(A-7)	Ds	$\frac{100}{z\times BD}\times\left[F_v\left(D_{dv}+D_{wv}\right)+\left(1-F_v\right)\left(D_{dp}+D_{wp}\right)\right]$	$8.2\text{x}10^{-4}$ (forage), $4.1\text{x}10^{-5}$ (tilled)	mg/(kg yr)
(A-12)	Sc_{Tc}	$\frac{Ds\times\left(1-e^{(-ks\times Tc)}\right)}{ks}$	$1.1\text{x}10^{-10}$ (forage), $1.1\text{x}10^{-10}$ (tilled)	(mg/kg)
(A-6)	C_{soil}	$\frac{\left(\frac{Ds\times Tc-Sc_{TC}}{ks}\right)+\left(\frac{Sc_{TC}}{ks}\times\left[1-e^{(-ks(T_2-Tc)}\right]\right)}{\left(T_2-T_1\right)}$	$7.2\text{x}10^{-11}$ (forage), $7.2\text{x}10^{-11}$ (tilled)	mg/kg

The vegetation, beef and milk concentration calculations are shown in Tables C.1-4 and C.1-5. Parameters needed for the equations are given in Table C.1-1 or provided in the equations in Attachment A.

Table C.1-4
Vegetation Concentration Calculation

Eqn #	Para-meter	Equation	Value	Units
(A-23)	C_v	$F_v \times \frac{C_{av} \times Bv \times VG_{ag}}{\rho_s}$	0	mg/kg DW
(A-22)	C_d	$\frac{1000 \times (1 - F_v) \times (D_{dp} + (Fw \times D_{wp})) \times Rp \times [(1 - e^{(-kp \times Tp)})]}{Yp \times kp}$	1.1×10^{-5} (veg), 8.6×10^{-4} (forage), 2.5×10^{-4} (silage)	mg/kg DW
(A-21)	C_r	$C_{soil} \times Br$	4.5×10^{-13} (veg), 2.6×10^{-12} (forage), 2.6×10^{-12} (silage)	mg/kg DW
(A-20)	C_{veg}	$C_r + C_d + C_v$	1.1×10^{-5} (veg), 8.6×10^{-4} (forage), 2.5×10^{-4} (silage)	mg/kg DW

Table C.1-5
Beef and Milk Concentration Calculation

Eqn #	Para-meter	Equation	Value	Units
(A-24)	C_{beef}	$(F_i((Qp_f \times P_f) + (Qp_s \times P_s) + (Qp_g \times P_g)) + (Q_{soil} \times C_{soil})) \times BA_{beef}$	1.7×10^{-5}	mg/kg FW
(A-25)	C_{milk}	$(F_i((Qp_f \times P_f) + (Qp_s \times P_s) + (Qp_g \times P_g)) + (Q_{soil} \times C_{soil})) \times BA_{milk}$	7.5×10^{-7}	mg/kg FW

The final calculations shown in Table C.1-6 are for the ingestion dose and risk. The total risk to the receptor at location 6 is the sum of the inhalation risk (Table C.1-2) and the ingestion risk (Table C.1-6), as shown in Table C.1-7.

Table C.1-6
Ingestion Dose and Risk Calculation

Equation #	Parameter	Equation	Value	Units
(A-5)	$Dose_{soil}$	$\frac{R_{ing-soil}C_{soil}}{B_w} \times F_{location} \times \frac{1}{1000000}$	8.2×10^{-17}	mg/kg-d
(A-5)	$Dose_{veg}$	$\frac{R_{ing-veg}C_{veg}}{B_w} \times F_{location} \times \frac{1}{1000}$	2.7×10^{-9}	mg/kg-d
(A-5)	$Dose_{beef}$	$\frac{R_{ing-beef}C_{beef}}{B_w} \times F_{location} \times \frac{1}{1000}$	2.1×10^{-9}	mg/kg-d
(A-5)	$Dose_{milk}$	$\frac{R_{ing-milk}C_{milk}}{B_w} \times F_{location} \times \frac{1}{1000}$	9.7×10^{-11}	mg/kg-d
	$Dose_{ingestion}$	$Dose_{soil} + Dose_{veg} + Dose_{beef} + Dose_{milk}$	4.9×10^{-9}	mg/kg-d
(5-2)	$Risk_{ingestion}$	$Dose_{ingestion}\left(\frac{mg}{kg\,d}\right) \times SF_o\left(\frac{mg}{kg\,d}\right)^{-1}$	7.4×10^{-9}	-

Table C.1-7
Total Risk Calculation

Parameter	Equation	Value
$Risk_{total}$	$Risk_{inhalation} + Risk_{ingestion}$	1.3×10^{-7}

C.2 HUMAN SCREENING RESULTS

The procedure used to develop the list of COPCs followed the Chemical Selection Criteria outlined by the U.S. EPA in *Risk Assessment Guidance for Superfund* (U.S. EPA 1989*)*. In summary, the Selection Criteria specify that:

> *chemicals present at concentrations less than the detection limit, where the detection limit is sufficiently low, are considered not present at the site; all known or probable human carcinogens which are present must be evaluated for human health; chemicals which have the potential to bioaccumulate and are also persistent and toxic must be evaluated; for chemicals that have the potential to be degraded to other toxic chemicals, the breakdown products must be assessed; and where two or more chemicals that are similar in physical, chemical and biological properties and that have the same toxic end points are present (such as PAHs and dioxins and furans), it is acceptable to evaluate one representative contaminant to reduce the scope of the exercise. However, if this route is taken, modelling has to be conducted with the most toxic contaminant, using the highest concentration among the chemicals and the physical chemical properties of the most mobile chemical in the group.*

There are 6 major steps that were followed for screening COPC for human health.

1. Determination of the availability of toxicity data from standard referred agencies.
2. Determination of the potential to break down into toxic products.
3. Assessment for carcinogenicity.
4. Determination of a toxic potential for non-carcinogenic chemicals.
5. Assessment of ability of non-carcinogenic chemicals to be persistent or bioaccumulate.
6. Determination of a final list of Chemicals of Potential Concern for further assessment.

The screening assessment identified chemicals of concern from the expected emissions from the coal-fired generating station. Calculations were done for exposure to calculated soil and air concentrations. The contaminant concentrations in air and soil were derived following equations (A-2) and (A-6) from Attachment A.

The screening of the chemicals for the human health risk assessment based on soil concentrations calculated the contaminant exposure rate from soil ingestion equation (C-1):

$$Chemical\ Exposure\ Rate = \frac{C_s \times R_{ing-soil}}{B_w} \qquad \text{(C-1)}$$

where:

chemical exposure rate = (mg chemical/kg-d);

C_s	= (mg chemical/kg soil) for tilled and forage [calculated (A-6)];
$R_{ing\text{-}soil}$	= 80 mg soil/day for a child receptor [assumed for screening];
B_w	= 26 kg for child receptor [assumed for screening].

This calculated exposure rate is compared directly to the human toxicity data available which is listed in Table 2.2-1 in the report and the results of this screening are provided in Table C.2-1.

As seen in Tables C.2-1 and C.2-2, all carcinogenic chemicals are carried through to a more detailed assessment. The estimated ingestion rate of non-carcinogenic chemicals is compared to the oral reference dose (RfD_o). If the estimated exposure rate is greater than the TRV, the non-carcinogenic chemical is carried through the assessment. All non-carcinogens were dropped from further assessment. Additionally, a chemical cannot be carried through if there are no physical/chemical data or toxicity data available.

Table C.2-1
Soil Screening

Chemical	Carc/Non-carc	Sc - tilled (mg/kg)	Sc - Forage (mg/kg)	Cont Ingestion (mg/kg-d)	Cont Ingestion (mg/kg-d)	RfDo (mg/kg-d)	Carry Through (tilled)	Carry Through (forage)
Antimony	non-carc	$1.31x10^{-5}$	$1.31x10^{-5}$	$3.86x10^{-11}$	$3.86x10^{-11}$	$4.00x10^{-4}$	**	**
Arsenic	carc	1.11E-10	1.11E-10	3.27E-16	3.27E-16	3.00E-04	Arsenic	Arsenic
Barium	non-carc	3.81E-01	2.24E+00	1.12E-06	6.59E-06	7.00E-02	**	**
Beryllium	non-carc	1.94E-08	1.94E-08	5.70E-14	5.70E-14	2.00E-03	**	**
Boron	non-carc	8.76E-03	8.76E-03	2.58E-08	2.58E-08	2.00E-01	**	**
Bismuth	non-carc	1.03E-10	5.85E-10	3.04E-16	1.72E-15		no tox data	no tox data
Cadmium	non-carc	7.22E-10	7.22E-10	2.12E-15	2.12E-15	5.00E-04	**	**
Calcium	non-carc	2.51E-08	2.51E-08	7.37E-14	7.37E-14		no tox data	no tox data
Chlorine	non-carc	0.00E+00	0.00E+00	0.00E+00	0.00E+00		no tox data	no tox data
Chromium (Total)	non-carc	1.43E-10	1.43E-10	4.21E-16	4.21E-16	3.00E-03	**	**
Cobalt	non-carc	2.92E-04	5.87E-04	8.59E-10	1.73E-09	2.00E-02	**	**
Copper	non-carc	8.42E-03	2.52E-02	2.48E-08	7.42E-08	4.00E-02	**	**
Fluorine	non-carc	0.00E+00	0.00E+00	0.00E+00	0.00E+00		no tox data	no tox data
Iron	non-carc	1.97E-01	2.91E-01	5.79E-07	8.56E-07	3.00E-01	**	**
Lead	non-carc	1.94E-03	1.17E-02	5.70E-09	3.43E-08	3.60E-03	**	**
Lithium	non-carc	3.87E-05	3.87E-05	1.14E-10	1.14E-10		no tox data	no tox data
Magnesium	non-carc	3.84E-09	3.84E-09	1.13E-14	1.13E-14		no tox data	no tox data
Manganese	non-carc	6.02E-03	8.56E-03	1.77E-08	2.52E-08	1.40E-01	**	**
elemental mercury	non-carc	1.17E-08	1.17E-08	3.44E-14	3.44E-14		no tox data	no tox data
oxidized mercury	non-carc	1.67E-07	1.67E-07	4.90E-13	4.90E-13	3.00E-04	**	**
Particle-bound mercury	non-carc	2.34E-09	2.34E-09	6.87E-15	6.87E-15	3.00E-04	**	**
Molybdenum	non-carc	4.49E-03	1.58E-02	1.32E-08	4.64E-08	5.00E-03	**	**
Nickel	non-carc	4.37E-03	1.23E-02	1.28E-08	3.62E-08	2.00E-02	**	**
Palladium	non-carc	9.65E-04	6.66E-03	2.84E-09	1.96E-08		no tox data	no tox data
Phosphorous	non-carc	1.36E-07	3.06E-07	3.99E-13	8.99E-13		no tox data	no tox data
Potassium	non-carc	7.13E-12	7.13E-12	2.10E-17	2.10E-17		no tox data	no tox data
Selenium	non-carc	2.68E-04	2.69E-04	7.89E-10	7.92E-10	5.00E-03	**	**
Silver	non-carc	1.00E-05	1.00E-05	2.95E-11	2.95E-11	5.00E-03	**	**
Sodium	non-carc	3.77E-09	3.77E-09	1.11E-14	1.11E-14		no tox data	no tox data
Strontium	non-carc	4.60E-02	6.15E-02	1.35E-07	1.81E-07	6.00E-01	**	**
Thallium	non-carc	3.84E-03	1.02E-02	1.13E-08	3.01E-08	9.00E-05	**	**
Thorium	non-carc	1.57E-03	1.12E-02	4.63E-09	3.30E-08		no tox data	no tox data
Tin	non-carc	4.60E-02	2.61E-01	1.35E-07	7.67E-07	6.00E-01	**	**
Titanium	non-carc	6.01E-10	6.01E-10	1.77E-15	1.77E-15	4.00E+00	**	**
Uranium	non-carc	1.24E-04	1.49E-04	3.65E-10	4.38E-10	2.00E-04	**	**
Vanadium	non-carc	6.35E-03	1.99E-02	1.87E-08	5.85E-08	9.00E-03	**	**
Zinc	non-carc	1.00E-03	1.89E-03	2.95E-09	5.56E-09	3.00E-01	**	**
Acetaldehyde	non-carc	8.26E-18	8.26E-18	2.43E-23	2.43E-23		no tox data	no tox data
Acetophenone	non-carc	5.76E-17	5.76E-17	1.69E-22	1.69E-22	1.00E-01	**	**
Acrolein	non-carc	6.01E-18	6.01E-18	1.77E-23	1.77E-23	2.00E-02	**	**
Benzene	carc	6.54E-18	6.54E-18	1.92E-23	1.92E-23	4.00E-03	Benzene	Benzene
Benzyl chloride	carc	7.93E-16	7.93E-16	2.33E-21	2.33E-21		Benzyl chloride	Benzyl chloride
di-(2-Ethylhexyl) phthalate	carc	1.03E-11	8.06E-11	3.03E-17	2.37E-16	2.00E-02	di-(2-Ethylhexyl) phthalate	di-(2-Ethylhexyl) phthalate
Bromoform	carc	5.81E-21	5.81E-21	1.71E-26	1.71E-26	2.00E-02	Bromoform	Bromoform
Carbon disulfide	non-carc	2.52E-23	2.52E-23	7.42E-29	7.42E-29	1.00E-01	**	**
2-Chloroacetophenone	non-carc	6.00E-15	6.00E-15	1.76E-20	1.76E-20	8.60E-06	**	**
Chlorobenzene	non-carc	2.21E-18	2.21E-18	6.49E-24	6.49E-24	2.00E-02	**	**
Chloroform	non-carc	2.75E-19	2.75E-19	8.09E-25	8.09E-25	1.00E-02	**	**

Chemical	Carc/Non-carc	Sc - tilled (mg/kg)	Sc - Forage (mg/kg)	Cont Ingestion (mg/kg-d)	Cont Ingestion (mg/kg-d)	RfDo (mg/kg-d)	Carry Through (tilled)	Carry Through (forage)
Cumene	non-carc	4.54E-15	4.54E-15	1.34E-20	1.34E-20	1.00E-01	**	**
Cyanide	non-carc	1.42E-10	1.53E-10	4.18E-16	4.50E-16	2.00E-02	**	**
2,4-Dinitrotoluene	non-carc	2.96E-16	2.96E-16	8.72E-22	8.72E-22	2.00E-03	**	**
Dimethyl Sulphate	non-carc	4.11E-14	4.11E-14	1.21E-19	1.21E-19		no tox data	no tox data
Ethyl benzene	non-carc	3.73E-18	3.73E-18	1.10E-23	1.10E-23	1.00E-01	**	**
Ethyl Chloride	carc	1.59E-20	1.59E-20	4.68E-26	4.68E-26	4.00E-01	Ethyl Chloride	Ethyl Chloride
1,2-Dichloroethane	non-carc	1.79E-18	1.79E-18	5.26E-24	5.26E-24		no tox data	no tox data
Ethylene Dibromide	carc	5.28E-19	5.28E-19	1.55E-24	1.55E-24	9.00E-03	Ethylene Dibromide	Ethylene Dibromide
Formaldehyde	non-carc	2.04E-15	2.04E-15	5.99E-21	5.99E-21	1.50E-01	**	**
Hexane	non-carc	3.44E-18	3.44E-18	1.01E-23	1.01E-23		no tox data	no tox data
Isophorone	carc	1.14E-14	1.14E-14	3.36E-20	3.36E-20	2.00E-01	Isophorone	Isophorone
Bromomethane	non-carc	3.71E-19	3.71E-19	1.09E-24	1.09E-24	1.40E-03	**	**
Chloromethane	non-carc	2.56E-18	2.56E-18	7.53E-24	7.53E-24		no tox data	no tox data
2-Butanone	non-carc	4.67E-17	4.67E-17	1.37E-22	1.37E-22	6.00E-01	**	**
Methyl Hydrazine	carc	1.46E-13	1.46E-13	4.29E-19	4.29E-19		Methyl Hydrazine	Methyl Hydrazine
Methyl Methacrylate	non-carc	1.71E-14	1.71E-14	5.04E-20	5.04E-20	1.40E+00	**	**
tert Butyl methyl ether	non-carc	1.60E-12	1.66E-12	4.72E-18	4.88E-18		no tox data	no tox data
Dichloromethane	carc	6.82E-19	6.82E-19	2.01E-24	2.01E-24	6.00E-02	Dichloromethane	Dichloromethane
phenol	non-carc	2.37E-12	1.48E-11	6.98E-18	4.36E-17	3.00E-01	**	**
Propionaldehyde	non-carc	3.26E-13	3.26E-13	9.58E-19	9.58E-19		no tox data	no tox data
Tetrachloroethylene	non-carc	1.42E-18	1.42E-18	4.18E-24	4.18E-24	1.00E-02	**	**
Toluene	non-carc	7.11E-18	7.11E-18	2.09E-23	2.09E-23	2.00E-01	**	**
1,1,1-Trichloroethane	non-carc	2.43E-19	2.43E-19	7.14E-25	7.14E-25	2.80E-01	**	**
Styrene	non-carc	1.33E-17	1.33E-17	3.92E-23	3.92E-23	2.00E-01	**	**
m-Xylene	non-carc	1.63E-18	1.63E-18	4.81E-24	4.81E-24	2.00E-01	**	**
Vinyl acetate	non-carc	5.17E-17	5.17E-17	1.52E-22	1.52E-22	1.00E+00	**	**
Benzo(a)pyrene	carc	1.33E-06	1.02E-05	3.91E-12	3.01E-11		Benzo(a)pyrene	Benzo(a)pyrene
Benzo(b)fluoranthene	carc	1.22E-06	3.30E-06	3.59E-12	9.72E-12		Benzo(b)fluoranthene	Benzo(b)fluoranthene
Chrysene	carc	3.80E-06	2.29E-05	1.12E-11	6.72E-11		Chrysene	Chrysene
Indeno(1,2,3-cd)pyrene	carc	7.07E-07	5.52E-06	2.08E-12	1.62E-11		Indeno(1,2,3-cd)pyrene	Indeno(1,2,3-cd)pyrene
Benzo(g,h,i)perylene	non-carc	4.37E-06	3.25E-05	1.29E-11	9.56E-11		no tox data	no tox data
Fluoranthene	non-carc	1.81E-05	1.06E-04	5.34E-11	3.12E-10	4.00E-02	**	**
Napthalene	non-carc	1.96E-09	1.96E-09	5.78E-15	5.78E-15	2.00E-02	**	**
Phenanthrene	non-carc	9.58E-11	9.58E-11	2.82E-16	2.82E-16		no tox data	no tox data
Pyrene	non-carc	1.51E-06	1.51E-06	4.43E-12	4.43E-12	3.00E-02	**	**
TCDD, 2,3,7,8-	carc	1.08E-08	2.12E-08	3.19E-14	6.24E-14		TCDD, 2,3,7,8-	TCDD, 2,3,7,8-
TCDF, 2,3,7,8-	non-carc	1.06E-09	2.20E-09	3.11E-15	6.48E-15	1.00E-03	**	**
Acenaphthene	non-carc	1.57E-04	1.15E-03	4.62E-10	3.38E-09	6.00E-02	**	**
Anthracene	non-carc	6.88E-06	5.03E-05	2.02E-11	1.48E-10	3.00E-01	**	**
Benzo(a)anthracene	carc	9.21E-07	7.08E-06	2.71E-12	2.08E-11		Benzo(a)anthracene	Benzo(a)anthracene
Benzo(k)fluoranthene	carc	1.74E-06	1.34E-05	5.12E-12	3.93E-11		Benzo(k)fluoranthene	Benzo(k)fluoranthene
Biphenyl	non-carc	7.42E-05	5.43E-04	2.18E-10	1.60E-09	5.00E-02	**	**
Dibenzo(a,h)anthracene	carc	7.47E-08	5.74E-07	2.20E-13	1.69E-12		Dibenzo(a,h)anthracene	Dibenzo(a,h)anthracene
Fluorene	non-carc	2.18E-05	1.60E-04	6.42E-11	4.69E-10	4.00E-02	**	**

Chemical	Carc/Non-carc	Sc - tilled (mg/kg)	Sc - Forage (mg/kg)	Cont Ingestion (mg/kg-d)	Cont Ingestion (mg/kg-d)	RfDo (mg/kg-d)	Carry Through (tilled)	Carry Through (forage)
Quinoline	carc	8.49E-07	6.53E-06	2.50E-12	1.92E-11		Quinoline	Quinoline

The screening of chemicals for exposure to chemicals in air uses the non-carcinogenic human toxicity data available for inhalation pathways (RfD_i). The reference concentration is calculated from the RfD_i using the following equation (C-2):

$$Reference\,Concentration = \frac{RfD_i \times B_w}{R_{inh}} \times \frac{1000\,\mu g}{1\,mg} \times \frac{1\,d}{24\,hr} \qquad \text{(C-2)}$$

where:

reference concentration = [µg chemical/m^3]
RfD_i = reference dose (inhalation pathway)[mg chemical/(kg bw d)]
R_{inh} = 1.2 m^3/hr for a child receptor [assumed for screening]
B_w = 22 kg for child receptor [assumed for screening]

This reference concentration was compared directly to the estimated contaminant concentration in air, calculated following equation (A-2) shown in Attachment A. The results of the air screening are provided in Table C.2-2.

As seen in Table C.2-2, all carcinogenic chemicals are carried through to a more detailed assessment. The estimated concentration of non-carcinogenic chemicals in air is compared to the reference concentration (calculated from the inhalation reference dose (RfD_i)) and if the estimated concentration in air is greater than the reference concentration, the non-carcinogenic chemical is carried through the assessment. All non-carcinogens were dropped from further assessment.

Table C.2-2
Air Screening

Chemical	Carc/Non-carc	Ca ($\mu g/m^3$)	RfDi (mg/(kg-d)	TRV Concentration ($\mu g/m^3$)	Carry Through
Arsenic	carc	2.93E-05			Arsenic
Barium	non-carc	7.28E-03	1.43E-04	0.35	**
Beryllium	carc	9.14E-06	5.71E-06	0.01	Beryllium
Boron	non-carc	7.85E-04	5.71E-03	13.87	**
Bismuth	non-carc	3.03E-07			no tox data
Cadmium	carc	1.76E-06			Cadmium
Calcium	non-carc	1.61E-01			no tox data
Chlorine	non-carc	0.00E+00			no tox data
Chromium (Total)	carc	2.01E-04	2.20E-06	0.0053	Chromium (Total)
Cobalt	non-carc	1.57E-05	8.57E-06	0.02	**
Copper	non-carc	2.74E-04			no tox data
Fluorine	non-carc	0.00E+00			no tox data
Iron	non-carc	2.14E-02			no tox data
Lead	non-carc	3.89E-05			no tox data
Lithium	non-carc	2.09E-05			no tox data
Magnesium	non-carc	2.53E-02			no tox data
Manganese	non-carc	7.67E-04	1.40E-05	0.03	**
elemental mercury	non-carc	4.19E-06	8.60E-05	0.21	**
oxidized mercury	non-carc	5.33E-07	3.00E-04	0.73	**
particle-bound mercury	non-carc	1.78E-07	3.00E-04	0.73	**
Molybdenum	non-carc	2.65E-05			no tox data
Nickel	non-carc	1.62E-04			no tox data
Palladium	non-carc	2.60E-06			no tox data
Phosphorous	non-carc	8.15E-06			no tox data
Potassium	non-carc	2.80E-04			no tox data
Selenium	non-carc	6.59E-06			no tox data
Silver	non-carc	4.68E-06			no tox data
Sodium	non-carc	1.97E-02			no tox data
Strontium	non-carc	3.97E-03			no tox data
Thallium	non-carc	3.23E-05			no tox data
Thorium	non-carc	5.39E-06			no tox data
Tin	non-carc	1.55E-04			no tox data
Titanium	non-carc	3.97E-03	8.60E-03	20.89	**
Uranium	non-carc	5.08E-05			no tox data
Vanadium	non-carc	3.76E-04			no tox data
Zinc	non-carc	7.93E-05			no tox data
Acetaldehyde	carc	3.54E-05	2.57E-03	6.24	Acetaldehyde
Acetophenone	non-carc	9.31E-07			no tox data
Acrolein	non-carc	1.80E-05	5.71E-06	0.01	**
Benzene	carc	8.07E-05	8.60E-03	20.89	Benzene
Benzyl chloride	carc	4.35E-05			Benzyl chloride
di-(2-Ethylhexyl) phthalate	carc	4.53E-06	2.00E-02	48.57	di-(2-Ethylhexyl) phthalate
Bromoform	carc	2.42E-06	2.00E-02	48.57	Bromoform
Carbon disulfide	non-carc	8.07E-06	2.00E-01	485.71	**
2-Chloroacetophenone	non-carc	4.35E-07	8.57E-06	0.02	**
Chlorobenzene	non-carc	1.37E-06	1.70E-02	41.29	**
Chloroform	carc	3.66E-06	1.40E-02	34.00	Chloroform
Cumene	non-carc	3.29E-07	1.10E-01	267.14	**
Cyanide	non-carc	1.55E-04	8.57E-04	2.08	**
2,4-Dinitrotoluene	non-carc	1.74E-08	2.00E-03	4.86	**

Chemical	Carc/Non-carc	Ca (μg/m^3)	RfDi (mg/(kg-d)	TRV Concentration (μg/m^3)	Carry Through
Dimethyl Sulphate	non-carc	2.98E-06			no tox data
Ethyl benzene	non-carc	5.84E-06	2.90E-01	704.29	**
Ethyl Chloride	carc	2.61E-06	2.86E+00	6945.71	Ethyl Chloride
1,2-Dichloroethane	non-carc	2.48E-06			no tox data
Ethylene Dibromide	carc	7.45E-08	2.60E-03	6.31	Ethylene Dibromide
Formaldehyde	carc	1.49E-05			Formaldehyde
Hexane	non-carc	4.16E-06	5.71E-02	138.67	**
Isophorone	carc	3.60E-05	2.00E-01	485.71	Isophorone
Bromomethane	non-carc	9.93E-06	1.40E-03	3.40	**
Chloromethane	non-carc	3.29E-05			no tox data
2-Butanone	non-carc	2.42E-05	1.40E+00	3400.00	**
Methyl Hydrazine	carc	1.06E-05			Methyl Hydrazine
Methyl Methacrylate	non-carc	1.24E-06	2.00E-01	485.71	**
tert Butyl methyl ether	non-carc	2.17E-06	8.57E-01	2081.29	**
Dichloromethane	carc	1.80E-05	8.57E-01	2081.29	Dichloromethane
phenol (using pentachlorophenol chemical properties)	non-carc	9.93E-07	3.00E-01	728.57	**
Propionaldehyde	non-carc	2.36E-05			no tox data
Tetrachloroethylene	non-carc	2.67E-06	1.00E-02	24.29	**
Toluene	non-carc	1.49E-05	1.10E-01	267.14	**
1,1,1-Trichloroethane	non-carc	1.24E-06			no tox data
Styrene	non-carc	1.55E-06	2.90E-01	704.29	**
m-Xylene	non-carc	2.30E-06	2.90E-02	70.43	**
Vinyl acetate	non-carc	4.72E-07	5.71E-02	138.67	**
Benzo(a)pyrene	carc	3.80E-09			Benzo(a)pyrene
Benzo(b)fluoranthene	carc	4.09E-09			Benzo(b)fluoranthene
Chrysene	carc	1.11E-08			Chrysene
Indeno(1,2,3-cd)pyrene	carc	2.02E-09			Indeno(1,2,3-cd)pyrene
Benzo(g,h,i)perylene	non-carc	1.17E-08			no tox data
Fluoranthene	non-carc	4.97E-08	4.00E-02	97.14	**
Napthalene	non-carc	1.75E-07	8.57E-04	2.08	**
Phenanthrene	non-carc	3.21E-07			no tox data
Pyrene	non-carc	3.21E-08	3.00E-02	72.86	**
TCDD, 2,3,7,8-	carc	2.56E-10			TCDD, 2,3,7,8-
TCDF, 2,3,7,8-	non-carc	2.44E-10	1.00E-03	2.43	**
Acenaphthene	non-carc	4.21E-07			no tox data
Anthracene	non-carc	1.84E-08			no tox data
Benzo(a)anthracene	carc	2.63E-09			Benzo(a)anthracene
Benzo(k)fluoranthene	carc	4.97E-09			Benzo(k)fluoranthene
Biphenyl	non-carc	1.99E-07			no tox data
Dibenzo(a,h)anthracene	carc	2.13E-10			Dibenzo(a,h)anthracene
Fluorene	non-carc	5.84E-08			no tox data
Quinoline	carc	2.42E-09			Quinoline

ATTACHMENT D

Sensitivity Analysis
(Consumption of Milk and Meat from Local Area)

ATTACHMENT D

This attachment provides ingestion dose for a hypothetical residential receptor at the maximum POI location consuming 44 % of milk and meat from the study area (Tables D.1-1 for scenario OS3). The existing study assumed 5 % of milk and meat is from the study area. The comparison of the total risk for these two different intakes is also shown in Table D.1-2. Upon examing Table D.1-2, the total risk levels for scenario OS3 using 44 % of milk and meat from the study area results in increases in risk levels for some COPC; however, these increases do not result in a change in the conclusion of the assessment.

Table D.1-1
Total Ingestion Dose Using 44 % Intake from Local Area- Current Coal Source Scenario

Dose Calculation	Ingestion (mg/(kg-d))				Total
COPC	**Soil**	**Veg**	**Beef**	**Cow Milk**	**Ingestion**
Arsenic	8.18E-17	2.68E-09	1.89E-08	8.52E-10	2.24E-08
Beryllium	2.22E-14	3.34E-10	1.17E-09	1.59E-12	1.51E-09
Cadmium	8.25E-16	2.73E-10	1.15E-10	9.41E-12	3.98E-10
Chromium (Total)	1.64E-16	9.70E-09	1.88E-07	7.71E-08	2.75E-07
Acetaldehyde	2.98E-16	5.29E-13	8.91E-16	3.20E-16	5.30E-13
Benzene	1.52E-16	7.89E-14	1.91E-14	6.65E-15	1.05E-13
Benzyl chloride	1.84E-14	7.08E-12	1.88E-12	6.70E-13	9.65E-12
di-(2-Ethylhexyl) phthalate	1.87E-09	1.63E-08	3.31E-07	1.02E-07	4.52E-07
Bromoform	1.35E-19	4.72E-17	1.38E-17	5.13E-18	6.62E-17
Chloroform	9.91E-18	5.82E-15	1.13E-15	3.99E-16	7.36E-15
Ethyl Chloride	3.69E-19	1.00E-15	1.80E-19	8.82E-20	1.00E-15
Ethylene Dibromide	1.22E-17	7.02E-15	1.61E-19	8.91E-20	7.03E-15
Formaldehyde	7.34E-14	1.30E-10	8.80E-13	3.14E-13	1.32E-10
Isophorone	2.65E-13	2.36E-10	2.28E-11	8.23E-12	2.67E-10
Methyl Hydrazine	3.38E-12	1.07E-07	1.31E-14	7.16E-15	1.07E-07
Dichloromethane	1.58E-17	2.32E-14	1.23E-15	4.25E-16	2.49E-14
Benzo(a)pyrene	7.54E-12	5.20E-12	4.71E-10	1.33E-10	6.17E-10
Benzo(b)fluoranthene	2.43E-12	4.63E-12	3.53E-10	1.08E-10	4.68E-10
Chrysene	1.68E-11	1.96E-11	1.36E-09	3.96E-10	1.80E-09
Indeno(1,2,3-cd)pyrene	4.07E-12	1.89E-12	1.84E-10	5.30E-11	2.43E-10
TCDD, 2,3,7,8-	1.56E-14	3.18E-14	2.38E-12	7.35E-13	3.16E-12
Benzo(a)anthracene	4.55E-12	4.68E-12	3.39E-10	9.76E-11	4.46E-10
Benzo(k)fluoranthene	1.00E-11	6.50E-12	5.84E-10	1.69E-10	7.69E-10
	6.20E-10	2.99E-09	5.05E-10	2.29E-10	4.34E-09
Dibenzo(a,h)anthracene	4.31E-13	2.12E-13	2.10E-11	5.91E-12	2.75E-11
Quinoline	4.81E-12	3.41E-10	3.40E-13	1.65E-13	3.46E-10

Table D.1-2
Comparison of Total Risk Levels for the Two Different Consumption Percentages of Milk and Meat From Local Area

	Scenario OS3 -Adult	
	existing – 5 % milk and meat	**44 % milk and meat**
COPC	**Total Risk**	**Total Risk**
Arsenic	$1.34x10^{-7}$	$1.60x10^{-7}$
Beryllium	$2.19x10^{-8}$	$2.19x10^{-8}$
Cadmium	$3.17x10^{-9}$	$3.17x10^{-9}$
Chromium (Total)	**$2.41x10^{-6}$**	**$2.41x10^{-6}$**
Acetaldehyde	$7.79x10^{-11}$	$7.79x10^{-11}$
Benzene	$6.23x10^{-10}$	$6.23x10^{-10}$
Benzyl chloride	$2.11x10^{-9}$	$2.11x10^{-9}$
di-(2-Ethylhexyl) phthalate	$9.63x10^{-10}$	$6.35x10^{-9}$
Bromoform	$2.66x10^{-12}$	$2.66x10^{-12}$
Chloroform	$8.42x10^{-11}$	$8.42x10^{-11}$
Ethyl Chloride	$2.16x10^{-12}$	$2.16x10^{-12}$
Ethylene Dibromide	$4.26x10^{-11}$	$4.26x10^{-11}$
Formaldehyde	$1.96x10^{-10}$	$1.96x10^{-10}$
Isophorone	$1.00x10^{-11}$	$1.00x10^{-11}$
Methyl Hydrazine	$1.21x10^{-7}$	$1.21x10^{-7}$
Dichloromethane	$8.49x10^{-12}$	$8.49x10^{-12}$
Benzo(a)pyrene	$6.02x10^{-10}$	$4.51x10^{-9}$
Benzo(b)fluoranthene	$4.42x10^{-11}$	$3.42x10^{-10}$
Chrysene	$1.75x10^{-12}$	$1.31x10^{-11}$
Indeno(1,2,3-cd)pyrene	$2.45x10^{-11}$	$1.78x10^{-10}$
TCDD, 2,3,7,8-	$7.11x10^{-8}$	$4.85x10^{-7}$
Benzo(a)anthracene	$4.35x10^{-11}$	$3.26x10^{-10}$
Benzo(k)fluoranthene	$7.56x10^{-12}$	$5.63x10^{-11}$
Dibenzo(a,h)anthracene	$2.74x10^{-11}$	$2.01x10^{-10}$
Quinoline	$1.04x10^{-9}$	$1.04x10^{-9}$