

HUMAN HEALTH RISK ASSESSMENT
In Support of Proposed
Mid-Michigan Energy Station
Midland, Michigan

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LIST OF ACRONYMS

ADD	Average daily dose
AERMIC	American Meteorological Society/Environmental Protection Agency Regulatory Model Improvement Committee
AERMOD	AMS/EPA Regulatory Model Improvement Committee (AERMIC) Model
AQD	Air Quality Division
BAF	Bioaccumulation factor
BCF	Bioconcentration factor
BW	Body weight
C _a	Concentration in air
CAA	Clean Air Act
CDC	Centers for Disease Control
COPC	Compound of potential concern
DEM	Digital elevation model
DW	Dry weight of soil or plant/animal tissue
FW	Fresh weight (or whole/wet weight) of plant or animal tissue
HEAST	Health Effects Assessment Summary Tables
HHRA	Human Health Risk Assessment
HHRAP	Human Health Risk Assessment Protocol
HI	Hazard index
HQ	Hazard quotient
IEUBK	Integrated Exposure Uptake Biokinetic (Model)
IRIS	Integrated Risk Information System
LOAEL	Lowest observed adverse effect level
MDL	Method detection limit
MDEQ	Michigan Department of Environmental Quality
MW	Megwatt
NAAQS	National Ambient Air Quality Standards
NAD	North American datum
NOAEL	No observed adverse effect level
NWS	National Weather Service
PSD	Prevention of significant deterioration
RfC	Reference air concentration

RfD	Reference dose
RME	Reasonable maximum exposure
s	Second
URF	Unit risk factor
USEPA	U.S. Environmental Protection Agency
USLE	Universal soil loss equation
UTM	Universal transverse mercator

1.0 INTRODUCTION

Mid-Michigan Energy, LLC (MME) is a single purpose, limited liability corporation formed to develop, construct, own and operate a proposed 750-MW supercritical or ultra-supercritical pulverized coal (PC)-fired electric power generation facility in the City of Midland in Midland County, Michigan.

The Michigan Department of Environmental Quality (MDEQ) has requested that a human health risk assessment (HHRA) be conducted as part of the permitting process for the new facility. The MME HHRA focuses on mercury, lead and dioxin emissions and is based on current federal risk assessment guidance titled Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities released by the Office of Solid Waste (OSW) in final form in September 2005 (USEPA, 2005).

The purpose of the HHRA is to present, in a detailed fashion, the methods, assumptions, and some of the variable values that have been used to estimate potential health impacts on human receptors resulting from operation of the proposed MME facility. The HHRA consists of the following components:

- Facility description;
- Emissions characterization;
- Air modeling;
- Exposure assessment;
- Toxicity assessment;
- Risk characterization;
- Uncertainty Analysis; and
- Summary and Conclusions.

Potential risks have been estimated for several exposure scenarios and several identified sensitive populations. It must be emphasized that preparation of a HHRA according to current USEPA guidance will provide a conservative evaluation of the potential human health risks associated with operation of the proposed MME facility. Through the incorporation of a series of reasonable maximum exposure (RME) default parameters, the HHRA is designed not to underestimate potential risks.

2.0 SITE DESCRIPTION

This section provides general information about both the proposed MME facility and the area surrounding the facility. Its purpose is several-fold, including to provide the reader with some characteristics of the proposed facility under evaluation, to provide some understanding of the surrounding land use, and to clarify the process used to identify potential receptors which have been evaluated in the MME HHRA.

2.1 Location and Topography

The proposed MME facility will be located on a 132-acre (approximate) acre site near the intersection of South Saginaw Road and Waldo Avenue. Current HHRA guidance developed by USEPA (2005) recommends that the study area used to evaluate potential risks/hazards should extend at least 10-kilometers (km) in all directions from the proposed MME facility. In fact, experience indicates that the maximum impacts from combustion units tend to occur within 3-km of the emission source. Figure 2-1 shows a digitized topographic map of the area surrounding MME facility out to a distance of approximately 5-km. The figure represents a mosaic of several digitized topographic maps and shows the location of the main stack as well as the property boundary for the MME facility.

Figure 2-2 provides a digitized high-resolution aerial photograph of the area surrounding the facility representing conditions in 2005 out to a distance of approximately 10-km. Several features of importance to the HHRA can be seen in Figure 2-2, including areas of significant population density (i.e., the City of Midland) and major surface water bodies (i.e., the Tittibawassee River). Examination of Figure 2-2 shows that land use immediately to the west and north of the facility is primarily industrial with land use to the south and east of the facility primarily agricultural with some residential presence.

2.2 Demographics and Land Use

Midland County encompasses an area of approximately 1,367 square kilometers. Based on the 2000 census, the population of the county was 82,874 with a median household income, in 1999 dollars, of \$45,674 and a median family income of \$55,483. The per capita income was \$23,383 with 5.7 percent of families below the poverty level. The demographic profile of Midland County residents, compiled during the 2000 census, is as follows:

- Caucasian (95.5 percent);
- African American (1.0 percent);
- Asian (1.5 percent);
- Native American (0.4 percent);
- Native Hawaiian (0.1 percent)
- Other (0.4 percent);

-
- Reporting more than one race (1.1 percent); and
 - Hispanic/Latino of any race (1.6 percent).

Based on the 2000 census, the population of the City of Midland was 41,685 with a median household income, in 1999 dollars, of \$48,444 and a median family income of \$64,949. The per capita income was \$26,818 with 5.5 percent of families below the poverty level. The demographic profile of City of Midland residents, compiled during the 2000 census, is as follows:

- Caucasian (93.4 percent);
- African American (1.8 percent);
- Asian (2.7 percent);
- Native American (0.3 percent);
- Native Hawaiian (0.1 percent)
- Other (0.6 percent); and
- Reporting more than one race (1.2 percent); and
- Hispanic/Latino of any race (1.9 percent).

2.3 Facility and Combustion Unit Description

A full description of the proposed MME facility is provided in Section 3 of this Prevention of Significant Deterioration Air Permit to Install Application, dated September 2007. The information will not be repeated within the HHRA, however, if interested, the reader is referred to those sections of the application.

3.0 EMISSIONS CHARACTERIZATION

Based on discussions with the MDEQ, mercury, lead and dioxin are three chemicals of potential concern (COPCs) for the MME HHRA and the goal of the HHRA is to evaluate the potential human health impacts associated with stack emissions generated during 'worst-case' normal operation of the new facility. Although the selection of COPCs for the HHRA was straightforward, the development of representative emission rates for those COPCs was somewhat more complicated. Although a general description of the approach used is provided in the following sections, the reader is referred to Appendix C of this Prevention of Significant Deterioration Air Permit to Install Application, dated September 2007.

3.1 Estimation of Mercury Emission Rates

Mercury emission rates evaluated in the HHRA are provided in Table 3-1. Emission rates for mercury were based upon the chemical analysis of representative coal feed stocks and the design features of the MME facility. Since the permit will limit the emissions of mercury on a maximum potential basis, the emission estimates used in the HHRA are conservative (i.e., actual emissions should be lower than those evaluated in the HHRA). There were assumed to be multiple emission sources for mercury. In addition to the mercury present in the coal, contributions from the combustion of natural gas in the Auxiliary Boiler and Steam Boilers I, II and III were also considered.

3.1.1 Speciation of Mercury Emissions

Unlike most other COPCs, mercury emitted from combustion units can exist in several different chemical forms. As will be discussed in later sections of the HHRA, these different mercury species have different toxic characteristics. They also have different physicochemical characteristics which can influence their air dispersion characteristics and their susceptibility to wet and dry deposition. This requires that, where possible, the emissions of mercury be partitioned into the three forms of mercury that are known to encompass the total mercury emissions. These three forms are: vapor phase elemental mercury (Hg⁰), vapor phase divalent mercury (Hg²⁺), and particle bound mercury (HgP).

U.S. Department of Energy documentation (DOE, 2006) was the source of the mercury speciation shown in Table 3-2. The values were based on mercury testing conducted on Hawthorn Unit 5 which is a PC-fired boiler utilizing PRB coal, low NO_x burner/overfire air, selective catalytic reduction, spray dry absorber, activated carbon injection (non-halogenated) and fabric filter baghouse. The Hawthorn Unit 5 speciation is expected to be representative of MME facility mercury speciation in emissions from the PC Boiler due to the similar emission control systems and fuel source. Once the total mercury emission rate was developed for the five boilers, that total rate was divided according to the partitioning provided in Table 3-2 to establish the emission rates of each form of mercury.

As surrogate mercury partitioning data were not available for the natural gas fired boilers, a conservative approach was adopted to ensure that mercury emission impacts would not be underestimated. When elemental mercury was being modeled, the assumption was made that the mercury emissions from each of the natural gas fired boilers were present entirely as elemental mercury. When divalent mercury was being modeled, the assumption was made that the mercury emissions from each of the natural gas fired boilers were present entirely as divalent mercury.

3.2 Estimation of Lead Emission Rates

Lead emission rates evaluated in the HHRA are provided in Table 3-1. In contrast with the situation with mercury, lead emissions are assumed to be limited to the combustion of coal in the PC Boiler. Stack emission rates for lead were based upon the chemical analysis of representative coal feed stocks and the design features of the MME facility. Since the permit will limit the emissions of lead on a maximum potential basis, the emission estimates used in the HHRA are conservative (i.e., actual emissions should be lower than those evaluated in the HHRA).

3.3 Estimation of Dioxin Emission Rates

Dioxin emission rates evaluated in the HHRA are provided in Table 3-1. There is limited information describing the emissions of dioxins/furans from coal fired electric generating facilities. Appendix C of the HHRA summarizes the available literature used to develop the emission estimates evaluated in the HHRA. Although the literature suggests that several dioxin/furan congeners could be present in the emissions from the PC Boiler, the HHRA has taken the conservative approach that all potential dioxin/furan congeners would be present as 2,3,7,8-TCDD. As that congener (along with 1,2,3,7,8-pentachlorodibenzo(p)dioxin) is assigned the highest carcinogenic potency, that decision should result in an overestimation of potential dioxin/furan impacts.

4.0 AIR MODELING

4.1 Introduction

A detailed description of the air modeling conducted in support of the HHRA has been provided in Section 5 (i.e., Air Dispersion Modeling methodology) of this Prevention of Significant Deterioration Air Permit to Install Application, dated September 2007. This HHRA is being submitted as an appendix to the application and, therefore, that information is not repeated. Instead, the current section only provides information to supplement the information in Section 5. Note that Appendix G of the application contains detailed modeling background information and electronic files generated during the air modeling activity.

Effective December 9, 2005, the recommended dispersion model in the USEPA's Guideline on Air Quality Models is AERMOD, the air dispersion modeling program developed by the American Meteorological Society/Environmental Protection Agency Regulatory Model Improvement Committee (AERMIC). AERMOD version 07026 was used to calculate impact concentrations and deposition values for the various COPCs addressed in the HHRA.

4.2 Air Modeling Inputs

4.2.1. Stack Height and Building Downwash Consideration

The AERMOD dispersion model considers the influence of building structures on exhaust stack plumes. These conditions may occur when the height of an exhaust stack is less than its Good Engineering Practice (GEP) stack height (generally 2.5 times the height of the influencing structure). A building may have an influence on an exhaust plume if the distance between the building and the stack is less than five times the height or width (whichever is smaller) of the building.

The location of the influencing structures at the Facility relative to the exhaust stacks were entered in the USEPA BPIP-PRIME program. The BPIP-PRIME program calculates the projected influence of building widths and heights, depending upon wind direction, for use in the building downwash algorithms of the AERMOD model.

4.2.2. Meteorological Data

The most recent five years of available surface and upper air meteorological data (2002-2006) recorded at the nearest National Weather Service (NWS) Station was used to determine impact concentrations and deposition values. The meteorological data used was recorded at the Midland-Bay City-Saginaw (MBS) Airport in Freeland, Michigan (NWS Station Number 14845). The meteorological data files were obtained from the MDEQ-AQD website.

4.2.3. Receptors

Receptor positions (i.e., locations where pollutant impact concentrations and deposition values are determined) were established based on USEPA risk guidance. An initial grid of receptors with 100-m spacing, extending from the PC boiler exhaust stack to 3-km, was utilized. The model was augmented with an additional receptor grid with 500-m spacing which extended from the PC boiler stack to a minimum distance of 10-km.

4.2.4. Terrain Considerations

The AERMOD dispersion model is capable of accounting for terrain elevation when calculating impact concentrations. To ensure that the model inputs simulated actual conditions at the facility as accurately as possible, terrain elevations were included in this modeling analysis. The elevations were based upon Digital Elevation Model (DEM) terrain data gathered by the United States Geological Survey (USGS). The DEM data was obtained from a commercial source (i.e., WebGIS.com) approved by the MDEQ.

4.2.5. Deposition Parameters

In addition to the physical parameters (e.g., stack and building heights and locations) and pollutant emission rates which are all presented in the Prevention of Significant Deterioration Air Permit to Install Application, dated September 2007, AERMOD requires additional parameters to calculate the wet and dry deposition of vapors and particulate emissions.

4.2.5.1. Dry Deposition Parameters

The deposition of pollutants in the vapor phase is affected by several variables including the seasonal category and land use surrounding the source, which must be input into the model. The seasonal categories, shown below, were entered for each of the twelve months of the year based on the location of the proposed source.

- 1 - Midsummer with lush vegetation
- 2 - Autumn with unharvested cropland
- 3 - Late autumn after frost and harvest, or winter with no snow
- 4 - Winter with snow on the ground
- 5 - Transitional spring with partial green coverage or short annuals

To determine land use, an area circumscribed with a 3-km radius surrounding the source was divided into 36 sections, each representing a 10-degree arc. Each of the 36 parcels was then examined using USGS land use/land cover information as well as an aerial photograph to determine the predominant land use represented by the following categories:

-
- 1 - Urban Land
 - 2 - Agricultural Land
 - 3 - Rangeland
 - 4 - Forest
 - 5 - Suburban Area, Grassy
 - 6 - Suburban Area, Forested
 - 7 - Bodies of Water
 - 8 - Barren Land, Mostly Desert
 - 9 - Nonforested Wetlands

Values input for the seasonal category and land use classifications for the proposed facility are present in the input modeling files provided in Appendix G of the Prevention of Significant Deterioration Air Permit to Install Application, dated September 2007.

4.2.5.2. Gas Deposition Variables

Deposition of vapor phase constituents is also affected by gas deposition variables including the material's diffusivity in air, diffusivity in water, cuticular resistance, and Henry's law coefficient. Where available, values of these variables for each pollutant modeled were taken from the HHRA guidance (USEPA, 2005). Where values for these variables were not present in the USEPA guidance, values were obtained from the Deposition Parameterizations for the Industrial Source Complex (ISC3) Model, often referred to as the ANL report.

The values for the gas deposition for each pollutant modeled can be found in the input modeling files provided in Appendix G of the Prevention of Significant Deterioration Air Permit to Install Application, dated September 2007.

4.2.6. Particle Size Distribution

Deposition of constituents present in particulate form is affected by the particle size distribution of the particulate, as well as the density of the particulate. Pursuant to USEPA and MDEQ-AQD guidance, the density of particulate from the boiler was assumed to be 1 g/cm³. The particle size distribution was based upon Table 1.1-6 Cumulative Particle Size Distribution and Size Specific Emission Factors for Dry Bottom Boilers Burning Pulverized Bituminous and Subbituminous Coal of USEPA AP-42. Based on Table 1.1-6 and USEPA HHRA guidance, the particle size distribution based on fraction of total mass, and distribution based on fraction of surface area, were determined for particulate and particle bound constituents as presented in Table 4-1.

While many of the modeling parameters such as the seasonal and land use categories within AERMOD are non-pollutant specific, other variables such as diffusivity in air and water and cuticular resistance *are* dependent on the constituent which is being modeled. Therefore, separate modeling was conducted for each COPC (i.e., mercury, lead, vapor phase dioxin, and particle bound dioxin).

4.3 Air Modeling Output

As described in Section 5 of the Prevention of Significant Deterioration Air Permit to Install Application, dated September 2007, COPC-specific AERMOD output files were generated for each of the five modeling years (i.e., 2002 through 2006). In addition, a plot (i.e., *.plt) file was generated for each run; these files contain the output data in a format which permits post-processing on a grid node-specific basis. As can be seen from the example data provided in Table 4-2, each line of the plot file contains output data for a discrete modeling node; the output data include the predicted air concentration, dry deposition flux, wet deposition flux and total deposition flux. The AERMOD runs for lead and mercury were based on estimated stack emission rates (see Table 3-1) rather than the default 1-g/s emission rate recommended in the HHRA guidance (USEPA, 2005). This approach was found to yield very low air concentration and deposition flux values when expressed in the default units of $\mu\text{g}/\text{m}^3$ and $\text{g}/\text{m}^2\text{-yr}$, respectively. To compensate for this problem, the AERMOD Control (CO) file was adjusted to generate air concentration and deposition flux values in units of ng/m^3 and $\mu\text{g}/\text{m}^2\text{-yr}$, respectively. Although the raw output data in the plot files are expressed in these units, they were converted to the default units for use in the HHRA.

Although there are seventeen dioxin/furan congeners considered by USEPA to be potential human carcinogens, the air modeling for the current HHRA assumed that the dioxin/furan congeners would all be emitted as 2,3,7,8-TCDD. Although this simplified the air modeling (i.e., one congener was being modeled rather than seventeen), it did represent a very conservative approach. As the HHRA guidance (USEPA, 2005) indicates that emitted 2,3,7,8-TCDD partitions between the vapor phase (66.4-percent) and particle-bound phase (33.6-percent), each was modeled separately at the default 1-g/s emission rate recommended in the guidance. The resulting air concentration and deposition flux values at each modeling node were then multiplied by the anticipated emission rate from Table 3-1 to yield the values considered in the HHRA.

4.3.1. Post-Processing of Output Data

The COPC-specific air modeling output plot files were averaged over the five modeling years to generate an average annual value for each output parameter at each modeled grid node. As each grid node is georeferenced (i.e., each has an easting and northing UTM coordinate), geographic information system (GIS) techniques can be used to evaluate the output data. Such an approach is necessary as the grid node pattern developed for the air

modeling is not uniform, with a higher density of nodes (i.e., 100-m spacing) close to the facility and a lower density (i.e., 500-m spacing) at further distances (see Figure 4-1). The GIS software used in the HHRA was ArcGIS™ with its extension ArcGIS SpatialAnalyst™, developed by ESRI, Redlands, California.

The GIS approach for processing the AERMOD output data involved raster analysis in which a uniform raster grid (25-m by 25-m) was created covering the entire study area; each 25-m cell in the grid has known geographic (i.e., x-y) coordinates and is assigned a z-value. For each AERMOD output parameter [e.g., air concentration of lead], spline interpolation methods were used to assign parameter values to each grid cell, resulting in a cell with x-y-z values (i.e., utm-x, utm-y and air concentration of lead (z)). Conceptually, spline interpolation is comparable to bending a sheet of rubber to pass through all the 'z' data points while minimizing the curvature of the surface. It fits a mathematical function to a specified number of nearest input points (in this case 12 points), while still passing through the sample points.

Based on the above description, spline interpolation was used to generate a georeferenced surface for each output parameter. As these surfaces are georeferenced, they can also be linked to aerial photos or topographic maps to identify the specific locations of greatest impact (i.e., residential locations or surface water bodies). Once these locations are identified, location-specific air modeling output parameters can be identified from the identified grid nodes or directly from the interpolated surface. The georeferenced surfaces can also be used to generate a series of contour lines linking locations of identical air concentrations or particle deposition flux. The air modeling outputs for each of the COPCs are presented in Figures 4-2 through 4-18 as follows:

- Elemental mercury: Figure 4-2;
- Divalent mercury: Figures 4-3 through 4-6;
- Lead: Figures 4-7 through 4-10;
- 2,3,7,8-TCDD [Vapor Phase]: Figures 4-11 through 4-14; and
- 2,3,7,8-TCDD [Particle-Bound Phase]: Figures 4-15 through 4-18.

It is important to note that different units have been used to describe air concentrations and deposition fluxes in Figures 4-2 through 4-18.

4.3.2. Evaluation of Emission Impacts on Candidate Residence Locations

One of the initial steps in the HHRA is the identification of reasonable receptor locations where emission impacts can be quantified and potential health risks determined. Ideally, the selected locations will represent worst-case exposures for the associated receptors which will ensure that impacts on receptors at other locations will be lower.

The results of the air modeling combined with information gathered during a driving reconnaissance of the area surrounding the MME facility identified the Candidate Residence Area shown in Figure 4-19. This area is located just across Waldo Avenue from the facility toward the southeast. An evaluation of the air modeling outputs for each of the COPCs indicated that the maximum impact locations within the Candidate Residence Area were not collocated. In an effort to ensure that emission impacts would not be underestimated, a composite residence receptor was created which comprised the highest value for each of the air modeling output parameters within the Candidate Residence Area. Table 4-3 shows the air parameter values selected for evaluating impacts at the residence location in the current HHRA.

4.3.3. Evaluation of Emission Impacts on Candidate Farm Locations

The results of the air modeling combined with information gathered during a driving reconnaissance of the area surrounding the MME facility identified the Candidate Farm Area shown in Figure 4-20. This area is located just across the Tittabawassee River from the facility and immediately to the south of the MCV Cooling Pond. Although portions of the area are currently wooded, they are zoned agricultural and could be developed into farms in the future. An evaluation of the air modeling outputs for each of the COPCs indicated that the maximum impact locations within the Candidate Farm Area were also not collocated. In an effort to ensure that emission impacts would not be underestimated, a composite farm receptor was created which comprised the highest value for each of the air modeling output parameters within the Candidate Farm Area. This also represents a conservative approach, as selected air modeling impact values typically represent average values over the aerial extent of a given farm property rather than maximum values. Table 4-4 shows the air parameter values selected for evaluating impacts at the farm location in the current HHRA.

4.3.4. Evaluation of Emission Impacts on Candidate Surface Water Bodies

Evaluation of surface water impacts using the HHRA guidance (USEPA, 2005) requires the separation of air modeling impacts on the surface water body from impacts on the watershed. The MME HHRA initially evaluated three candidate surface water bodies, the Tittabawassee River, Kiewassee Lake and an Unnamed Lake located approximately 6.5-km from the MME facility toward the southeast. Figure 4-21 shows the locations of the three candidate surface water bodies that were evaluated in the selection process.

The aerial boundaries for all three water bodies were developed from a georeferenced 2006 aerial photograph. The extent of each watershed was developed from commercially-available digital terrain maps which were mosaiced together to yield the terrain surface shown in Figure 4-22. Hydrology Tools, a component of ArcGIS SpatialAnalyst™, was then used to generate the flow line patterns shown in the figure; the lines are based on

relative differences in elevation and the assumption that water flows from higher to lower elevations. The flow line patterns for the Tittabawassee River were used to delineate the watershed area shown in Figure 4-23. It should be noted that the aerial extent of both the Tittabawassee River (and its watershed) were limited to a distance of 10-km from the proposed MME boiler stack location even though the actual watershed is considerably larger.

Due to the relatively small size of both Kiewassee Lake and the Unnamed Lake, the digital elevation information shown in Figure 4-22 was of little help in determining the spatial extent of their respective watersheds. In addition, both water bodies appear to be drainage/seep lakes with no identifiable entrance and exit streams. Two different functional watersheds were developed for Kiewassee Lake (Figure 4-24). The first was delineated by the bounding roads/highways, while the second was arbitrarily limited to an area encompassing a 100-m buffer around the lake. As there was no compelling evidence favoring the selection of either watershed, both were included in the surface water screening process summarized in Appendix A. In this screening process, all candidate surface water bodies were evaluated using the risk assessment process described in subsequent sections of this report. However, in order to keep the HHRA report to a manageable size, only those detailed results from the most-impacted water body have been included in the report. This is a conservative approach, as the modeled emission impacts on the other water bodies will be lower. For the unnamed lake, the watershed was delineated by the bounding roads/highways (Figure 4-25).

It should be noted that raster analysis was used to provide the average air parameter impacts on each candidate water body and the watershed. The approach is relatively simple. First, two separate raster grids were created, one for the water body and one for the watershed. Each grid cell was assigned a z-value of 1 and the raster calculator in ArcGIS SpatialAnalyst™ was used to isolate that portion of each air modeling output surface which overlaps the raster grid. Statistical analysis of the isolated surface grids was then used to generate the average z-value across the grid.

Experience has shown that it is not possible to evaluate which candidate water body will be most impacted based solely on the air modeling impacts. However, based on fate-and-transport modeling and the evaluation of fish tissue impacts (shown in Appendix A), Kiewassee Lake with a functional watershed extending 100-m from the lake in all directions was conservatively selected as the most impacted of the candidate surface water bodies and it was carried throughout the remainder of the HHRA. Table 4-5 shows the air parameter values selected for evaluating surface water impacts on Kiewassee Lake in the current HHRA.

5.0 EXPOSURE ASSESSMENT

5.1 Introduction

The purpose of the exposure assessment is to quantify human exposure to the targeted COPCs. The routes, duration, frequency, and magnitude of potential exposures are estimated in the exposure assessment which typically comprises the following steps:

- Identification of potentially exposed populations;
- Identification of potential exposure pathways;
- Quantification of media concentrations; and
- Quantification of exposure intakes.

The available guidance specifies particular exposure scenarios that should be evaluated and provides default values for most input parameters. The guidance does allow the flexibility to use available site-specific information to refine risk estimates to better represent plausible receptors and exposure scenarios. If the final estimated risks from the HHRA are below levels of concern, additional analysis of the risks from expected stack emissions is usually considered to be unnecessary.

The evaluation endpoints selected for use in the HHRA are estimates of individual risk for several exposure scenarios and the selected exposure scenarios are considered by MDEQ to be of greatest significance for anticipated emissions from the MME facility. The following subsections present the exposure assessment according to the steps outlined above.

5.2 Definition of the Study Area

For the purposes of the HHRA, the study area is defined as the land area within 10-km of the MME Station boiler stack (see Figure 2-2). Although potentially-sensitive human populations may reside beyond the study area, COPC air concentrations and particle/vapor deposition rates beyond that distance would typically be orders-of-magnitude lower. Consequently, the risk assessment methodology, to be used in the HHRA, should also be protective of individuals located outside of the study area.

5.3 Identification of Potentially Exposed Populations

Consistent with USEPA (2005) guidance and our understanding of MDEQ requirements, the MME HHRA will evaluate chronic exposure to stack emissions in the following population subgroups:

- Resident;
- Resident Child;

-
- Farmer (formerly Subsistence Farmer);
 - Farmer Child (formerly Subsistence Farmer Child);
 - Fisher (formerly Subsistence Fisher);
 - Fisher Child (formerly Subsistence Fisher Child); and
 - Breast-milk fed infant.

5.4 Identification of Potential Exposure Pathways

An exposure pathway describes the course a COPC can take from the facility stack to a potentially exposed individual. As emissions disperse downwind of the MME facility, individuals may be exposed directly to COPCs via inhalation; COPCs also may be deposited on soil, surface water, and vegetation. Consequently, individuals may be exposed indirectly to COPCs by ingesting affected soils, surface water (if used as a source of potable water), vegetation, livestock, poultry products, or fish.

The default receptors described above (i.e., Resident, Farmer and Fisher) could potentially be exposed to COPCs via both direct and indirect pathways. A discussion of these pathways is presented in the following sections.

5.4.1. Direct Exposures

Individuals may be exposed directly to emitted COPCs by inhaling vapors and particulates released to the air. The direct exposure pathway will be evaluated for all targeted population. Current agency guidance recommends that the evaluation of elemental mercury emissions be limited to direct inhalation exposures only (USEPA, 2005).

5.4.2. Indirect Exposures

The potentially exposed population subgroups of interest to the MDEQ may be exposed indirectly to COPCs by ingesting media affected by stack emissions. The following indirect exposure pathways are currently recommended in USEPA HHRA guidance for quantitative evaluation in the HHRA (USEPA, 2005):

- Incidental ingestion of surface soil [all receptors];
- Consumption of drinking water derived from surface water [all receptors, where applicable];
- Consumption of above-ground produce [all receptors];
- Consumption of below-ground (root) produce [all receptors];
- Consumption of beef and milk [Farmer and Farmer Child];
- Consumption of poultry, eggs, and pork [Farmer and Farmer Child];
- Consumption of fish [Fisher and Fisher Child]; and
- Consumption of breast milk by nursing infants [separate evaluation].

COPCs could potentially be deposited on area soils via wet and dry particle deposition, wet and dry deposition of vapors, and diffusion of vapor. Individuals may be exposed indirectly to COPCs through incidental ingestion of affected surface soil. Such exposures are most significant for younger children, who may come in contact with the soil when playing outdoors. Individuals may also be exposed to COPCs in soil while handling outdoor pets, gardening, or engaging in other outdoor activities. All receptors are assumed to be exposed to COPCs through this exposure pathway.

Consumption of affected food is an additional indirect exposure pathway to COPCs. COPCs could be deposited on homegrown produce within the study area and/or COPCs may be taken up from the soil directly into plants. Ingestion of homegrown produce has been evaluated for all population subgroups. The Farmer and Farmer Child may also be exposed to COPCs indirectly by consuming pork and beef and milk from cattle that inhale air or ingest stack emissions present in surface soils, surface water, silage, forage, and grain. They may also be exposed to COPCs through the ingestion of poultry products (eggs and meat). The Fisher and Fisher Child may be exposed to COPCs indirectly by consuming fish that bioaccumulate COPCs which deposit on watershed soils and migrate to sediments and surface waters.

Receptors may also be exposed to COPCs in surface water if the impacted surface water is used as a direct drinking water source (USEPA, 2005). It is our understanding that Midland obtains its municipal water from Whitestone Point in Lake Huron approximately 80-km (i.e., 50-miles) from Midland (Midland, 2006). As a consequence, this pathway has not been included in the MME HHRA.

According to USEPA guidance, the potential transport of COPCs from combustion facilities into groundwater is considered to be insignificant compared to the other exposure pathways (USEPA, 2005). Dermal contact with surface water and soil was also not considered in the HHRA, as current USEPA guidance indicates that dermal contact is likely to be much less significant than ingestion pathways (USEPA, 2005). The derivation of expected COPC concentrations in area soils, produce, surface water and fish is presented in Section 5.5.

5.5 Quantification of Media COPC Concentrations

This section describes the approaches used in the HHRA to estimate media COPC concentrations; included are the modeling procedures used to estimate COPC concentrations in air, soil, homegrown produce, surface water, and fish. Exposure estimates have been based primarily on USEPA HHRA guidance (USEPA, 2005). It should be noted that, under this guidance, different algorithms are recommended for the estimation of soil concentrations of carcinogenic and non-carcinogenic COPCs. As mercury and lead are both considered by USEPA to be non-

carcinogenic, the 2,3,7,8-TCDD (and sixteen other dioxin/furan congeners) are considered to be potential carcinogens in humans. Table 5-1 summarizes the fate and transport parameters used in the following section to estimate media COPC concentrations.

5.5.1. Modeled COPC Concentrations in Air

Air concentrations were derived from both the average annual vapor-phase and particulate-phase concentrations in air. Inhalation exposures are assumed to occur only for the operational life of the MME Station boiler (estimated for the current HHRA to be 30 years). When the air modeling is based on a unit emission rate (i.e., 1 g/s), air concentrations are calculated using the following equation [See Table B-5-1 from USEPA, 2005]:

$$C_a = Q \cdot [F_v \cdot C_{yv} + (1.0 - F_v) \cdot C_{yp}]$$

[Equation 5-1]

where:

- C_a = COPC air concentration ($\mu\text{g}/\text{m}^3$);
- Q = COPC stack emission rate (g/s);
- F_v = COPC fraction in vapor phase (unitless);
- C_{yv} = Normalized air concentration-vapor phase COPC ($\mu\text{g}\cdot\text{s}/\text{g}\cdot\text{m}^3$); and
- C_{yp} = Normalized air concentration-particle phase COPC ($\mu\text{g}\cdot\text{s}/\text{g}\cdot\text{m}^3$).

In the proposed MME HHRA actual/estimated COPC emission rates were modeled which meant that the above equation needed to be slightly modified as follows:

$$C_a = C_{yv} + C_{yp}$$

[Equation 5-2]

where:

- C_a = COPC air concentration ($\mu\text{g}/\text{m}^3$);
- C_{yv} = Air concentration-vapor phase COPC ($\mu\text{g}/\text{m}^3$); and
- C_{yp} = Air concentration-particle phase COPC ($\mu\text{g}/\text{m}^3$).

Note that the units for C_{yv} and C_{yp} are changed in Equation 5-2. In the MME HHRA, lead is assumed to exist completely in the particle phase, further simplifying Equation 5-2. Vapor phase mercury (both Hg^{+2} and Hg^0) and 2,3,7,8-TCDD and particle-bound 2,3,7,8-TCDD were each modeled separately. As described earlier, no measurable emissions of particle-bound Hg^{+2} are anticipated from the MME facility. The resulting air concentrations for mercury, lead and 2,3,7,8-TCDD at the selected residential and farm locations are summarized in Tables 5-2 and 5-3, respectively.

5.5.2. Modeled COPC Concentrations in Soil

Emissions from the MME facility could interact with the soil surface through the wet and dry deposition of both

particulate and vapor phase COPCs. COPC concentrations have been calculated in surface soil in order to COPC estimate intake by human receptors (through incidental ingestion) and by pigs, poultry, and grazing beef cattle and milk cows through incidental soil ingestion. Soil concentrations in targeted area watersheds were also calculated as the initial step in estimating water body and fish tissue COPC concentrations.

As previously described, AERMOD was used to estimate air concentrations and deposition flux rates for each COPC across the modeling grid. Soil concentrations were then developed at selected receptor locations. Soil concentrations are a function of the COPC-specific deposition term, which includes COPC-specific parameters and modeled stack emission and dispersion/deposition rates. COPC concentrations in surface soil were calculated based on a mixing depth of 2-cm and continuous operation of the MME facility boiler for 30 years; COPC concentrations in tilled soil were based on a mixing depth of 20-cm (i.e., 9-inches) (USEPA, 2005). Tilled soil concentrations are used to estimate the uptake of COPCs into both root vegetables and above-ground produce.

Modeling soil concentrations is a somewhat complicated process due, in part, to the different algorithms used to evaluate carcinogenic and non-carcinogenic COPCs as well as the different exposure conditions used under the various scenarios. It is important to remember that carcinogenic COPCs can also produce noncarcinogenic impacts on exposed receptors. As the modeling of soil concentrations for carcinogenic and noncarcinogenic endpoints involves a different series of algorithms, it will not be uncommon to observe different concentrations based on carcinogenic and noncarcinogenic endpoints. For example, according to USEPA (2005) guidance, exposure of the Farmer Child, Fisher Child, and Child Resident to carcinogenic COPCs is restricted to a six-year period. In order to provide sufficient conservatism, that exposure is assumed to occur between years 24 and 30 of the unit's 30-year operational life. In contrast, the assumption is made that the Farmer is exposed to facility emissions for the 30 years of anticipated unit operation plus an additional 10 years. Obviously, the soil concentrations used to estimate COPC exposure have to be calculated separately under these different scenarios.

Soil concentrations for both carcinogenic and non-carcinogenic COPCs utilize a deposition term (D_s) which is calculated using the following equation [i.e., See Table B-1-1 from USEPA, 2005]:

$$D_s = \frac{UCF \cdot Q}{z \cdot BD} \cdot [F_v (Dydv + Dywv) + (Dydp + Dywp) \cdot (1 - F_v)]$$

[Equation 5-3]

where:

D_s	=	Deposition term (mg/kg-yr);
Q	=	COPC stack emission rate (g/s);
F_v	=	COPC fraction in vapor phase (unitless);
$Dydv$	=	Normalized annual dry deposition flux-vapor phase COPCs (s/m^2 -yr);

- Dy_{wv} = Normalized annual wet deposition flux-vapor phase COPCs (s/m²-yr);
- Dy_{dp} = Normalized annual dry deposition flux-particle phase COPCs (s/m²-yr);
- Dy_{wp} = Normalized annual wet deposition flux-particle phase COPCs (s/m²-yr);
- z = Soil mixing depth (cm);
- BD = Soil bulk density (g/cm³); and
- UCF = Units conversion factor (100 mg-m²/kg-cm²).

Again, the nature of the air modeling conducted for the MME HHRA requires that Equation 5-3 be slightly modified to account for actual/estimated COPC emission rates being modeled rather than the default unit (i.e., 1 g/s) emission rate. This modification results in the following equation:

$$D_s = \frac{UCF}{z \cdot BD} \cdot [Dy_{dv} + Dy_{wv} + Dy_{dp} + Dy_{wp}]$$

[Equation 5-4]

where:

- D_s = Deposition term (mg/kg-yr);
- Dy_{dv} = Annual dry deposition flux-vapor phase COPCs (g/m²-yr);
- Dy_{wv} = Annual wet deposition flux-vapor phase COPCs (g/m²-yr);
- Dy_{dp} = Annual dry deposition flux-particle phase COPCs (g/m²-yr);
- Dy_{wp} = Annual wet deposition flux-particle phase COPCs (g/m²-yr);
- z = Soil mixing depth (cm);
- BD = Soil bulk density (g/cm³); and
- UCF = Units conversion factor (100 mg-m²/kg-cm²).

Note that the units for Dy_{dv}, Dy_{wv}, Dy_{dp} and Dy_{wp} are changed in Equation 5-4. The values calculated for D_s in tilled and untilled soil at the selected residence and farm locations are summarized in Tables 5-4 and 5-5, respectively. It should be noted that, under existing HHRA guidance (USEPA, 2005), the assumption is made that 2-percent of the total divalent mercury deposition (D_s) exists as methylmercury (MeHg).

For the estimation of soil concentrations of non-carcinogenic COPCs, the following equation has been used [i.e., See Table B-1-1 from USEPA, 2005]:

$$C_s = \frac{D_s \cdot (1 - \exp(-k_s \cdot tD))}{k_s}$$

[Equation 5-5]

where:

- C_s = Soil COPC concentration at time tD (mg/kg);
- k_s = Soil loss constant (yr⁻¹); and
- tD = Time period over which deposition occurs (yr).

Two different equations were used for the estimation of soil concentrations of carcinogenic COPCs, based on the exposure duration. For an exposure duration longer than the anticipated 30-year operating life of the combustion

unit (i.e., the Farmer at a 40-year exposure), the soil concentration has been estimated using the following equation [i.e., See Table B-2-1 from USEPA, 2005]:

$$C_s = \frac{\left(\frac{D_s \cdot tD - C_{s_{tD}}}{k_s} \right) + \left(\frac{C_{s_{tD}}}{k_s} \cdot [1 - \exp(-k_s \cdot (T_2 - tD))] \right)}{(T_2 - T_1)} \quad \text{for } T_1 < tD < T_2$$

[Equation 5-6]

where:

- T_2 = Exposure duration (yr); and
- T_1 = Beginning of exposure duration (yr).

For exposure durations shorter than or equal to the anticipated operating life of the combustion unit (i.e., the remaining receptors), soil concentrations have been estimated using the following equation [i.e., See Table B-2-1 from USEPA, 2005]:

$$C_s = \frac{D_s}{k_s \cdot (tD - T_1)} \cdot \left[\left(tD + \frac{\exp(-k_s \cdot tD)}{k_s} \right) - \left(T_1 + \frac{\exp(-k_s \cdot T_1)}{k_s} \right) \right] \quad \text{for } T_2 \leq tD$$

[Equation 5-7]

COPC concentrations in both tilled and untilled soil have been estimated for the Resident, the Resident Child, the Fisher, and the Fisher Child at the selected residence location (Table 5-6). The selected farm location was used to estimate soil concentrations for the Farmer and the Farmer Child. Estimated COPC concentrations in both tilled and untilled soil at the selected farm location are presented in Table 5-7. Again, note that both tables describe soil concentrations based on noncarcinogenic endpoints (designated with a “NC”) and carcinogenic endpoints (designated with a “C”). The different soil concentration values reported for the child and adult categories based on carcinogenic endpoints reflects the different exposure periods for the various receptors, as described previously.

In the preceding equations, the soil loss constant term, k_s , reflects the loss of COPCs from soil due to leaching, degradation, erosion, volatilization, and surface runoff and is calculated using the following equation [i.e., See Table B-1-2 from USEPA, 2005]:

$$k_s = k_{sl} + k_{sr} + k_{sg} + k_{sv} + k_{se}$$

[Equation 5-8]

where:

- k_s = Soil loss constant due to all processes (yr^{-1});
- k_{sl} = Soil loss constant due to leaching (yr^{-1});
- k_{sr} = Soil loss constant due to surface runoff (yr^{-1});
- k_{sg} = Soil loss constant due to degradation (yr^{-1});

k_{sv} = Soil loss constant due to volatilization (yr⁻¹); and
 k_{se} = Soil loss constant due to soil erosion (yr⁻¹).

Estimated values for the soil loss constant (k_s) are summarized in Table 5-8 and the calculations for the individual soil loss constants are described in the following sections.

Soil loss due to leaching (Table 5-9) was estimated using the following equation [i.e., See Table B-1-5 from USEPA, 2005]:

$$k_{sl} = \frac{P + I - RO - E_v}{z \cdot (\theta_{sw} + Kd_s \cdot BD)}$$

[Equation 5-9]

where:

k_{sl} = Soil loss coefficient due to leaching (yr⁻¹);
 P = Annual average precipitation (cm/yr);
 I = Annual average irrigation (cm/yr);
 E_v = Annual average evapotranspiration (cm/yr);
 RO = Annual average runoff (cm/yr);
 θ_{sw} = Soil volumetric water content (mL/cm³);
 z = Soil mixing depth (cm);
 BD = Bulk soil density (g/cm³); and
 K_{d_s} = Soil-to-water partition coefficient (mL/g).

Soil loss from surface runoff (Table 5-10) was estimated using the following equation [i.e., See Table B-1-4 from USEPA, 2005]:

$$k_{sr} = \frac{RO}{z \cdot (\theta_{sw} + Kd_s \cdot BD)}$$

[Equation 5-10]

where:

k_{sr} = Soil loss coefficient due to surface runoff (yr⁻¹);
 RO = Annual average runoff (cm/yr);
 θ_{sw} = Soil volumetric water content (mL/cm³);
 z = Soil mixing depth (cm);
 BD = Bulk soil density (g/cm³); and
 K_{d_s} = Soil-to-water partition coefficient (mL/g).

The soil loss constant due to volatilization (Table 5-11) was estimated using the following equation [i.e., See Table B-1-6 from USEPA, 2005]:

$$k_{sv} = \left[\frac{UCF \cdot H}{z \cdot Kd_s \cdot R \cdot T_a \cdot BD} \right] \cdot \left[\frac{D_a}{z} \cdot \left(1 - \left(\frac{BD}{\rho_s} \right) - \theta_{sw} \right) \right]$$

[Equation 5-11]

where:

k _{sv}	=	Soil loss constant due to volatilization (yr ⁻¹);
UCF	=	Units conversion factor (3.1536x10 ⁷ s/yr);
H	=	Henry's law constant (atm·m ³ /mol);
K _{d_s}	=	Soil-water partitioning coefficient (cm ³ /g);
R	=	Universal gas constant (atm·m ³ /mol·K);
T _a	=	Ambient air temperature (K);
BD	=	Soil bulk density (g/cm ³);
D _a	=	Diffusivity of contaminant in air (cm ² /s);
z	=	Soil mixing depth (cm);
ρ _s	=	Solids particle density (g/cm ³); and
θ _{sw}	=	Volumetric soil water content (mL/cm ³).

Default values for the soil loss constant due to degradation are provided in the current guidance (USEPA, 2005). There is considerable controversy concerning whether or not the soil loss constant due to erosion (k_{se}) should be included in the estimation of k_s. Current HHRA guidance indicates that k_{se} should be set equal to zero (USEPA, 2005), based on the argument that impacted soils erode both onto and away from any targeted area with no net impact. This might not always be the case, particularly at sites with elevated terrain. In the MME HHRA, the conservative decision was made to set k_{se} equal to zero as the terrain is relatively flat.

As will be discussed in a later section, consideration of k_{se} is appropriate when dealing with watershed soil losses. When used for that purpose, k_{se} was estimated using the following equation [i.e., See Table B-2-3 from USEPA, 2005]:

$$k_{se} = \frac{0.1 \cdot X_e \cdot SD \cdot ER}{BD \cdot z} \cdot \left(\frac{K_{d_s} \cdot BD}{\theta_{sw} + (K_{d_s} \cdot BD)} \right)$$

[Equation 5-12]

where:

k _{se}	=	Soil loss constant due to erosion (yr ⁻¹);
X _e	=	Unit soil loss (kg/m ² -yr);
SD	=	Sediment delivery ratio (unitless);
ER	=	Soil enrichment ratio (unitless);
K _{d_s}	=	Soil-to-water partition coefficient (mL/g);
BD	=	Soil bulk density (g/cm ³);
θ _{sw}	=	Soil volumetric water content (ml/cm ³); and
z	=	Soil mixing depth (cm).

Although this same series of equations was used for tilled soil, it should be noted that the value for soil mixing depth (z) is different; a soil mixing depth of 20-cm is used for tilled soil. The equations used to calculate the soil loss constants k_{sl} and k_{sr} both take the soil mixing depth into account resulting in different k_s values.

5.5.3. Modeled COPC Concentrations in Surface Water

The calculation of surface water COPC concentrations is needed in order to estimate human exposures through

the consumption of fish tissue (and drinking water where appropriate). The process involves estimating the average surface soil concentration of COPCs throughout the selected watershed followed by estimating the COPC loading to the water body. Pathways contributing to water body load include:

- Direct deposition;
- Runoff from impervious watershed surfaces;
- Runoff from pervious watershed surfaces;
- Vapor phase diffusion of COPCs; and
- Soil erosion from the watershed.

Table 4-4 summarizes the air modeling results for mercury, lead and 2,3,7,8-TCDD associated with the Kiewassee Lake and its watershed. Consistent with the HHRA guidance (USEPA, 2005), those impacts represent average parameter values determined over the selected water body and watershed areas. Table 5-12 summarizes the fate and transport parameters used in the modeling of COPC movement in Kiewassee Lake and its watershed as described in the following sections.

The equation used to calculate watershed soil concentrations for non-carcinogenic COPCs uses the deposition term (D_s) calculated using the following equation adapted from Equation 5-4:

$$D_s = \frac{UCF}{z \cdot BD} \cdot [Dytwv + Dytwp]$$

[Equation 5–13]

where:

- | | | |
|---------|---|--|
| D_s | = | Watershed deposition term (mg/kg-yr); |
| $Dytwv$ | = | Annual average total deposition flux onto watershed-vapor phase COPCs (g/m^2 -yr); |
| $Dytwp$ | = | Annual average total deposition flux onto watershed-particle phase COPCs (g/m^2 -yr); |
| z | = | Soil mixing depth (cm); |
| BD | = | Soil bulk density (g/cm^3); and |
| UCF | = | Units conversion factor ($100 \text{ mg}\cdot\text{m}^2/\text{kg}\cdot\text{cm}^2$). |

The values calculated for D_s in watershed soil are summarized in Table 5-13. It should be noted that, under the existing HHRA guidance (USEPA, 2005), the assumption is made that 2-percent of the divalent mercury deposition (D_s) onto watershed soils exists as methylmercury (MeHg).

For the estimation of watershed soil concentrations of COPCs based on noncarcinogenic endpoints, the following equation has been used:

$$Cs_{tD} = \frac{Ds \cdot (1 - \exp(-ks \cdot tD))}{ks}$$

[Equation 5-14]

where:

- Cs_{tD} = Watershed soil COPC concentration at time tD (mg/kg);
- ks = Soil loss constant (yr⁻¹); and
- tD = Time period over which deposition occurs (yr).

As the exposure periods for the Fisher and Fisher Child (i.e., 30-yr and 6-yr) do not exceed the estimated operating life of the facility, the estimation of watershed soil concentrations of COPCs based on carcinogenic endpoints were estimated using the following equation [i.e., See Table B-2-1 from USEPA, 2005]:

$$Cs = \frac{Ds}{ks \cdot (tD - T_1)} \cdot \left[\left(tD + \frac{\exp(-ks \cdot tD)}{ks} \right) - \left(T_1 + \frac{\exp(-ks \cdot T_1)}{ks} \right) \right] \text{ for } T_2 \leq tD$$

[Equation 5-15]

where:

- T₂ = Exposure duration (yr); and
- T₁ = Beginning of exposure duration (yr).

Estimated COPC concentrations in watershed soils are summarized in Table 5-14. The soil loss constant term (ks), reflects the loss of COPCs from soil due to leaching, degradation, erosion, volatilization, and surface runoff. The watershed soil loss values for leaching (ksl), volatilization (ksv) and surface runoff (ksr) are identical to those calculated for untilled residential soils (see Tables 5-9 through 5-11). In contrast to the situation with residential soils, where the soil loss constant due to erosion (kse) was set equal to zero, in watershed soils kse was calculated using Equation 5-12. Table 5-15 summarizes the values calculated for kse in watershed soils; the combined watershed soil loss constant (ks) values are summarized in Table 5-16.

The total COPC loading (L_T) to Kiewassee Lake was calculated using the following equation [i.e., See Table B-4-7 from USEPA, 2005]:

$$L_T = L_{Dep} + L_{Dif} + L_{RI} + L_R + L_E + L_I$$

[Equation 5-16]

where:

- L_T = Total COPC load to the surface water body (g/yr);
- L_{dep} = Total particle and wet vapor phase deposition to water body (g/yr);
- L_{dif} = Vapor phase diffusion load to water body (g/yr);
- L_{RI} = Runoff load to water body from impervious surfaces (g/yr);
- L_R = Runoff load to water body from pervious surfaces (g/yr);
- L_E = Soil erosion load to water body (g/yr); and
- L_I = Internal transfer (g/yr).

Current HHRA guidance (USEPA 2005) recommends using a default value of zero for L_I unless site-specific conditions indicate a need to consider internal transfer. As described previously, the air modeling conducted for the MME HHRA was based on actual/estimated COPC emission rates rather than the default unit (i.e., 1 g/s) emission rate. As a consequence, the following equations describing the estimation of L_{dep} , L_{dif} and L_{RI} were slightly modified from the equations found in the HHRA guidance (USEPA, 2005).

Table 5-17 describes the wet and dry deposition of particle-phase and vapor-phase COPCs (L_{Dep}) directly onto the surface of Kiewassee Lake. It was estimated using the following equation [i.e., modified Table B-4-8 from USEPA, 2005]:

$$L_{Dep} = [Dytwv + Dytwp] \cdot A_w \quad \text{[Equation 5-17]}$$

where:

- L_{dep} = Total particle and wet vapor phase deposition to water body (g/yr);
- $Dytwv$ = Annual average total (wet and dry) deposition flux onto surface water body-vapor phase COPCs ($\text{g}/\text{m}^2\text{-yr}$);
- $Dytwp$ = Annual average total (wet and dry) deposition flux onto surface water body-particle phase COPCs ($\text{g}/\text{m}^2\text{-yr}$); and
- A_w = Total water body surface area (m^2).

Table 5-18 describes COPC vapor-phase diffusion loading (L_{Dif}) directly onto the water surface of Kiewassee Lake. It was estimated using the following equation [i.e., modified Table B-4-12 from USEPA, 2005]:

$$L_{Dif} = \frac{K_v \cdot C_{ywv} \cdot A_w \cdot UCF}{\frac{H}{R \cdot T_{wk}}} \quad \text{[Equation 5-18]}$$

where:

- L_{dif} = Vapor phase diffusion load of COPCs to water body (g/yr);
- K_v = Diffusion mass transfer coefficient (m/yr);
- C_{ywv} = Average air concentration above water-vapor phase COPCs ($\mu\text{g}/\text{m}^3$);
- A_w = Water body surface area (m^2);
- H = Henry's law constant ($\text{atm}\cdot\text{m}^3/\text{mol}$);
- R = Universal gas law constant ($\text{atm}\cdot\text{m}^3/\text{mol}\cdot^\circ\text{K}$);
- T_{wk} = Water body temperature ($^\circ\text{K}$); and
- UCF = Units conversion factor ($10^{-6} \text{ g}/\mu\text{g}$).

Table 5-19 describes COPC runoff loading to Kiewassee Lake from impervious watershed surfaces (L_{RI}). It was estimated using the following equation [i.e., See Table B-4-9 from USEPA, 2005]:

$$L_{RI} = [Dytwv + Dytwp] \cdot A_i$$

[Equation 5-19]

where:

- L_{RI} = Runoff load to water body from impervious surfaces (g/yr);
- $Dytwv$ = Annual average total (wet and dry) deposition onto watershed-vapor phase COPCs (g/m²-yr);
- $Dytwp$ = Annual average total (wet and dry) deposition onto watershed-particle phase COPCs (g/m²-yr); and
- A_i = Impervious watershed area (m²).

Table 5-20 describes COPC runoff loading to Kiewassee Lake from pervious watershed soil surfaces (L_R). It was estimated using the following equation [i.e., See Table B-4-9 from USEPA, 2005]:

$$L_R = RO \cdot (A_L - A_i) \cdot \frac{Cs \cdot BD}{\theta_{sw} + Kd_s \cdot BD} \cdot UCF$$

[Equation 5-20]

where:

- L_R = Runoff load to water body from pervious surfaces (g/yr);
- RO = Average annual runoff from pervious areas (cm/yr);
- A_L = Total watershed area receiving COPC deposition (m²);
- A_i = Impervious watershed area receiving chemical deposition (m²);
- Cs = Average COPC concentration in watershed soils (mg/kg);
- BD = Soil bulk density (g/cm³);
- θ_{sw} = Volumetric soil water content (cm³/cm³);
- Kd_s = Soil-water partition coefficient (L/kg); and
- UCF = Unit conversion factor (10⁻² kg-cm²/mg-m²).

Table 5-21 describes COPC loading to Kiewassee Lake resulting from watershed soil erosion (L_E). It was calculated using the following equation [i.e., See Table B-4-11 from USEPA, 2005]:

$$L_E = X_e \cdot (A_L - A_i) \cdot SD \cdot ER \cdot \frac{Cs \cdot Kd_s \cdot BD}{\theta_{sw} + Kd_s \cdot BD} \cdot UCF$$

[Equation 5-21]

where:

- L_E = Soil erosion load of COPCs to water body (g/yr);
- X_e = Unit soil loss (kg/m²-yr);
- A_L = Total watershed area receiving COPC deposition (m²);
- A_i = Impervious watershed area receiving COPC deposition (m²);
- SD = Watershed sediment delivery ratio (unitless);
- ER = Soil enrichment ratio (unitless);
- Cs = Average COPC concentration in watershed soils (mg/kg);
- BD = Soil bulk density (g/cm³);
- θ_{sw} = Volumetric soil water content (cm³/cm³);
- Kd_s = Soil-water partition coefficient (L/kg); and
- UCF = Unit conversion factor (10⁻³ [g/kg]/[mg/kg]).

The unit soil loss (X_e) term and the watershed sediment delivery ratio (SD) terms (see Table 5-22) are estimated for each of the COPCs using the following equations [i.e., See Tables B-4-13 and B-4-14 from USEPA, 2005]:

$$X_e = RF \cdot K \cdot LS \cdot C \cdot PF \cdot \frac{UCF_1}{UCF_2}$$

[Equation 5-22]

where:

- X_e = Unit soil loss (kg/m²-yr);
- RF = USLE rainfall factor (yr⁻¹);
- K = USLE erodibility factor (ton/acre);
- LS = USLE length-slope factor (unitless);
- C = USLE cover management factor (unitless);
- PF = USLE supporting practice factor (unitless);
- UCF₁ = Unit conversion factor (907.18 kg/ton); and
- UCF₂ = Unit conversion factor (4047 m²/acre).

and

$$SD = a \cdot (A_L)^{-b}$$

[Equation 5-23]

where:

- SD = Watershed sediment delivery ratio (unitless);
- a = Empirical intercept coefficient (unitless);
- A_L = Total watershed area receiving COPC deposition (m²); and
- b = Empirical slope coefficient (unitless).

Total COPC loading to the targeted water body (i.e., Keweenaw Lake) resulting from MME Station emissions is summarized in Table 5-23. The total water body concentration in the water column and sediments is calculated from the water body load; this total concentration is partitioned into a dissolved water concentration, a total water column concentration, and a bed sediment concentration. The total water body concentration for each COPC, shown in Table 5-24, was calculated using the following equation [i.e., See Table B-4-15 from USEPA, 2005]:

$$C_{wtot} = \frac{L_T}{Vf_x \cdot f_{wc} + k_{wt} \cdot A_W \cdot (d_{wc} + d_{bs})}$$

[Equation 5-24]

where:

- C_{wtot} = Modeled total water body COPC concentration, including water column and bed sediment (mg/L);
- L_T = Total COPC load into water body, including deposition, diffusion, runoff, and erosion (g/yr);
- Vf_x = Average volumetric flow rate through water body (m³/yr);
- f_{wc} = Fraction of total water body chemical concentration that occurs in the water column (unitless);
- A_W = Total water body surface area (m²);

- d_{wc} = Depth of the water column (m);
- d_{bs} = Depth of the upper benthic layer (m); and
- k_{wt} = Total water body dissipation rate constant (yr^{-1}).

In the above equation, values for f_{wc} and k_{wt} were calculated using the following equations [i.e., See Tables B-4-16 and B-4-17 from USEPA, 2005]:

$$f_{wc} = \frac{(1 + Kd_{sw} \cdot TSS \cdot UCF) d_{wc} / d_z}{d_{wc} / d_z (1 + Kd_{sw} \cdot TSS \cdot UCF) + d_{bs} / d_z (\theta_{bs} + Kd_{bs} \cdot C_{BS})}$$

[Equation 5-25]

where:

- f_{wc} = Fraction of total water body COPC concentration that occurs in water column (unitless);
- Kd_{sw} = Suspended sediment/surface water partition coefficient (L/kg);
- Kd_{bs} = Bed sediment/sediment pore water partition coefficient (cm^3/g);
- TSS = Total suspended solids (mg/L);
- UCF = Unit conversion factor (10^{-6} kg/mg);
- θ_{bs} = Bed sediment porosity (unitless);
- d_{wc} = Depth of water column (m);
- d_{bs} = Depth of the upper benthic layer (m);
- d_z = Total water body depth (m); and
- C_{BS} = Bed sediment concentration (g/cm^3).

and

$$k_{wt} = f_{wc} \cdot k_v + f_{bs} \cdot k_b$$

[Equation 5-26]

where:

- k_{wt} = Overall total water body dissipation rate (yr^{-1});
- f_{wc} = Fraction of total COPC concentration in water column (unitless);
- f_{bs} = Fraction of total COPC concentration in benthic sediment (unitless);
- k_v = Water column volatilization rate (yr^{-1}); and
- k_b = Benthic burial rate (yr^{-1}).

Values for the water column volatilization rate (k_v) and the benthic burial rate (k_b), used to calculate the overall total water body dissipation rate (k_{wt}) were developed using the following equations [i.e., See Tables B-4-18, B-4-19, B-4-20, B-4-21, and B-4-22, respectively, from USEPA, 2005]:

$$k_v = \frac{K_v}{d_z \cdot (1 + Kd_{sw} \cdot TSS \cdot UCF)}$$

[Equation 5-27]

where:

- k_v = Water column volatilization rate constant (yr^{-1});
- K_v = Overall transfer rate (m/yr);
- d_z = Total water body depth (m);

- $K_{d_{sw}}$ = Suspended sediment/surface water partition coefficient (L/kg);
 TSS = Total suspended solids (mg/L); and
 UCF = Units conversion factor (10^6 kg/mg).

$$K_v = \left[K_L^{-1} + \left(K_G \cdot \frac{H}{R \cdot T_{wk}} \right)^{-1} \right]^{-1} \cdot \theta^{(T_{wk} - 293)}$$

[Equation 5-28]

where:

- K_v = Overall transfer rate (m/yr);
 K_L = Liquid phase transfer coefficient (m/yr);
 K_G = Gas phase transfer coefficient (m/yr);
 H = Henry's law constant (atm·m³/mol);
 R = Universal gas constant (atm·m³/mol·°K);
 T_{wk} = Water body temperature (°K); and
 θ = Temperature correction factor (unitless).

$$K_L = \sqrt{\frac{10^{-4} \cdot D_w \cdot u}{d_z}} \cdot UCF \quad (\text{Flowing Streams/Rivers})$$

[Equation 5-29]

where:

- K_L = Liquid phase transfer coefficient (m/yr);
 D_w = Diffusivity of COPC in water (cm²/s);
 u = Current velocity (m/s);
 d_z = Total water body depth (m); and
 UCF = Units conversion factor (3.1536×10^7 s/yr).

$$K_G = (C_d^{0.5} \cdot W) \cdot \frac{k^{0.33}}{\lambda_z} \cdot \left(\frac{\mu_a}{\rho_a \cdot D_a} \right)^{-0.67} \cdot UCF \quad (\text{Quiescent Lakes/Ponds})$$

or

$$K_G = 36,500 \quad (\text{Flowing Streams/Rivers})$$

[Equation 5-30]

where:

- K_G = Gas phase transfer coefficient (m/yr);
 C_d = Drag coefficient (unitless);
 W = Average annual wind speed (m/s);
 k = Von Karman's constant (unitless);
 λ_z = Dimensionless viscous sublayer thickness (unitless);
 μ_a = Viscosity of air (g/cm-s);
 ρ_a = Density of air (g/cm³);
 D_a = Diffusivity of COPC in air (cm²/s); and
 UCF = Units conversion factor (3.1536×10^7 s/yr).

$$k_b = \left(\frac{X_e \cdot A_L \cdot SD \cdot UCF_1 - Vf_x \cdot TSS}{A_w \cdot TSS} \right) \times \left(\frac{TSS \cdot UCF_2}{C_{BS} \cdot d_{bs}} \right)$$

[Equation 5-31]

where:

k_b	=	Benthic burial rate constant (yr^{-1});
X_e	=	Unit soil loss ($kg/m^2/yr$);
A_L	=	Watershed area receiving fallout (m^2);
SD	=	Watershed sediment delivery ratio (unitless);
UCF_1	=	Units conversion factor (10^3 g/kg);
Vf_x	=	Average volumetric flow rate through water body (m^3/yr);
TSS	=	Total suspended solids (mg/L or g/m^3);
A_w	=	Water body surface area (m^2);
C_{BS}	=	Benthic solids concentration (kg/L);
d_{bs}	=	Depth of upper benthic layer (m); and
UCF_2	=	Units conversion factor (10^{-6} kg/mg).

Total suspended solids (TSS) is a parameter used in several of the previous equations. As there are apparently no TSS data for Kiewassee Lake, the MDEQ indicated that two default values (i.e., 2 and 10 mg/L) should be considered for the current HHRA. As discussed in later sections, use of the 10 mg/L TSS value resulted in higher fish consumption risks; as a consequence, results for Kiewassee Lake presented throughout the remainder of the HHRA are based on use of the 10 mg/L TSS value. The values for f_{wc} , k_{wt} , k_v , K_v , K_L , K_G and k_b , calculated for Kiewassee Lake, are summarized in Tables 5-25 through 5-31, respectively.

The total water column COPC concentration (C_{wctot}), summarized in Table 5-32, includes values for both the dissolved chemical and chemical sorbed to suspended solids. It was calculated using the following equation [i.e., See Table B-4-23 from USEPA, 2005]:

$$C_{wctot} = f_{wc} \cdot C_{wtot} \cdot \frac{d_{wc} + d_{bs}}{d_{wc}}$$

[Equation 5-32]

where:

C_{wctot}	=	Total COPC concentration in water column (mg/L);
f_{wc}	=	Fraction of total water body COPC concentration that occurs in water column (unitless);
C_{wtot}	=	Total COPC concentration in surface water system, including water column and bed sediment (mg/L);
d_{wc}	=	Total depth of water column (m); and
d_{bs}	=	Depth of the upper benthic layer (m).

Table 5-33 shows the concentration of COPCs dissolved in the water column (C_{dww}). It was calculated using the following equation [i.e., See Table B-4-24 from USEPA, 2005]:

$$C_{dw} = \frac{C_{wctot}}{1 + Kd_{sw} \cdot TSS \cdot UCF}$$

[Equation 5-33]

where:

- C_{dw} = COPC water concentration in the dissolved phase (mg/L);
- C_{wctot} = Total water column COPC concentration (mg/L);
- Kd_{sw} = Suspended sediment/surface water partition coefficient (L/kg);
- TSS = Total suspended solids (mg/L); and
- UCF = Units conversion factor (10^{-6} kg/mg).

As discussed earlier, the approach followed in the MME HHRA assumes that a small amount of methylmercury is formed from the divalent mercury present in watershed soils. However, the current HHRA guidance also assumes that the modeled C_{dw} value for divalent mercury (Hg^{+2}) should itself be apportioned into divalent mercury (85-percent) and methylmercury (15-percent) (USEPA, 2005). In Table 5-33, this results in a C_{dw} for divalent mercury of $1.5E-8$ mg/L (i.e., 85-percent of the C_{dw} for Hg^{+2}) and a C_{dw} for methylmercury of $3.2E-9$ mg/L (i.e., the modeled C_{dw} for methylmercury plus 15-percent of the C_{dw} for Hg^{+2}).

The concentration of COPCs sorbed to bed sediments (C_{sb}) is calculated using the following equation [i.e., See Table B-4-25 from USEPA, 2005]:

$$C_{sb} = f_{bs} \cdot C_{wtot} \cdot \frac{Kd_{bs}}{\theta_{bs} + Kd_{bs} \cdot C_{BS}} \cdot \frac{d_{wc} + d_{bs}}{d_{bs}}$$

[Equation 5-34]

where:

- C_{sb} = Modeled COPC concentration sorbed to bed sediments (mg/kg);
- f_{bs} = Fraction of total water body COPC concentration that occurs in the benthic sediment (unitless);
- C_{wtot} = Total COPC concentration in surface water system, including water column and bed sediment (mg/L);
- Kd_{bs} = Bed sediment/sediment pore water partition coefficient (L/kg);
- θ_{bs} = Bed sediment porosity (unitless);
- d_{wc} = Total depth of water column (m);
- d_{bs} = Depth of the upper benthic layer (m); and
- C_{BS} = Bed sediment concentration (g/cm^3).

The modeled concentrations of COPCs sorbed to bed sediments (C_{sb}) resulting from MME facility emissions are summarized in Table 5-34.

5.5.4. Modeled COPC Concentrations in Aboveground Produce

Above-ground plants are defined as those plants whose edible portion is above the ground surface (USEPA 2005).

COPCs may be transported to above-ground plants through direct deposition of particulates onto exposed plant surfaces, by transfer of vapor directly into the plant tissue (i.e., air-to-leaf transfer), and through the uptake of COPCs in soil through the roots.

The concentration of COPCs in above-ground plants due to direct deposition is estimated using the following equation [i.e., See Table B-2-7 from USEPA, 2005]:

$$Pd = \frac{UCF \cdot Q \cdot (1 - F_v) \cdot [Dydp + (Fw \cdot Dywp)] \cdot Rp \cdot [1.0 - \exp(-kp \cdot Tp)]}{Yp \cdot kp}$$

[Equation 5-35]

where:

- Pd = COPC concentration in plant due to direct deposition (mg/kg DW);
- Q = COPC stack emission rate (g/s);
- Dydp = Normalized annual dry deposition flux-particle phase COPCs (s/m²-yr);
- Dywp = Normalized annual wet deposition flux-particle phase COPCs (s/m²-yr);
- Fw = Fraction of wet deposition that adheres to plant surfaces (unitless);
- Fv = COPC fraction in vapor phase (unitless);
- Rp = Interception fraction of the edible portion of plant tissue (unitless);
- kp = Plant surface loss coefficient (yr⁻¹);
- Tp = Length of plant's exposure to deposition per harvest (yr);
- Yp = Yield of edible portion of the plant (kg DW/m²); and
- UCF = Units conversion factor (10³ mg/g).

Again, the nature of the air modeling conducted for the MME HHRA requires that Equation 5-10 be slightly modified to account for actual/estimated COPC emission rates being modeled rather than the default unit (i.e., 1 g/s) emission rate. This modification results in the following equation:

$$Pd = \frac{UCF \cdot [Dydp + (Fw \cdot Dywp)] \cdot Rp \cdot [1.0 - \exp(-kp \cdot Tp)]}{Yp \cdot kp}$$

[Equation 5-36]

where:

- Pd = COPC concentration in plant due to direct deposition (mg/kg DW);
- Dydp = Annual dry deposition flux-particle phase COPCs (g/m²-yr);
- Dywp = Annual wet deposition flux-particle phase COPCs (g/m²-yr);
- Fw = Fraction of wet deposition that adheres to plant surfaces (unitless);
- Rp = Interception fraction of the edible portion of plant tissue (unitless);
- kp = Plant surface loss coefficient (yr⁻¹);
- Tp = Length of plant's exposure to deposition per harvest (yr);
- Yp = Yield of edible portion of the plant (kg DW/m²); and
- UCF = Units conversion factor (10³ mg/g).

For the modeling of mercury, Equation 5-36 is also normally used to calculate Pd and, consistent with current USEPA guidance, the resulting Pd value is then apportioned into divalent mercury (78-percent) and methyl

mercury (22-percent). However, as described earlier, particle bound mercury is not expected to be present in MME emissions. Tables 5-35 and 5-36 summarize the modeled concentrations of lead and 2,3,7,8-TCDD in plants resulting from direct deposition at the selected residence and farm locations, respectively.

The concentration of COPCs in above-ground plants due to direct uptake of vapor phase chemical (Pv) is estimated using the following equation [i.e., See Table B-2-8 from USEPA, 2005]:

$$P_v = Q \cdot F_v \cdot \frac{C_{yv} \cdot B_v \cdot V_{G_{ag}}}{\rho_a}$$

[Equation 5-37]

where:

Pv	=	COPC concentration in plant due to air-to-plant transfer (mg/kg DW);
Q	=	COPC stack emission rate (g/s);
Fv	=	COPC fraction in vapor phase (unitless);
Cyv	=	Normalized air concentration-vapor phase COPC ($\mu\text{g}\cdot\text{s}/\text{g}\cdot\text{m}^3$);
V _{G_{ag}}	=	Empirical correction factor for above-ground plants (unitless);
Bv	=	Air-plant biotransfer factor ([mg COPC/kg plant DW]/[μg COPC/g air]);
ρ_a	=	Density of air (g/m^3).

Again, the nature of the air modeling conducted for the MME HHRA requires that Equation 5-37 be slightly modified to account for estimated COPC emission rates being modeled rather than the default unit (i.e., 1 g/s) emission rate. This modification results in the following equation:

$$P_v = \frac{C_{yv} \cdot B_v \cdot V_{G_{ag}}}{\rho_a}$$

[Equation 5-38]

where:

Pv	=	Concentration of COPC in plant due to air-to-plant transfer (mg/kg DW);
Fv	=	COPC fraction in vapor phase (unitless);
Cyv	=	Air concentration-vapor phase COPC ($\mu\text{g}/\text{m}^3$);
Bv	=	Air-plant biotransfer factor ([mg COPC/kg plant DW]/[μg COPC/g air]);
V _{G_{ag}}	=	Empirical correction factor for above-ground plants (unitless); and
ρ_a	=	Density of air (g/m^3).

For the modeling of mercury, Equation 5-38 was also used to calculate Pv, however, consistent with current USEPA guidance, the resulting Pv value was then apportioned into divalent mercury (78-percent) and methyl mercury (22-percent). Tables 5-37 and 5-38 summarize the modeled COPC concentrations in plants resulting from air-to-plant transfer at the selected residence and farm locations, respectively.

The concentration of COPCs in above-ground plants due to direct uptake from soil (Pr) was estimated using the following equation [i.e., See Table B-2-9 from USEPA, 2005]:

$$Pr_{ag} = Cs \cdot Br_{ag}$$

[Equation 5-39]

where:

- Pr_{ag} = COPC concentration in above-ground plant due to direct uptake from soil (mg/kg DW);
- Cs = Soil COPC concentration in tilled soil (mg/kg); and
- Br_{ag} = Plant-soil bioconcentration factor for above-ground produce ([mg COPC/kg plant DW]/[mg COPC/kg soil]).

Tables 5-39 and 5-40 summarize the modeled COPC concentrations in above-ground plants resulting from uptake from tilled soil at the selected residence and farm locations, respectively.

5.5.5. Modeled COPC Concentrations in Belowground Produce

Below-ground plants are defined as those plants, such as carrots and potatoes, whose edible portion is below the ground surface, (USEPA 2005). Contamination of below-ground plants via direct deposition of particles and vapor transfer is not considered, because the root (or tuber) is protected from direct contact with airborne COPCs. Below-ground plant COPC concentrations depend on the tilled soil concentrations at the locations of interest and are estimated using the following equation [i.e., See Table B-2-10 from USEPA, 2005]:

$$Pr_{bg} = Cs \cdot Br_{rveg} \cdot VG_{rveg}$$

[Equation 5-40]

where:

- Pr_{bg} = COPC concentration in below-ground plant due to direct uptake from soil (mg/kg DW);
- Cs = Soil COPC concentration (mg/kg);
- Br_{rveg} = Plant-soil bioconcentration factor = RCF/Kd_s produce ([mg COPC/kg plant DW]/[mg COPC/kg soil]);
- RCF = Root concentration factor (unitless);
- VG_{rveg} = Empirical correction factor for below-ground plants (unitless); and
- Kd_s = Soil-water partition coefficient (cm^3/g).

Under current HHRA guidance (USEPA, 2005), a below-ground vegetable correction factor (VG_{rveg}) of 0.01 is used in the calculation of Pr_{bg} for all organic COPCs with $\log K_{ow}$ values equal to or greater than 4. A correction value of 1.0 is used for organic COPCs with $\log K_{ow}$ less than 4 and for metals. Tables 5-41 and 5-42 summarize the modeled COPC concentrations in below-ground plants resulting from uptake from tilled soil at the selected residence and farm locations, respectively.

5.5.6. Modeled COPC Concentrations in Beef and Milk

COPCs may transport to and bioaccumulate in beef cattle or milk cows via incidental soil ingestion and ingestion of affected aboveground forage, silage, and grain. COPC concentrations in soil at the selected farm location are summarized in Table 5-7. COPC concentrations in animal feed (i.e., silage, forage, and grain) were estimated using the approaches described for above-ground plants in Section 5.5.4. Individual pathway results are described in Tables 5-43 through 5-45 and summarized in Tables 5-46 and 5-47 based on potential carcinogenic and noncarcinogenic endpoints, respectively. These soil and feed concentrations, as well as estimates of soil and plant intake and chemical-specific biotransfer factors, were used to derive COPC concentrations in beef and milk.

COPC concentrations in beef tissue (A_{beef}) were estimated using the following equation [i.e., See Table B-3-10 from USEPA, 2005]:

$$A_{beef} = (\sum (F_i \cdot Qp_i \cdot P_i) + (Qs \cdot Cs \cdot Bs)) \cdot Ba_{beef} \cdot MF \quad \text{[Equation 5-41]}$$

where:

- A_{beef} = Modeled COPC concentration in beef (mg/kg FW);
- F_i = Fraction of ingested plant material grown on impacted soil (unitless);
- Qp_i = Daily quantity of plant consumed (kg DW/d);
- P_i = COPC concentration in plant type 'i' consumed by beef cattle (mg/kg DW);
- Qs = Daily quantity of untilled soil consumed (kg/d);
- Cs = COPC concentration in untilled soil (mg/kg);
- Bs = Soil bioavailability factor (unitless);
- MF = Metabolism factor (unitless); and
- Ba_{beef} = Biotransfer factor for beef (d/kg FW).

COPC concentrations in milk (A_{milk}) were estimated using the following equation [i.e., See Table B-3-11 from USEPA, 2005]:

$$A_{milk} = (\sum (F_i \cdot Qp_i \cdot P_i) + (Qs \cdot Cs \cdot Bs)) \cdot Ba_{milk} \cdot MF \quad \text{[Equation 5-42]}$$

where:

- A_{milk} = Modeled COPC concentration in milk (mg/kg FW);
- F_i = Fraction of ingested plant material grown on impacted soil (unitless);
- Qp_i = Daily quantity of plant consumed (kg DW/d);
- P_i = COPC concentration in plant type 'i' consumed by dairy cattle (mg/kg DW);
- Qs = Daily quantity of untilled soil eaten by animal (kg/d);
- Cs = COPC concentration in untilled soil (mg/kg);
- Bs = Soil bioavailability factor (unitless);
- MF = Metabolism factor (unitless); and
- Ba_{milk} = Biotransfer factor for milk (d/kg FW).

In the above equations, a metabolism factor (MF) is used to account for the enhanced degradation of selected COPCs (i.e., bis(2-ethyl- hexyl)phthalate) by mammalian species. In the current HHRA, a MF of 1.0 has been used for all COPCs.

A summary of the exposure parameters used to estimate COPC concentrations in beef and milk is provided in Table 5-48. Modeled concentrations of COPCs in beef resulting from MME emissions are summarized in Tables 5-49 and 5-50 for carcinogenic and noncarcinogenic endpoints, respectively. Modeled concentrations of COPCs in milk resulting from MME emissions are summarized in Tables 5-51 and 5-52 for carcinogenic and noncarcinogenic endpoints, respectively.

5.5.7. Modeled COPC Concentrations in Pork

COPC concentrations in pork are estimated on the basis of the amount of COPCs pigs are assumed to consume through ingestion of silage, grain, and surface soil (USEPA, 2005). The concentration of COPCs in pork (A_{pork}) is calculated using the following equation [i.e., See Table B-3-12 from USEPA, 2005]:

$$A_{pork} = (\sum (F_i \cdot Qp_i \cdot P_i) + (Qs \cdot Cs \cdot Bs)) \cdot Ba_{pork} \cdot MF$$

[Equation 5-43]

where:

A_{pork}	=	Modeled COPC concentration in pork (mg/kg FW);
F_i	=	Fraction of ingested plant material grown on impacted soil (unitless);
Qp_i	=	Daily quantity of plant consumed (kg DW/d);
P_i	=	Concentration of COPC in plant type 'i' consumed by pigs (mg/kg DW);
Qs	=	Daily quantity of untilled soil consumed (kg/d);
Cs	=	COPC concentration in untilled soil (mg/kg);
Bs	=	Soil bioavailability factor (unitless);
MF	=	Metabolism factor (unitless); and
Ba_{pork}	=	Biotransfer factor for pork (d/kg FW).

In the above equation, the metabolism factor (MF) is used to account for the enhanced degradation of selected COPCs (i.e., bis(2-ethyl- hexyl)phthalate) by mammalian species. In the current HHRA, a MF of 1.0 has been used for all COPCs.

A summary of the exposure parameters used to estimate COPC concentrations in pork is provided in Table 5-48. Modeled concentrations of COPCs in pork resulting from MME emissions are summarized in Tables 5-53 and 5-54 for carcinogenic and noncarcinogenic endpoints, respectively.

5.5.8. Modeled COPC Concentrations in Poultry Products

COPC concentrations in poultry (eggs and tissue) are estimated on the basis of the amount of COPCs poultry are assumed to consume through ingestion of grain and surface soil (USEPA, 2005). The concentrations of COPCs in eggs/poultry ($A_{\text{eggs/poultry}}$) were calculated using the following equation [i.e., See Tables B-3-13 and B-3-14 from USEPA, 2005]:

$$A_{\text{eggs/poultry}} = (\sum (F_i \cdot Qp_i \cdot P_i) + (Qs \cdot Cs \cdot Bs)) \cdot Ba_{\text{egg / poultry}} \quad \text{[Equation 5-44]}$$

where:

$A_{\text{eggs/poultry}}$	=	Modeled COPC concentration in eggs/poultry (mg/kg FW);
Qp_i	=	Daily quantity of grain consumed (kg DW/d);
Qs	=	Daily quantity of untilled soil consumed (kg/d);
Cs	=	Untilled soil concentration (mg/kg);
BS	=	Soil bioavailability factor (unitless);
F_i	=	Fraction of grain that is contaminated (unitless);
P_i	=	COPC concentration in grain (mg/kg DW); and
$Ba_{\text{eggs/poultry}}$	=	Biotransfer factor for eggs/poultry (d/kg FW).

A summary of the exposure parameters used to estimate COPC concentrations in eggs/poultry is provided in Table 5-48. Modeled concentrations of COPCs in eggs resulting from MME emissions are summarized in Tables 5-55 and 5-56 for carcinogenic and noncarcinogenic endpoints, respectively. Modeled concentrations of COPCs in poultry meat resulting from MME emissions are summarized in Tables 5-57 and 5-58 for carcinogenic and noncarcinogenic endpoints, respectively.

5.5.9. Modeled COPC Concentrations in Edible Fish Tissue

A concern of the MDEQ is the potential partitioning of COPCs from surface water into fish, which could then be consumed by local recreational fishermen and their families. Although all three of the concentration parameters (C_{wctot} , C_{dw} , and C_{sb}) were calculated for each COPC (See Section 5.5.3), only one value is ultimately selected for the estimation of fish tissue concentrations; its selection is based on chemical-specific parameters of the individual COPC. COPC concentrations in fish are estimated by applying a bioconcentration factor (BCF), a bioaccumulation factor (BAF), or a biota-sediment bioaccumulation factor (BSAF) to the appropriate surface water compartment concentration.

The BCF is used to calculate the COPC concentration in fish based on the dissolved phase water COPC concentration (C_{dw}). The relationship is described by the following equation [i.e., See Table B-4-26 from USEPA, 2005]:

$$C_{\text{fish}} = C_{\text{dw}} \cdot BCF \quad \text{[Equation 5-45]}$$

where:

C_{fish}	=	Modeled COPC concentration in fish (mg/kg FW);
C_{dw}	=	COPC concentration in dissolved phase water (mg/L); and
BCF	=	Bioconcentration factor (L/kg).

The BAF is also used to calculate the COPC concentration in fish based on the dissolved phase water COPC concentration (C_{dw}). The relationship is described by the following equation [i.e., See Table B-4-27 from USEPA, 2005]:

$$C_{fish} = C_{dw} \cdot BAF$$

[Equation 5-46]

where:

C_{fish}	=	Modeled COPC concentration in fish (mg/kg FW);
C_{dw}	=	COPC concentration in dissolved phase water (mg/L); and
BAF	=	Bioaccumulation factor (L/kg FW).

The BSAF is used to calculate the COPC concentration in fish tissue based on the modeled sediment COPC concentration (C_{sb}). This relationship is described by the following equation [i.e., See Table B-4-28 from USEPA, 2005]:

$$C_{fish} = \frac{C_{sb} \cdot f_{lipid} \cdot BSAF}{OC_{sed}}$$

[Equation 5-47]

where:

C_{fish}	=	Modeled COPC concentration in fish (mg/kg FW);
C_{sb}	=	COPC concentration sorbed to bed sediment (mg/kg);
f_{lipid}	=	Fish lipid content (unitless);
OC_{sed}	=	Fraction organic carbon in bottom sediment (unitless); and
BSAF	=	Biota-sediment accumulation factor (unitless).

Modeled concentrations of COPCs in the various surface water compartments and fish tissue resulting from MME facility emissions are summarized in Tables 5-59 and 5-60 based on potential carcinogenic and noncarcinogenic endpoints, respectively.

It is important to note that two variations of Equation 5-46 have been considered in the current HHRA for the modeling of methylmercury concentrations into fish tissue (Table 5-60). The first approach, described in Table B-4-27 of the HHRA guidance (USEPA, 2005), recommends that the dissolved phase water concentration of methylmercury should be multiplied by the BAF for methylmercury as shown in the following equation:

$$C_{fish} = C_{dw(MeHg)} \cdot BAF_{MeHg}$$

[Equation 5-48]

In the second approach, recommended by the MDEQ, fish tissue methylmercury concentrations are estimated using the following relationship derived from the Great Lakes Water Quality Initiative (GLWQI):

$$C_{fish} = C_{wctot(Hg)} \cdot BAF_{MeHg}$$

[Equation 5-49]

The BAFs used to generate the results for methylmercury in Table 5-60 are composite values reflecting a diet consisting of 76-percent trophic level 4 (TL4) fish and 24-percent trophic level 3 (TL3) fish, as recommended by the MDEQ (see Table 5-61). It is important to note that the MDEQ approach, described by Equation 5-49, correlates fish tissue methylmercury concentrations with the total concentration of mercury in the water column (i.e., C_{wctot}). This requires the use of a different set of BAF values (see Table 5-61), although the TL4:TL3 ratio remains the same as in the first approach.

5.6 Quantification of COPC Exposure by Pathway

Estimates of indirect COPC intake or dose are based on the modeled COPC concentrations in media, as described in Section 5.5, and the estimated magnitude of exposure. Potential receptor exposures to COPCs are expressed as lifetime average daily doses (LADDs) and average daily doses (ADDs) (for evaluating carcinogenic effects and non-carcinogenic effects, respectively). LADDs and ADDs are expressed in units of milligrams of COPC per kilogram of body weight per day (mg/kg-day). A summary of the exposure parameters used to estimate COPC intakes in human receptors is provided in Table 5-62. Under current agency guidance (USEPA, 2005), the Resident (Adult and Child) and Fisher (Adult and Child) are assumed to reside at the selected residence location; the Farmer (Adult and Child) is assumed to reside at the selected farm location.

5.6.1 COPC Exposure Resulting from Inhalation

Individuals can be exposed to emitted COPCs by inhalation of ambient air. Under the draft guidance, an approach was provided which permitted the calculation of a daily COPC intake via inhalation; that approach has been abandoned in the current guidance and has been replaced by an approach which utilizes an exposure concentration (EC) which acknowledges receptor differences in exposure patterns. The revised approach is described in greater detail in Section 5.7.

5.6.2 COPC Exposure Resulting from the Incidental Ingestion of Soil

Individuals can be exposed to emitted COPCs by inadvertent hand-to-mouth transfer of surface soil. Daily intake of chemical resulting from the ingestion of soil will be estimated using the following equation [See Table C-1-1 from USEPA, 2005]:

$$I_{soil} = Cs \cdot CR_{soil} \cdot F_{soil}$$

[Equation 5-50]

where:

- I_{soil} = Daily intake of COPCs from soil ingestion (mg/d);
- Cs = Soil COPC concentration (untilled) (mg/kg);
- CR_{soil} = Daily consumption rate of soil (kg/d); and
- F_{soil} = Fraction of consumed soil contaminated (unitless).

The daily intake of COPCs by the Resident/Fisher, Resident Child/Fisher Child, Farmer and Farmer Child resulting from the incidental ingestion of surface soils (I_{soil}) is summarized in Tables 5-63, 5-64, 5-65 and 5-66, respectively.

5.6.3. COPC Exposure Resulting from the Ingestion of Home-Grown Vegetables

The daily intake of COPCs via ingestion of homegrown plants has been calculated using the following equation [See Table C-1-2 from USEPA, 2005]:

$$I_{veg} = [(Pd + Pv + Pr_{ag}) \cdot CR_{ag}] + (Pr_{ag} \cdot CR_{pp}) + (Pr_{bg} \cdot CR_{bg}) \cdot F_{ag}$$

[Equation 5-51]

where:

- I_{veg} = Daily COPC intake from produce ingestion (mg/d);
- Pd = COPC concentration in above-ground produce due to direct deposition (mg/kg DW);
- Pv = COPC concentration in above-ground produce due to air-plant transfer (mg/kg DW);
- Pr_{ag} = COPC concentration in above-ground produce due to root uptake (mg/kg DW);
- Pr_{bg} = COPC concentration in below-ground produce due to root uptake (mg/kg DW);
- CR_{ag} = Daily consumption rate of above-ground produce (kg DW/d);
- CR_{pp} = Daily consumption rate of protected above-ground produce (kg DW/d);
- CR_{bg} = Daily consumption rate of below-ground produce (kg DW/d); and
- F_{ag} = Fraction of produce contaminated (unitless).

The daily intake of COPCs by the Resident/Fisher, Resident Child/Fisher Child, Farmer and Farmer Child resulting from the ingestion of homegrown produce (I_{veg}) is summarized in Tables 5-67, 5-68, 5-69 and 5-70, respectively.

5.6.4. COPC Exposure Resulting from the Ingestion of Beef

The daily intake of COPCs resulting from the ingestion of beef has been estimated using the following equation [See Table C-1-3 from USEPA, 2005]:

$$I_{beef} = A_{beef} \cdot CR_{beef} \cdot F_{beef}$$

[Equation 5-52]

where:

I_{beef}	=	Daily intake of COPC from beef ingestion (mg/d);
A_{beef}	=	COPC concentration in beef (mg/kg FW);
CR_{beef}	=	Daily consumption rate of beef (kg FW/d); and
F_{beef}	=	Fraction of beef contaminated (unitless).

The daily intake of COPCs in the Farmer and Farmer Child resulting from the incidental ingestion of homegrown beef (I_{beefs}) is summarized in Tables 5-71 and 5-72, respectively.

5.6.5. COPC Exposure Resulting from the Ingestion of Milk

The daily intake of COPCs resulting from the ingestion of milk has been estimated using the following equation [See Table C-1-3 from USEPA, 2005]:

$$I_{\text{milk}} = A_{\text{milk}} \cdot CR_{\text{milk}} \cdot F_{\text{milk}} \quad \text{[Equation 5-53]}$$

where:

I_{milk}	=	Daily intake of COPC from milk ingestion (mg/d);
A_{milk}	=	COPC concentration in milk (mg/kg FW);
CR_{milk}	=	Daily consumption rate of milk (kg FW/d); and
F_{milk}	=	Fraction of milk contaminated (unitless).

The daily intake of COPCs in the Farmer and Farmer Child resulting from the incidental ingestion of homegrown milk (I_{milk}) is also summarized in Tables 5-71 and 5-72, respectively.

5.6.6. COPC Exposure Resulting from the Ingestion of Pork

The daily intake of COPCs resulting from the ingestion of pork has been estimated using the following equation [See Table C-1-3 from USEPA, 2005]:

$$I_{\text{pork}} = A_{\text{pork}} \cdot CR_{\text{pork}} \cdot F_{\text{pork}} \quad \text{[Equation 5-54]}$$

where:

I_{pork}	=	Daily intake of COPC from pork ingestion (mg/d);
A_{pork}	=	COPC concentration in pork (mg/kg FW);
CR_{pork}	=	Daily consumption rate of pork (kg FW/d); and
F_{pork}	=	Fraction of pork contaminated (unitless).

The daily intake of COPCs in the Farmer and Farmer Child resulting from the incidental ingestion of homegrown pork (I_{pork}) is summarized in Tables 5-73 and 5-74, respectively.

5.6.7. COPC Resulting from the Ingestion of Poultry Products

The daily intake of COPCs resulting from the ingestion of poultry products has been estimated using the following equation [See Table C-1-3 from USEPA, 2005]:

$$I_{poultry/egg} = A_{poultry/egg} \cdot CR_{poultry/egg} \cdot F_{poultry/egg}$$

[Equation 5-55]

where:

$I_{poultry/egg}$	=	Daily intake of COPC from poultry/egg ingestion (mg/d);
$A_{poultry/egg}$	=	COPC concentration in poultry/egg (mg/kg FW);
$CR_{poultry/egg}$	=	Daily consumption rate of poultry/egg (kg FW/d); and
$F_{poultry/egg}$	=	Fraction of poultry/egg contaminated (unitless).

The daily intake of COPCs in the Farmer and Farmer Child resulting from the incidental ingestion of homegrown poultry products ($I_{poultry/egg}$) is summarized in Tables 5-75 and 5-76, respectively.

5.6.8. COPC Exposure Resulting from the Ingestion of Fish

The Fisher and Fisher Child are the only receptors to consume locally-caught fish. The daily intake of COPCs resulting from the ingestion of fish taken from a nearby surface water body has been estimated using the following equation [See Table C-1-4 from USEPA, 2005]:

$$I_{fish} = C_{fish} \cdot CR_{fish} \cdot F_{fish}$$

[Equation 5-56]

where:

I_{fish}	=	Daily intake of COPC from fish ingestion (mg/kg-d);
C_{fish}	=	COPC concentration in fish (mg/kg FW);
CR_{fish}	=	Daily consumption rate of fish (kg FW/d); and
F_{fish}	=	Fraction of fish contaminated (unitless).

The daily intake of COPCs in the Fisher and Fisher Child resulting from the ingestion of fish (I_{fish}) is summarized in Tables 5-77 and 5-78, respectively. Note that two different I_{fish} values are presented in each of those tables, a reflection of the two different approaches (i.e., USEPA and MDEQ) considered in the current HHRA.

5.6.9. COPC Exposure Resulting from the Ingestion of Drinking Water

The daily intake of COPCs resulting from the ingestion of drinking water has not been included in the default exposure scenarios, as there is no evidence that surface water within 10-km of the MME facility is used as a source of potable water.

5.7 Quantification of COPC Exposures by Receptor Group

Direct Exposures

As described earlier, all receptors can be exposed to airborne COPCs through the direct (inhalation) exposure pathway. The Resident and Fisher (both adult and child) are exposed at the targeted residence location; the Farmer (both adult and child) is (are) exposed to airborne COPCs at the targeted farm location.

Although previous guidance recommended that direct inhalation exposures be expressed as a daily COPC dose, the current guidance recommends converting modeled air concentrations of COPCs at a target receptor location to an exposure concentration (EC). The EC, which considers both exposure frequency and exposure duration, is calculated for both carcinogens and non-carcinogens using the following equation [See Tables C-2-1 and C-2-2 from USEPA, 2005]:

$$EC = \frac{C_a \cdot EF \cdot ED}{AT \cdot UCF_1}$$

[Equation 5-57]

where:

EC	=	Exposure concentration ($\mu\text{g}/\text{m}^3$);
C_a	=	COPC air concentration ($\mu\text{g}/\text{m}^3$);
EF	=	Exposure frequency (d/yr);
ED	=	Exposure duration (yr);
AT	=	Averaging time (yr); and
UCF_1	=	Units correction factor (365 d/yr).

Indirect Exposures

The combined daily intake of COPCs resulting from indirect exposure pathways is obtained by summing the appropriate contributions from the ingestion of surface soil, home-grown garden produce, locally-raised beef, milk, pork, eggs, poultry, and fish. The total intake for each receptor is described in the following sections. The total estimated daily intake of COPCs through the appropriate pathways is then used in the determination of indirect risk to exposed receptors. As described previously, for each receptor scenario evaluated, the estimation of potential carcinogenic risk is based on the lifetime average daily dose (LADD); the estimation of potential non-carcinogenic risk is based on the average daily dose (ADD). The Lifetime Average Daily Dose (LADD) is calculated using the following equation:

$$LADD = \frac{I_{\text{Medium}} \cdot EF \cdot ED}{BW \cdot AT_C \cdot UCF}$$

[Equation 5-58]

where:

LADD	=	Lifetime average daily dose (mg/kg-d);
I_{Medium}	=	Daily COPC intake through exposure to impacted medium (mg/d);
ED	=	Exposure duration (yr);
EF	=	Exposure frequency (d/yr);
BW	=	Body weight (kg);
AT_C	=	Averaging time, carcinogens (yr); and
UCF	=	Unit conversion factor (365 d/yr).

The average daily dose (ADD) for non-carcinogenic endpoints is calculated using the following equation:

$$ADD = \frac{I_{\text{Medium}} \cdot EF \cdot ED}{BW \cdot AT_{\text{NC}} \cdot UCF}$$

[Equation 5-59]

where:

ADD	=	Average daily dose (mg/kg-d);
I_{Medium}	=	Daily COPC intake through exposure to impacted medium (mg/d);
ED	=	Exposure duration (yr);
EF	=	Exposure frequency (d/yr);
BW	=	Body weight (kg);
AT_{NC}	=	Averaging time (yr) (Note: $AT=ED$); and
UCF	=	Unit conversion factor (365 d/yr).

5.7.1. COPC Exposure in the Resident and Resident Child

5.7.1.1. Direct Exposures

COPC exposure concentrations (ECs) are summarized in Tables 5-79 and 5-80, respectively, for the Resident and Resident Child at the selected residence location. It must be noted that values are provided for both carcinogenic and non-carcinogenic effects, a consequence of the different averaging times used in the calculation of ECs.

5.7.1.2. Indirect Exposures

Under current HHRA guidance (USEPA, 2005), the Resident and Resident Child are exposed (via indirect pathways) to COPCs through the ingestion of soil, drinking water (where applicable), and homegrown produce. The total daily intake of COPCs via all indirect pathways is described by the following equation [See Table C-1-6 from USEPA, 2005]:

$$I_{\text{tot}} = I_{\text{soil}} + I_{\text{dw}} + I_{\text{veg}}$$

[Equation 5-60]

where:

I_{tot}	=	Total indirect daily COPC intake (mg/d);
I_{soil}	=	Daily intake of COPC from ingestion of soil (mg/d);
I_{dw}	=	Daily intake of COPC from ingestion of drinking water (mg/d); and
I_{veg}	=	Daily intake of COPC from ingestion of produce (mg/d).

The total indirect daily intakes of COPCs in the Resident and Resident Child resulting from the incidental ingestion of soil, the ingestion of drinking water and the ingestion of homegrown plants are summarized in Tables 5-81 and 5-82, respectively. As discussed earlier, exposure to COPCs through the ingestion of drinking water is not considered to be a complete exposure pathway in the MME HHRA, therefore, I_{dw} is set equal to zero. It should be noted that values are provided based on both carcinogenic and non-carcinogenic endpoints.

5.7.2. COPC Exposure in the Farmer and Farmer Child

5.7.2.1. Direct Exposures

COPC exposure concentrations (ECs) are summarized in Tables 5-83 and 5-84, respectively, for the Farmer and Farmer Child at the selected farm location. It should be noted that values are provided for both carcinogenic and non-carcinogenic effects, a consequence of the different averaging times used in the calculation of ECs.

5.7.2.2. Indirect Exposure

Under current guidance, the Farmer and the Farmer Child can be exposed (via indirect pathways) to COPCs through the ingestion of soil, homegrown produce, beef, milk, pork, and poultry products at the selected farm location. As discussed previously, consumption of surface water as a source of potable water is not a complete exposure pathway in the current HHRA. The total daily COPC intake for the Farmer and Farmer Child, via all indirect pathways, is described by the following equation [See Table C-1-6 from USEPA, 2005]:

$$I_{tot} = I_{soil} + I_{dw} + I_{veg} + I_{beef} + I_{milk} + I_{pork} + I_{eggs} + I_{poultry}$$

[Equation 5-61]

where:

I_{tot}	=	Total indirect daily COPC intake (mg/d);
I_{soil}	=	Daily intake of COPC from ingestion of soil (mg/d);
I_{dw}	=	Daily intake of COPC from ingestion of drinking water (mg/d);
I_{veg}	=	Daily intake of COPC from ingestion of produce (mg/d);
I_{beef}	=	Daily intake of COPC from ingestion of beef (mg/d);
I_{milk}	=	Daily intake of COPC from ingestion of milk (mg/d);
I_{pork}	=	Daily intake of COPC from ingestion of pork (mg/d);
I_{eggs}	=	Daily intake of COPC from ingestion of eggs (mg/d); and
$I_{poultry}$	=	Daily intake of COPC from ingestion of poultry (mg/d).

Indirect pathway COPC intakes by the Farmer resulting from exposure to MME facility emissions at the selected farm location are summarized in Tables 5-85 and 5-86. Indirect pathway COPC intakes by the Farmer Child resulting from exposure to MME facility emissions are summarized in Tables 5-87 and 5-88. It should be noted that values are provided based on both carcinogenic and non-carcinogenic endpoints.

5.7.3. COPC Exposure in the Fisher and Fisher Child

5.7.3.1. Direct Exposure

COPC exposure concentrations (ECs) are summarized in Tables 5-89 and 5-90, respectively, for the Fisher and Fisher Child at the selected residence location. It should be noted that values are provided for both carcinogenic and non-carcinogenic effects, a consequence of the different averaging times used in the calculation of ECs.

5.7.3.2. Indirect Exposure

Under current guidance (USEPA, 2005), the Fisher and Fisher Child are exposed (via indirect pathways) to

COPCs through the ingestion of soil, drinking water (where applicable), homegrown produce, and fish. The total daily intake via all indirect pathways is described by the following equation [See Table C-1-6 from USEPA, 2005]:

$$I_{tot} = I_{soil} + I_{dw} + I_{veg} + I_{fish}$$

[Equation 5-62]

where:

- I_{tot} = Total indirect daily COPC intake (mg/d);
- I_{soil} = Daily intake of COPC from ingestion of soil (mg/d);
- I_{dw} = Daily intake of COPC from ingestion of drinking water (mg/d);
- I_{veg} = Daily intake of COPC from ingestion of produce (mg/d); and
- I_{fish} = Daily intake of COPC from ingestion of fish (mg/d).

The total indirect daily intakes of COPCs in the Fisher and Fisher Child resulting from the incidental ingestion of soil, the ingestion of drinking water, the ingestion of homegrown plants and the ingestion of fish are summarized in Tables 5-91 and 5-92, respectively. As discussed earlier, exposure to COPCs through the ingestion of drinking water is not considered to be a complete exposure pathway in the MME HHRA, therefore, I_{dw} is set equal to zero. It should be noted that values are provided based on both carcinogenic and non-carcinogenic endpoints.

5.7.4. Dioxin/Furan Exposure in the Nursing Infant

Consistent with current USEPA guidance, exposure of the breast-feeding infant to dioxins/furans has been evaluated in the current HHRA. Exposures have been calculated in a different manner than for the other receptors. COPC exposure for the nursing infant is assumed to occur only through the ingestion of mother's milk; no other exposure pathways are considered. To estimate the COPC concentration in milk, however, maternal exposure is assumed to occur through those adult exposure pathways that are specific to the particular default receptor scenario (i.e., Resident, Farmer, and Fisher). Estimated average daily maternal intakes of 2,3,7,8-TCDD resulting from MME facility emissions, under the default receptor scenarios, are provided in Table 5-93. Not surprisingly, maternal 2,3,7,8-TCDD intake is estimated to be highest under the Farmer scenario.

The resulting concentration of 2,3,7,8-TCDD in breast milk is calculated using the following equation [See Table C-3-1 from USEPA, 2005]:

$$C_{milkfat} = \frac{m \cdot UCF_1 \cdot h \cdot f_1}{0.693 \cdot f_2}$$

[Equation 5-63]

where:

- $C_{milkfat}$ = Modeled dioxin concentration in milk fat of breast milk (pg/kg milkfat);
- m = Average maternal intake of dioxin for each adult scenario (mg/kg-d);
- UCF_1 = Units conversion factor (10^9 pg/mg);
- h = half-life of dioxin in adults (d);

f_1 = Fraction of ingested dioxin stored in fat (); and
 f_2 = Fraction of mother's weight that is fat ().

Default parameter values used in the estimation of dioxin exposure through breast milk are summarized in Table 5-62. Estimated 2,3,7,8-TCDD concentrations in maternal breast milk (fat) for the Resident, Farmer, and Fisher exposure scenarios are shown in Table 5-94.

The average daily dioxin dose for the nursing infant is calculated using the following equation [See Table C-3-2 from USEPA, 2005]:

$$ADD_{infant} = \frac{C_{milkfat} \cdot f_3 \cdot f_4 \cdot IR_{milk} \cdot ED}{BW_{infant} \cdot AT}$$

[Equation 5-64]

where:

ADD_{infant} = Average daily dioxin dose for infant via breast milk (pg/kg-d);
 $C_{milkfat}$ = Dioxin concentration in milk fat of breast milk (pg/kg milkfat);
 f_3 = Fraction of breast milk that is fat ();
 f_4 = Fraction of ingested dioxin that is absorbed ();
 IR_{milk} = Daily ingestion rate of breast milk (kg/d);
 ED = Exposure duration (yr);
 BW_{infant} = Body weight of infant (kg); and
 AT = Averaging time (yr).

Several of the parameters used to evaluate potential exposure of the breast-feeding infant to dioxin/furan congeners have changed in the 2005 guidance. The recommended ingestion rate for breast milk is now 0.688 kg/day, and the recommended infant body weight is now 9.4 kg [See Table C-3-2 from USEPA, 2005]. Average daily dioxin dose estimates for breast-feeding infants, resulting from MME facility emissions under the default receptor exposure scenarios, are summarized in Table 5-95.

5.7.5. Lead Exposure in the Developing Child

Exposures to lead are evaluated in a different manner than that just described for mercury. In order to evaluate potential health effects from lead, USEPA developed the integrated exposure uptake biokinetic (IEUBK) model, which utilizes four interrelated modules to estimate blood lead levels in children exposed to lead-contaminated media (USEPA, 1994). These modules include:

- Exposure Component. Uses lead concentrations in environmental media to calculate the amount of lead entering a child's body. The exposure component uses media-specific consumption rates and lead concentrations to estimate media-specific intake rates.

-
- Uptake Component. Uses lead intake into the lungs and digestive tract and considers absorption of lead to calculate the amount of lead that enters a child's bloodstream.
 - Biokinetic Component. Considers the transfer of lead between blood and other body tissues, or elimination of lead from the body in determining a blood lead concentrations.
 - Probability Distribution Component. Shows the probability of a certain outcome (e.g., a blood lead concentration greater than a level of concern in an exposed child based on the model parameters).

In the MME HHRA, IEUBKwin (Version 1-Build 263) has been used to evaluate the potential human health impacts from lead emissions (USEPA, 2005). The IEUBK model is an integrated approach for evaluating multiple lead exposures and, as lead is a naturally-occurring metal, it is important to understand the extent of background lead exposures prior to evaluating the potential impact of the MME facility. As described in the following sections, the contribution of background exposures to blood lead levels was evaluated as the first step. Then, the fate and transport algorithms, described in the previous sections, were used to estimate the change in lead concentrations in environmental media within the selected location resulting from MME facility emissions. Where site-specific media lead concentrations are not available, either area/region-specific values or model default values have been used; default media consumption rates have been used throughout. It was determined that the potential for lead exposure was higher at the selected farm location than at the selected residence location, although the differences were not great.

5.7.5.1. Air Input Data for IEUBK

Default values have been used for the IEUBK operating parameters linked to inhalation exposures (Table 5-96), including time spent outdoors (i.e., 1 to 4 hr/day), ventilation rates (i.e., 2 to 7 m³/day), indoor air lead concentration as a percent of outdoor air concentration (i.e., 30-percent), and percent of inhaled lead absorbed from the lungs (i.e., 32-percent) (USEPA, 2002).

Background Lead Concentrations in Air

Where site-specific air data are not available, USEPA (2002) recommends using a default air lead concentration of 0.1 µg/m³, however, statewide air monitoring data indicate that average ambient air concentrations of lead are probably less than 0.02 µg/m³ (MDEQ, 2005). An evaluation of those data indicates that higher air lead concentrations are associated with major urban areas in the state and, in fact, a default ambient air lead concentration of 0.01 µg/m³ has been reported for Region 122 which includes Midland County (MDEQ, 2005). Therefore, an ambient air lead concentration of 0.01 µg/m³ was considered to be an appropriate background air input value for Midland County in the IEUBK model.

Lead Concentrations in Air Resulting from Operation of the MME Facility

The modeled airborne lead concentration within the selected farm location has been chosen as an input for the IEUBK model (see Table 5-3). That level is based on the AERMOD outputs, as summarized in Table 4-2. An air lead concentration of $0.01 \mu\text{g}/\text{m}^3$ was selected as the air input value for the IEUBK model, based on an incremental air lead concentration of $0.000035 \mu\text{g}/\text{m}^3$ at the selected farm location combined with the $0.01 \mu\text{g}/\text{m}^3$ background value (Table 5-97).

5.7.5.2. Soil and Dust Input Data for IEUBK

Default values have been used for the IEUBK operating parameters linked to soil/dust ingestion exposures (5-96), including soil/dust ingestion rates (i.e., 0.085 to 0.135 g/d), soil ingestion as a percentage of total soil/dust ingestion (i.e., 45-percent), and percent of ingested soil/dust lead absorbed from the gastrointestinal tract (i.e., 30-percent) (USEPA, 2002).

Background Lead Concentrations in Soil/Dust

Where site-specific data are not available, USEPA (2002) recommends a default soil lead concentration of 200 mg/kg, however, the MDEQ has recommended using a soil lead concentration of 21.0 mg/kg as the appropriate background soil lead level for sites like the current study area. That value represents the average background surface soil lead concentration (plus 1 standard deviation) under MDEQ's Residential and Commercial I Part 201 Generic Cleanup Criteria and Screening Levels for Soils (MDEQ, 2006b).

The selection of an input value for background lead concentrations in indoor dust is complicated. In many urban settings deteriorating lead-based paint can yield dust with high levels of lead; that is not believed to be case here. By default, the IEUBK model defaults to 'Multiple Source Analysis' because it assumes that site-specific dust concentrations are not measured. That is the case in the MME HHRA, as algorithms to model the concentrations of chemicals in dust are not included in USEPA's 2005 risk assessment guidance. If there are no other sources contributing to indoor dust lead (such as lead-based paint), the guidance indicates that a default ratio (i.e., 0.70) can be used to relate indoor dust lead to outdoor soil lead (USEPA, 2002); that would yield a background dust lead concentration of 14.7 mg/kg. To account for the contribution of airborne lead to dust lead through deposition USEPA recommends an additive increment of 100 mg/kg in house dust for each μg lead/ m^3 air. Based on a background air lead concentration of $0.01 \mu\text{g}/\text{m}^3$ this would represent an increment of 1 mg/kg resulting in a background dust lead concentration of 15.7 mg/kg.

A second option for the consideration of lead in house dust is to use a default dust lead value of 200 mg/kg, which is believed to be representative of homes with lead paint, where the lead paint is believed to be in good condition. Both options for the consideration of background dust lead concentrations have been evaluated in the MME HHRA. Background soil and dust lead concentrations considered in the HHRA are summarized in Table 5-97.

Lead Concentrations in Soil/Dust Resulting from Operation of the MME Facility

The modeled surface soil lead concentration within the selected farm location was chosen as an input for the IEUBK model (see Table 5-7). Surface soil concentrations, calculated as described in Section 5.5.2, consider contributions from both wet and dry deposition of airborne lead. It should be noted that the current HHRA guidance (USEPA, 2005) recommends using a mixing depth of 2 cm for surface soil; this represents a change from previous guidance that recommended a mixing depth of 1 cm (USEPA, 1998). A lead concentration of 21.04 mg/kg was selected as the soil input value for the IEUBK model, based on an incremental soil lead concentration of 0.037 mg/kg at the farm location combined with the 21.0 mg/kg background value (Table 5-97).

Due to the uncertainty associated with the availability of lead-based paint in rural Midland County, the two different options for evaluating lead dust concentrations, described above, were modeled. Under the first option, the dust lead concentration was estimated to be 15.73 mg/kg. That value was based on the default relationship between soil lead and dust lead (i.e., 0.70×21.04 mg/kg) and the deposition of airborne lead (i.e., 1 mg/kg contribution to dust based on $0.01 \mu\text{g lead}/\text{m}^3$). Under the second option, the dust lead concentration was assumed to be 200 mg/kg. Dust lead concentrations considered in the MME HHRA are summarized in Table 5-97.

5.7.5.3. Dietary Input Data for IEUBK

Default values have been used for the IEUBK operating parameters linked to dietary exposures (Table 5-96), including the gastrointestinal absorption of dietary lead (i.e., 50-percent) (USEPA, 2002).

Background Lead Concentrations in Diet

The model provides an opportunity to include information on that portion of the total lead intake that enters the body through the consumption of food. As area-specific data describing either the concentration of lead in the various dietary sources or consumption patterns are not available, default dietary data found in the guidance (USEPA 2002) have been used. These default values include dietary lead intake estimates as a function of age (i.e., 5.53 to 7.00 $\mu\text{g Pb}/\text{day}$).

Lead Concentrations in Diet Resulting from Operation of the MME Facility

Modeled dietary intakes of lead in the HHRA, resulting from the consumption of homegrown plants and locally grown beef, milk, pork and poultry products (i.e., 0.0167 µg/d), appear to represent a minimal incremental impact on the default dietary intake range. It was added to the default dietary lead intake for each age group (Table 5-97).

5.7.5.4. Drinking Water Input Data for IEUBK

The drinking water input data are divided into two sections: water consumption rates and environmental concentrations. Table 5-96 summarizes those IEUBK operating parameters linked to drinking water exposures. Default values have been used for age-related water consumption rates (i.e., 0.20 to 0.59 L/d) and percent of ingested water lead absorbed from the gut (i.e., 50-percent) (USEPA, 2002).

Background Lead Concentrations in Drinking Water

When entering a water lead concentration, the user has two options: either to use one concentration for all drinking water sources (Option 1), or to use source-dependent values (Option 2). Option 1 was selected for the MME HHRA; however, the levels of lead measured in the Midland City municipal drinking water supply are higher than the default value (i.e., 4 µg/L) found in the guidance (USEPA, 2002). In its 2006 Drinking Water Quality Report, the Midland Municipal Water Treatment Plant reported a 90th percentile lead concentration of 13.0 µg/L in drinking water during 2006 (Midland, 2006); that conservative value has been selected for input in the IEUBK (Table 5-97).

Lead Concentrations in Drinking Water Resulting from Operation of the MME Facility

Area drinking water should not be impacted by lead emissions from the new MME facility. However, as the IEUBK model does consider the consumption of drinking water as a potential source of lead, the background concentration (i.e., 13.0 µg/L) has been used (see Table 5-97).

5.7.5.5. Maternal Input Data for IEUBK

Although IEUBK allows the user to consider the impact of lead transferred from the mother to the fetus *in utero*, the information needed to modify this exposure pathway are limited. Therefore, as shown in Table 5-96, the default value (i.e., maternal blood lead level at childbirth = 2.5 µg/dL) was used without modification (USEPA, 2002).

5.7.5.6. Alternative Source Input Data for IEUBK

IEUBK allows the user to input lead concentrations from sources that are not covered under other input categories. Examples would include the direct ingestion of lead-based paint and the use of cosmetics or home remedies. For the MME HHRA, no alternate sources of lead have been considered.

6.0 TOXICITY ASSESSMENT

The purpose of the toxicity assessment portion of the HHRA is to present sources of toxicity data for the emissions constituents. Toxicity is defined as the ability of a chemical to induce adverse effects at some dosage in biological systems. The purpose of the toxicity assessment is two-fold:

- To identify the adverse health effects that may arise from direct or indirect exposure of humans to the emissions constituents (hazard assessment); and
- To provide an estimate of the quantitative relationship between the magnitude and duration of exposure and the probability or severity of adverse effects (dose-response assessment).

6.1 Categorization of Chemicals as Carcinogens or Non-carcinogens

Both carcinogenic and non-carcinogenic health effects have been evaluated quantitatively for exposures in the current HHRA. The endpoints for these two types of effects are assessed differently because the mechanisms by which chemicals cause cancer are assumed to be fundamentally different from the processes that cause non-carcinogenic effects. The principal difference reflects the assumption that non-carcinogenic effects exhibit a threshold dose below which no adverse effects occur, whereas USEPA assumes no such threshold exists for carcinogenic effects.

As used here, the term carcinogen refers to any chemical for which there is sufficient evidence that exposure may result in continuing uncontrolled cell division (i.e., cancer) in humans or animals. Conversely, the term non-carcinogen refers to any chemical for which the carcinogenic evidence is negative or insufficient. These definitions are under continual review by USEPA and are subject to change as new information becomes available and the weight-of-evidence is modified. Because exposure to some chemicals may result in both carcinogenic and non-carcinogenic effects, both endpoints associated with such a constituent have been evaluated quantitatively in the HHRA, when sufficient toxicity data are available.

6.2 Assessment of Carcinogenic Endpoints

In contrast to non-carcinogenic effects (for which thresholds are assumed to exist), thresholds have not been demonstrated convincingly for many carcinogenic effects. Consequently, most federal regulatory agencies assume that any exposure to a carcinogen entails some finite risk of cancer. However, depending on the potency of a specific carcinogen and the level of exposure, such a risk is likely to be extremely small.

Several mathematical models have been developed to estimate low-dose cancer risks from high-dose cancer bioassays. Consistent with current theories of carcinogenesis, USEPA has selected the linearized multistage

model to estimate toxicity values (USEPA 1989). In applying this model, USEPA uses the 95% upper confidence limit (UCL) of the slope of the dose-response curve to estimate cancer slope factors (CSFs). Using these procedures, regulatory agencies are unlikely to underestimate actual CSFs for humans. CSFs are expressed in terms of risk per milligram per kilogram per day $[(\text{mg}/\text{kg}\text{-day})^{-1}]$. Inhalation cancer risks are estimated through a comparison of the modeled exposure concentration (EC) against COPC-specific inhalation Unit Risk Factors (URFs) expressed as risk per micrograms per cubic meter $[(\mu\text{g}/\text{m}^3)^{-1}]$.

6.3 Assessment of Non-carcinogenic Endpoints

For evaluating non-carcinogenic effects, USEPA defines acceptable exposure levels as those levels to which the human population, including sensitive subgroups, may be exposed without adverse effects during a lifetime or part of a lifetime, incorporating an adequate margin of safety (USEPA 1989). The potential for non-carcinogenic health effects is usually assessed by comparing the estimated average daily intake (i.e., exposure dosage) to a reference dose (RfD). USEPA develops the RfD by identifying the no-observed-adverse-effect level (NOAEL) or lowest-observed-adverse-effect level (LOAEL) in the scientific literature. NOAELs and LOAELs may be derived from either human epidemiological studies or animal studies; however, because human data are often lacking, these levels are usually derived from laboratory animal studies in which relatively high doses are administered.

Uncertainty factors (UFs) are then applied to the NOAELs and LOAELs to compensate for the data limitations inherent in the experiments, in addition to uncertainties associated with extrapolating high-dose animal data to the relatively low-dose environmental exposure situations in humans. RfDs are expressed in units of mg/kg-day. The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily intake to humans (including sensitive subgroups) that should not result in an appreciable risk of deleterious effects. USEPA assigns a qualitative level of confidence (i.e., low, medium, or high) to the study used to derive the toxicity value, database, and RfD.

RfDs are developed for specific exposure routes (i.e., oral, inhalation, etc.). USEPA frequently provides non-carcinogenic toxicity criteria for inhalation exposure as reference air concentrations (RfCs) rather than RfDs. RfCs are derived using the same principles as those for oral RfDs; however, the analysis of inhalation exposures is more complex because of the dynamics of the respiratory system and its diversity across species, and differences in the physicochemical properties of constituents (USEPA 1989). RfCs are expressed as a concentration in air (in milligrams per cubic meter $[\text{mg}/\text{m}^3]$) for continuous 24-hour-per-day exposure.

RfDs are used as reference points for assessing the likelihood that potential adverse health effects would be associated with site-related exposures. Usually, adverse health effects are unlikely to be associated with exposures that are less than the RfD; the likelihood of adverse health effects in a human population increases as the predicted exposures exceed the RfD. However, it is not possible to state definitively that all exposures above the RfD will result in adverse effects.

6.4 Hierarchy of Toxicity Values

In December 2003, the Office of Superfund Remediation and Technology Innovation distributed OSWER Directive 9285.7-53 (USEPA, 2004) updating the hierarchy of sources of human toxicity values. This hierarchy, recommended in the current HHRA guidance (USEPA, 2005), includes the following sources, in order of Agency preference:

- Chronic Toxicity Benchmarks from USEPA's Integrated Risk Information System (IRIS) computer database (USEPA, 2007);
- Provisional Peer-reviewed Toxicity values (PPRTVs) (USEPA, 2004); and
- Other Peer-reviewed Toxicity Values
 - CalEPA chronic Reference Exposure Levels and Unit Risk estimates (CalEPA, 2002)
 - ATSDR chronic Minimum Risk Levels (MRLs) (ATSDR, 2005)
 - Health Effects Assessment Summary Tables (HEAST) (USEPA, 1997b)

Table 6-1 summarizes the chronic toxicity factors selected for use in the MME HHRA. There are several issues of interest to discuss relative to the selected values. There is no RfD value listed for elemental mercury. Even though there is no value listed in the IRIS database, the oral ingestion of elemental mercury is not considered in the USEPA HHRA guidance (USEPA, 2005); inhalation is the only exposure pathway evaluated. Conversely, there is no RfC available for methylmercury; the inhalation exposure pathway is not considered to be a human health concern compared to the oral exposure pathway. As discussed in several previous sections, USEPA has not developed a RfD or RfC for lead due to the apparent absence of threshold behavior in evaluated dose-response assessments.

6.5 Lead Assessment

Oral and inhalation RfDs (RfCs) have not been developed for lead, as available dose-response relationships correlating toxic effects with increasing exposure dose have failed to demonstrate the existence of threshold behavior. As a result, direct and indirect effects of lead cannot be evaluated in the same manner as other non-carcinogenic COPCs (i.e., mercury).

As described earlier, the integrated exposure uptake biokinetic (IEUBK) model provides an opportunity to evaluate the potential for lead to produce adverse impacts on exposed children. The model is based on a set of coupled first-order differential equations that describe changes in various body compartments of the developing child (i.e., 6 to 84 months), in response to changes in lead concentration and intake from various sources (USEPA, 1994). The model uses site-specific data on lead concentrations in air, water, soil, and household dust and average daily intake of lead from diet and from directly ingested paint chips, to estimate blood lead concentrations in children of different ages. In fact, the model estimates a plausible distribution of blood lead concentrations centered on a geometric mean blood lead concentration. From this distribution, the model estimates the risk (i.e., probability) that a child's blood lead concentration will exceed a user-defined level of concern. The Centers for Disease Control (CDC) have established 10 micrograms of lead per deciliter of blood (i.e., $\mu\text{g}/\text{dL}$) as the level of concern (CDC 1991).

7.0 RISK CHARACTERIZATION

7.1 Introduction

In this section, the information developed in the exposure and toxicity assessment sections is combined to obtain estimates of the potential risks posed to human health. Different processes are used to characterize potential risks resulting from exposure to emitted COPCs. Generally, the risk characterization for mercury and 2,3,7,8-TCDD follows the methodology described by USEPA (2005) and those methods are designed to be health-protective and tend to overestimate, rather than underestimate, risk. For lead, the fate and transport methodology described by USEPA (2005) has been used to provide an understanding of media lead concentrations and human exposures. However, because no toxicity criteria exist for lead, an alternate approach (i.e., IEUBK) must be used to evaluate the potential for adverse impacts to occur in exposed individuals.

7.2 Risk Characterization Approach

7.2.1. Potential Carcinogenic Effects

Potential cancer risks resulting from indirect exposure pathways are assessed by multiplying the estimated lifetime average daily dose (LADD) for a given carcinogenic COPC by its Cancer Slope Factor (CSF). This calculated risk is expressed as the incremental probability of an individual developing cancer over a lifetime and is an estimated upper-bound incremental probability. It is calculated using the following equation (USEPA 2005):

$$LICR = LADD \times CSF \quad \text{[Equation 7-1]}$$

where:

- LICR = Excess lifetime incremental cancer risk (unitless);
- LADD = Lifetime average daily dose (mg/kg-day); and
- CSF = Route-specific cancer slope factor (mg/kg-day)⁻¹.

The equations used to estimate potential carcinogenic effects for the inhalation pathway have been updated in the 2005 HHRA guidance. Consistent with these modifications, the current HHRA utilizes the following equation to quantify potential carcinogenic effects resulting from inhalation:

$$LICR_{inh(i)} = EC \cdot URF_{(i)} \quad \text{[Equation 7-2]}$$

where:

- LICR_{inh(i)} = Lifetime incremental cancer risk for direct inhalation of COPC_i (unitless);
- EC = Exposure concentration calculated per Equation 5-57 (µg/m³); and
- URF_(i) = Inhalation unit risk factor [(µg/m³)⁻¹].

Potential cancer risks have been estimated separately for exposure to each COPC and for each exposure pathway. The separate potential cancer risk estimates have then been summed across chemicals and across all

exposure pathways (both direct and indirect) to obtain the total cumulative LICR for the potentially exposed population. Cancer risks are summed because USEPA (1986) considers the cancer potency of simultaneous exposure to low doses of carcinogenic chemicals to be additive, regardless of the chemical's mechanisms of carcinogenicity or sites (i.e., organs) of action. LICRs are summed for each exposure scenario evaluated. (Note that potential cancer risk estimates are provided in scientific notation; therefore, 1E-05 is equivalent to 1×10^{-5} , which equals 0.00001 or a potential lifetime incremental cancer risk of 1 in 100,000). The MDEQ has established that a cumulative LICR value of 1E-05 represents an acceptable target level for combustion risk assessments within the state.

7.2.2. Potential Noncarcinogenic Effects

The potential for adverse effects resulting from indirect exposure to non-carcinogens is assessed by comparing the COPC-specific average daily dose (ADD) to its RfD. This comparison is made by calculating the ratio of the ADD to the corresponding RfD to yield a hazard quotient (HQ) using the following equation (USEPA, 1989):

$$HQ = \frac{ADD}{RfD}$$

[Equation 7-3]

where:

HQ = Hazard Quotient (unitless);
ADD = Average daily COPC dose (mg/kg-d); and
RfD = COPC-specific reference dose (mg/kg-d);

The equations used to estimate potential non-carcinogenic effects for the inhalation pathway have also been modified in the 2005 HHRAP guidance. Consistent with these modifications, the revised HHRA utilizes the following equation to quantify potential non-carcinogenic effects resulting from inhalation:

$$HQ_{inh(i)} = \frac{(EC \cdot UCF)}{RfC}$$

[Equation 7-4]

where:

HQ_{inh(i)} = Hazard quotient for direct inhalation of COPC_i (unitless);
EC = Exposure concentration calculated per Equation 5-57 ($\mu\text{g}/\text{m}^3$);
UCF = Units correction factor (0.001 mg/ μg); and
RfC = Reference concentration (mg/m^3).

For example, if the average daily intake of a COPC is equal to the RfD, the HQ is 1; if the average daily intake is less than or greater than the RfD, the HQ is less than or greater than 1, respectively. HQs for individual COPCs and exposure pathways are then summed to yield hazard indices (HIs). This methodology, unlike that used to

evaluate cancer risk, is not a true measure of the probability of risk (i.e., it does not predict the relative likelihood of the occurrence of adverse effects). Although exceedance of an RfD (i.e., an HQ greater than 1) is usually regarded as a regulatory benchmark by USEPA, risk assessors regard RfDs as relatively "soft" estimates, whose bounds of uncertainty can span an order of magnitude. The MDEQ has established that a cumulative HI value of 0.25 represents an acceptable target level for combustion risk assessments within the state.

As mentioned above, hazard quotients (HQs) for different exposure pathways are summed to yield a cumulative HI which accounts for exposure by a given receptor across multiple COPCs and exposure pathways. In the absence of data to the contrary, USEPA assumes dose- and effect-additivity for non-carcinogenic effects (USEPA 1989a). There is an exception to this additivity approach. The HHRA guidance states that additivity of HIs should be restricted to COPCs which act on common target organs (USEPA, 2005). In other words, it would be inappropriate to combine the HI from a chemical which acts primarily on the skin with the HI from a chemical that acts primarily on the liver. Segregation of HIs based on major health effects and target organs or systems is only undertaken when the total HI exceeds the benchmark target level and the individual COPC-specific HI values are less than the benchmark target level. Where appropriate, HIs designated by effect have been presented separately for each population subgroup evaluated.

7.3 Risk Characterization Under Default Exposure Scenarios

7.3.1. Summary of Direct and Indirect Risks for the Resident

Potential direct (inhalation) risks for the Resident, based on both carcinogenic and non-carcinogenic endpoints, are presented in Tables 7-1. Although 2,3,7,8-TCDD is the only COPC evaluated in the current HHRA which is considered by USEPA to be a potential carcinogen, the agency has not yet developed an inhalation cancer slope factor. This precludes the development of an inhalation Lifetime Incremental Cancer Risk (LICR). The Hazard Quotient (HQ) resulting from direct (inhalation) exposure to COPCs considered by USEPA to be noncarcinogens (i.e., Hg^{+2} and Hg^0) yields an HQ of 2E-04 with Hg^{+2} responsible for 72-percent of that value. Lead risks are evaluated separately in Section 7.3.8.

From Table 7-2, the cumulative LICR resulting from indirect exposure of the Resident to facility emissions is 7E-11 with 2,3,7,8-TCDD as the only contributor to this value. For non-carcinogenic endpoints (Table 7-3), the HI resulting from indirect exposure to facility emissions is 5E-05 with Hg^{+2} responsible for 66-percent of that value.

7.3.2. Summary of Direct and Indirect Risks for the Resident Child

Potential direct (inhalation) risks for the Resident Child, based on both carcinogenic and non-carcinogenic endpoints, are presented in Tables 7-4. The Hazard Quotient (HQ) resulting from direct (inhalation) exposure to COPCs considered by USEPA to be noncarcinogens (i.e., Hg^{+2} and Hg^0) yields an HQ of 2E-04 with Hg^{+2} responsible for 72-percent of that value. Lead risks are evaluated separately in Section 7.3.8.

From Table 7-5, the LICR resulting from indirect exposure to facility emissions is 7E-11 with 2,3,7,8-TCDD as the only contributor to this value. For non-carcinogenic endpoints (Table 7-6), the HQ resulting from indirect exposure to facility emissions is 2E-04 with Hg^{+2} responsible for 73-percent of that value.

7.3.3. Summary of Direct and Indirect Risks for the Farmer

Potential direct (inhalation) risks for the Farmer, based on both carcinogenic and non-carcinogenic endpoints, are presented in Tables 7-7. The Hazard Quotient (HQ) resulting from direct (inhalation) exposure to COPCs considered by USEPA to be noncarcinogens (i.e., Hg^{+2} and Hg^0) yields an HQ of 1E-04 with Hg^{+2} responsible for 74-percent of that value. Lead risks are evaluated separately in Section 7.3.8.

From Table 7-8, the LICR resulting from indirect exposure to facility emissions is 1E-08 with 2,3,7,8-TCDD as the only contributor to this value. For non-carcinogenic endpoints (Table 7-9), the HQ resulting from indirect exposure to facility emissions is 3E-04 with 2,3,7,8-TCDD responsible for 63-percent of that value.

7.3.4. Summary of Direct and Indirect Risks for the Farmer Child

Potential direct (inhalation) risks for the Farmer Child, based on both carcinogenic and non-carcinogenic endpoints, are presented in Tables 7-10. The Hazard Quotient (HQ) resulting from direct (inhalation) exposure to COPCs considered by USEPA to be noncarcinogens (i.e., Hg^{+2} and Hg^0) yields an HQ of 1E-04 with Hg^{+2} responsible for 74-percent of that value. Lead risks are evaluated separately in Section 7.3.8.

From Table 7-11, the LICR resulting from indirect exposure to facility emissions is 3E-09 with 2,3,7,8-TCDD as the only contributor to this value. For non-carcinogenic endpoints (Table 7-12), the HQ resulting from indirect exposure to facility emissions is 5E-04 with 2,3,7,8-TCDD responsible for 54-percent of that value.

7.3.5. Summary of Direct and Indirect Risks for the Fisher

Potential direct (inhalation) risks for the Fisher, based on both carcinogenic and non-carcinogenic endpoints, are presented in Tables 7-13. The Hazard Quotient (HQ) resulting from direct (inhalation) exposure to COPCs

considered by USEPA to be noncarcinogens (i.e., Hg^{+2} and Hg^0) yields an HQ of 2E-04 with Hg^{+2} responsible for 72-percent of that value. Lead risks are evaluated separately in Section 7.3.8.

From Table 7-14, the LICR resulting from indirect exposure to facility emissions is 6E-10 with 2,3,7,8-TCDD as the only contributor to this value. For non-carcinogenic endpoints (Table 7-15), two different HQ values are presented based on the different approaches used in the HHRA to model the movement of mercury from surface water into fish tissue. The HQ resulting from adherence to the USEPA guidance is 4E-02 with methylmercury responsible for more than 99-percent of that value. The HQ resulting from adherence to the approach recommended by MDEQ is 8E-03 with methylmercury responsible for more than 99-percent of that value.

7.3.6. Summary of Direct and Indirect Risks for the Fisher Child

Potential direct (inhalation) risks for the Fisher Child, based on both carcinogenic and non-carcinogenic endpoints, are presented in Tables 7-16. The Hazard Quotient (HQ) resulting from direct (inhalation) exposure to COPCs considered by USEPA to be noncarcinogens (i.e., Hg^{+2} and Hg^0) yields an HQ of 2E-04 with Hg^{+2} responsible for 72-percent of that value. Lead risks are evaluated separately in Section 7.3.8.

From Table 7-17, the LICR resulting from indirect exposure to facility emissions is 2E-10 with 2,3,7,8-TCDD as the only contributor to this value. For non-carcinogenic endpoints (Table 7-18), two different HQ values are again presented based on the different approaches used in the HHRA to model the movement of mercury from surface water into fish tissue. The HQ resulting from adherence to the USEPA guidance is 3E-02 with methylmercury responsible for more than 99-percent of that value. The HQ resulting from adherence to the approach recommended by MDEQ is 6E-03 with methylmercury responsible for 98-percent of that value.

In summary, Table 7-19 provides the cumulative LICRs for each of the default receptor groups with the highest cumulative value (i.e., 1E-08) observed in the Farmer. Table 7-20 summarizes the information for HIs with the highest value (i.e., 4E-02) observed in the Fisher. Both values are well below MDEQ's levels of concern.

7.3.7. Risk Characterization of Dioxin Exposure Through Breast Milk

The current agency approach for evaluating the significance of infant exposure to dioxins/furans via the maternal breast milk pathway is to compare it against some estimate of average or background maternal daily dose. More-specifically, the current HHRA has evaluated the significance of the estimated average daily dose to the infant by comparing it against the national average daily dose which would be received by an infant through ingestion of breast milk from a mother receiving an average 2,3,7,8-TCDD TEQ exposure. Based on an average 2,3,7,8-

TCDD TEQ concentration of 16-parts per trillion (ppt) in breast milk, the comparative average infant daily intake would be approximately 60-picograms (pg)/kg-day (USEPA, 2005).

In the current HHRA, the highest predicted average daily 2,3,7,8-TCDD TEQ dose modeled for a nursing infant was associated with the default Farmer scenario (see Table 5-95). When 2,3,7,8-TCDD TEQ dose estimates were compared against the national average infant daily intake of 60-pg TEQ/kg-d, all four receptor scenarios were found to be well below that reference level (See Table 7-21). These results are not really surprising, as the average daily 2,3,7,8-TCDD TEQ doses modeled for the adult receptors (see Table 5-93) were also well below the national average background exposure level of 1 pg (i.e., 1E-09 mg) TEQ/kg-d (USEPA, 2005).

7.3.8. Risk Characterization of Lead Exposure

As described in Section 6.3 (Lead Assessment), USEPA-verified RfDs are not available for lead. Consequently, the approach used to characterize the potential adverse health effects associated with lead exposure differs from that used to evaluate other COPCs. In the MME HHRA, site-specific and default exposure data have been used as inputs for USEPA's IEUBK model. The model output consists of a best estimate of a plausible range of blood lead concentrations for a hypothetical child under the specific exposure conditions established within the HHRA. The range of values is centered on the geometric mean blood lead concentration expected for a typical child with this exposure scenario. The portion of the upper tail of the probability distribution exceeding some chosen blood lead concentration (i.e., typically 10 µg/dL) provides an estimate of the risk of exceeding that level for a typical child of that age residing in the same household and with the same exposure history (USEPA, 2002). IEUBK was run for the age range of 0 to 84 months using a 15-minute time step for the numerical iteration.

7.3.8.1. Results of IEUBK Modeling

As described previously, the goal of the current HHRA was to determine the potential human health impacts associated with operation of the planned MME facility. For consideration of the incremental impact of lead emissions, four different scenarios were evaluated using the IEUBK model. These scenarios included:

- Scenario 1: Background conditions assuming no lead-based paint in homes [i.e., lead in interior dust results from two sources: soil being tracked into homes and deposition of airborne lead];
- Scenario 2: Background conditions assuming lead-based paint in good condition in homes [i.e., default interior dust lead value of 200 mg/kg];
- Scenario 3: Incremental impact from MME emissions assuming no lead-based paint in homes; and
- Scenario 4: Incremental impact from MME emissions assuming lead-based paint in good condition in homes.

The results are presented in graphical form in Figures 7-1 through 7-4 and summarized in Table 7-22. It is important to note that the IEUBK model outputs represent a distribution of predicted blood lead levels rather than a single value. Based on the input parameters described in Section 5.7.5, the IEUBK model predicted a background geometric mean blood lead concentration of 2.401 µg/dL in children living in homes that were free of lead-based paint (Scenario 1); only 0.120 percent of those children would be expected to exceed the CDC's health-based level of concern (i.e., 10 µg/dL). If those homes contained lead-based paint in good condition (Scenario 2), a background geometric mean blood lead concentration of 3.495 µg/dL could be expected, with approximately 1.265 percent of exposed children expected to exceed CDC's health-based level of concern.

When the impact of MME emissions was evaluated in children living in homes with no lead-based paint (Scenario 3), the IEUBK model predicted a slight increase in the geometric mean blood levels (i.e., from 2.401 to 2.404 µg/dL). Likewise, the percentage of children with blood levels exceeding CDC's health-based level of concern would only be expected to increase from 0.120 to 0.121. A similar pattern was predicted for children living in homes with lead-based paint in good condition (Scenario 4) where the IEUBK model again predicted a modest increase to 3.498 µg/dL in the geometric mean blood levels (i.e., from 3.495 to 3.498 µg/dL). The percentage of children with blood levels exceeding CDC's health-based level of concern also increased slightly from 1.265 to 1.271. Table 7-23 provides a summary of the outputs from the IEUBK run under Scenario 4 showing the modeled daily lead intakes by medium and by age and the resulting predicted blood lead levels.

None of the lead exposure scenarios evaluated in the current assessment indicates that anticipated lead emissions from the MME facility should even approach CDC's levels of concern. A reasonable estimation of background lead levels suggests that those levels are also below CDC's levels of concern.

8.0 UNCERTAINTY ANALYSIS

8.1 Introduction

The purpose of this section is to highlight the primary sources of uncertainty within each component of the HHRA and to qualitatively identify the potential impact of such uncertainty on the ultimate characterization of risk. In this manner, the uncertainty assessment provides a context for understanding the risk characterization results with respect to the true nature of the predicted risks; such an approach allows the predicted risks to be placed in the proper perspective and provides the risk manager and the general public with a more balanced picture. The HHRA process, as prescribed in the HHRA guidance developed by the Office of Solid Waste (OSW), is conservative by design (USEPA, 2005). In consciously requiring that risk be overestimated, regulatory agencies can be assured that the actual risks resulting from operation of a combustor unit will not be underestimated.

8.2 Stack Emission Estimates

Stack emission rates for mercury and lead were based upon the chemical analysis of representative coal feed stocks and the design features of the MME facility. Since the permit will limit the emissions of both mercury and lead on a maximum potential basis, the emission estimates used in the HHRA are conservative (i.e., actual emissions should be lower than those evaluated in the HHRA) and will tend to overestimate impacts. There is more uncertainty associated with the emission estimates for dioxins/furans evaluated in the HHRA. There is limited information describing the emissions of dioxins/furans from coal fired electric generating facilities. Appendix C of the HHRA summarizes the available literature used to develop the emission estimates evaluated in the HHRA. Although there is a high level of uncertainty associated with the applicability of these emission profiles to the MME facility, that uncertainty was offset to a degree by the decision to assume in the HHRA that all the potentially carcinogenic dioxin/furan congeners were present as 2,3,7,8-TCDD. As that congener (along with 1,2,3,7,8-pentachlorodibenzo(p)dioxin) is assigned the highest carcinogenic potency, that decision should result in an overestimation of dioxin/furan impacts.

Surrogate mercury speciation data were not available for natural gas fired boilers, so a conservative approach was adopted to ensure that mercury impacts from those sources would not be underestimated. As elemental mercury and divalent mercury emissions were modeled separately using AERMOD, the assumption was made that the mercury emissions for the natural gas fired boilers shown in Table 3-1 were entirely in the form of elemental mercury when elemental mercury was being modeled and entirely in the form of divalent mercury when divalent mercury was being modeled. So, although the speciation of mercury in natural gas fired boiler emissions is a source of uncertainty in the current HHRA, the approach adopted to deal with that uncertainty means that the mercury impacts described in the HHRA will overestimate mercury impacts from those sources.

8.3 Air Modeling of Stack Emissions

The air impacts from lead and mercury emissions were predicted using a computer model (i.e., AERMOD) developed by USEPA. AERMOD is a state-of-the-science modeling system developed to replace an earlier regulatory model (i.e., ISC). It has been promulgated as the guideline model for use in both state and federal PSD applications where an assessment of both ambient impacts and secondary impacts are required.

The location of the NWS station used as a source of the meteorological data can introduce considerable uncertainty into the air modeling activity; it is not unusual for the nearest NWS station to be more than 50-100 miles from the combustor being modeled. To reduce this uncertainty, MDEQ has processed meteorological data sets for 56 stations across MI, including the Midland-Bay City-Saginaw (MBS) Airport, the source of the meteorological data used in the current HHRA. The MBS Airport is located in Freeland, Saginaw County, MI approximately 10-km (i.e., 6.2-miles) from the MME facility, so there should be a high degree of confidence that the meteorological data used in the air modeling actually reflect conditions within the study area.

Deposition modeling requires information on the size distribution of emitted particles. The particle size distribution used in the air modeling was based upon data from USEPA's Compilation of Air Pollutant Emission Factors (AP-42) for a pulverized coal fired boiler with fabric filter control of particulate matter. As these represent the best data available from the USEPA, the uncertainty associated with their use is likely quite low. The potential impact of this uncertainty has been tempered through our use of residential and farm areas to derive air modeling impacts rather than specific predefined locations which might result in shifts in emission patterns being missed.

8.4 Fate-and-Transport Modeling

In the MME HHRA, multiple fate-and-transport pathways were evaluated to ensure compliance with USEPA HHRA guidance requirements. In addition to the relatively straightforward air modeling methodology used to predict ambient air concentrations and deposition fluxes at pre-selected receptor locations, a complex, interrelated series of algorithms was used to develop estimates of COPC exposure concentrations for the following indirect pathways:

- Incidental ingestion of surface (untilled) soil;
- Ingestion of above-ground produce;
- Ingestion of below-ground (root) produce;
- Ingestion of beef and milk;
- Ingestion of pork;
- Ingestion of poultry and eggs; and
- Ingestion of fish.

There are several examples in the current guidance where upper-bound exposure assumptions have been used so as not to underestimate risk. As an example, the default receptor scenario assumes that the adult resident spends 350 days out of every year at his/her home and that exposure to emitted chemicals at that location continues uninterrupted for 30 years. In reality, very few individuals are likely to spend that amount of time in their homes (USEPA, 1997a); such an exposure could be considered to be unrealistically-conservative, even though it is theoretically possible. A second example can be found in the evaluation of impacts from lead using the IEUBK model. One of the important input parameters for the model is the concentration of lead in house dust. As nothing was known about the presence of lead in the dust inside Midland County residences, a default value (i.e., 200 mg/kg) was used in one of the exposure scenarios. Although this value is appropriate to describe homes that have lead paint in good condition, it probably overestimates actual dust lead levels.

Another example of this conservatism can be seen in the Resident scenario, where the assumption is made that 2.6 ounces of homegrown produce are consumed every day and this consumption rate is in place 350 days/year for 30 years. In reality, most families consume a diet that is a blend of homegrown food and food purchased locally (i.e., in supermarkets) but grown/raised outside the immediate area.

MDEQ raised concerns that there were limited data available for several of the parameters used to characterize area surface water bodies. This was found to be the case with Total Suspended Solids (TSS). Although STORET data were available for the Tittabawassee River, no TSS data could be found for the two small lakes evaluated in the HHRA (i.e., Kiewassee Lake and the Unnamed Lake). In consultation with MDEQ, two different default TSS values (i.e., 2 and 10 mg/L) were evaluated for the two lakes in the selection process to identify the most-impacted water body of the three under consideration. The results, summarized in Appendix A, indicated that changing the TSS from 2 mg/L to 10 mg/L had a greater impact (i.e., increase) on fish consumption impacts when evaluated under the MDEQ approach for modeling mercury uptake into fish than under the USEPA approach recommended in the guidance. As potential mercury emission impacts on the Fisher and Fisher Child were evaluated for Kiewassee Lake at an assumed TSS value of 10 mg/L, the uncertainty associated with the TSS should be reduced. However, as the true value for this parameter remains unknown, some level of uncertainty remains.

An evaluation of the area within 10-km of the proposed MME facility produced no indication that surface water from that area is used as a significant source of drinking water. As it was impossible to contact every resident within that area to confirm this, there is a degree of uncertainty associated with our decision to exclude consumption of surface water as a complete exposure pathway in the HHRA. The knowledge that the source of drinking water for

Midland is more than 80-km (i.e., 50-miles) away in Lake Huron certainly reduces the degree of uncertainty associated with the decision to eliminate that exposure pathway.

8.5 Toxicity Assessment

USEPA has adopted a standardized approach for evaluating the various sources of uncertainty associated with the assessment of toxicity studies and their relevance to the chronic low-level exposure scenarios typically encountered in combustor risk assessments. Through the introduction of various levels of conservatism, the agency has ensured that the various toxicity criteria generated for use in the HHRA process would not underestimate the true toxicity of a COPC.

8.5.1. Uncertainties in the Toxicity Assessment of Carcinogens

When available, USEPA relies on human studies to develop toxicity values for carcinogens. However, when human data are unavailable, animal data are used. Many of the toxicity values used in the current risk assessment have been derived using animal data. However, because of species differences in target organ dose, metabolism, and response, a specific chemical may not produce the same toxic endpoint (i.e., carcinogenicity) in all species. For many substances that are carcinogenic in animals, there is uncertainty as to whether they are also carcinogenic in humans. The finding that relatively few substances are known human carcinogens may be due in part to the difficulty in conducting adequately designed epidemiologic investigations in human populations known to be exposed. Available human data are derived mainly from retrospective epidemiology studies of workers exposed to multiple chemicals, at doses that often cannot be verified.

USEPA-verified slope factors in IRIS are currently accompanied by a weight-of-evidence classification, which is an indication of the likelihood that the agent is a human carcinogen. Under the 1986 guidelines, tumor findings in animals or humans were the dominant component of this classification process. Other information about a chemical's properties, its structure-activity relationships to other carcinogenic chemicals and its activities in studies of carcinogenic processes was often limited and played only a modulating role as compared with tumor findings. Under the proposed new guidelines, weighing of the evidence would include addressing the likelihood of human carcinogenic effects of the chemical and the conditions under which such effects may be expressed.

One of the most controversial issues in the dose-response assessment of potential carcinogens has been the extrapolation of effects measured at experimental exposure levels to the levels commonly associated with environmental exposures. It was not uncommon, for example, to observe an increased tumor incidence in

experimental animals only at the highest doses tested. Even when that dose was the maximum tolerated dose, the assumption was made that the chemical would also produce tumors at doses approaching zero.

The new guidelines also propose use of straight-line extrapolation for default situations where linearity between the point of departure and zero is assumed. This is a change from the earlier guidance which recommended use of a linearized multistage (LMS) procedure. As stated in the 1996 guidance, this change was made because "the former modeling procedure gave an appearance of specific knowledge and sophistication unwarranted for a default". The LMS model uses the 95-percent upper confidence limit on the slope of the dose-response curve. One problem encountered in the use of the upper 95-percent confidence interval is that it essentially reduces the model to worse-case curve fitting; it provides an estimate of the upper bound on risk, since the estimate results in an assumption of a high sensitivity to the carcinogen. USEPA (1986) has also emphasized:

"that the linearized multistage model leads to a plausible upper limit to the risk that is consistent with some proposed mechanisms of carcinogenesis. Such an estimate, however, does not necessarily give a realistic prediction of the risk. The true value of the risk is unknown, and may be as low as zero."

Additionally, under the proposed new guidelines, the assessment of inhalation carcinogenesis will rely on methodologies developed for the inhalation RfC, such that the internal dose may also be considered in establishing the dose-response relationship rather than using the exposure concentration as a surrogate of the dose.

In summary, even though USEPA has indicated that the existing cancer guidelines are in need of revision, those changes have not yet been fully implemented. In general terms, however, it can be concluded that the existing approaches used for the development of cancer slope factors will tend to overestimate the magnitude of the risk. In addition, consideration of the complex biological and policy issues associated with the evaluation of potential carcinogens, reinforces our belief that route-to-route extrapolation of toxicity criteria, without the appropriate scientific justification, should be avoided.

8.5.2. Uncertainties in the Toxicity Assessment of Noncarcinogens

Health criteria for COPCs exhibiting chronic non-carcinogenic effects are generally developed using the Reference Dose (RfD) or Reference Concentration (RfC). The RfD/RfC is the estimated level of daily exposure to the human population (including sensitive subpopulations) below which it is unlikely that appreciable risk of deleterious effects will result over a lifetime of exposure. It is assumed when a no-observed-adverse-effect level (NOAEL) or lowest-observed-adverse-effect level (LOAEL) is selected as the basis for the RfD/RfC, toxic effects will be avoided.

USEPA usually bases the RfD/RfC for non-carcinogens on the most-sensitive animal species. The dose is then adjusted by the use of uncertainty factors and modifying factors to compensate for the various sources of uncertainty inherent in the underlying toxicity database. Commonly used uncertainty factors include the following:

- Uncertainty factor of 10 to account for intraspecies variability (i.e., sensitive individuals);
- Uncertainty factor of 10 to account for interspecies variability (i.e., mouse to man);
- Uncertainty factor of 10 to account for subchronic to chronic exposure adjustment (i.e., short-term to lifetime exposure); and
- Uncertainty factor of 10 to account for LOAEL to NOAEL adjustment.

Due to the conservative nature of this approach, the compounding effect of multiplying individual uncertainty factors together could result in RfD/RfC values which tend to overestimate the actual risk. The IRIS database also contains an evaluation of the quality of the data used to generate a RfD/RfC. In many cases the level of confidence in the study is classified as low.

8.6 Risk Characterization

The previous sections have provided a discussion of the uncertainties associated with each component of the risk assessment process. In order to address these uncertainties, current USEPA HHRA guidance require the use of conservative assumptions throughout the risk assessment process in order to err on the side of public health protection. The cumulative impact of applying conservative assumptions throughout the risk assessment is that the estimated risks are almost certain to overestimate the true risks.

The fate-and-transport modeling used to predict exposure concentrations in various media is anticipated to overestimate impacts. This starts with the conservatism built into the USEPA air models used to model impacts at particular geographic locations and continues through the multiple mathematical algorithms used to evaluate the step-by-step migration of chemicals through the various direct and indirect exposure pathways. Many of these algorithms are closely linked with other algorithms and the use of conservative, upper-bound, default exposure assumptions will actually result in an exposure scenario that is so conservative that it would be unlikely to occur.

The toxicity assessment incorporates uncertainty factors and modifying factors and assumes upper-bound dose-response relationships in order to account for the uncertainties inherent in extrapolating from toxicity studies conducted in laboratory animals using high exposure doses to the situation where humans are exposed at relatively low concentrations. An additional level of conservatism results from summing the risks independent of

the target organ. The assumption that all chemicals act in a similar manner is a conservative over-simplification of how chemicals are known to produce toxicity and will tend to overestimate the actual risk.

In the final characterization of risk, all of the conservative assumptions applied in the emissions characterization, fate-and-transport modeling, exposure assessment, and toxicity assessment are compounded in the derivation of a numerical estimate of risk. As a consequence, the projected risks associated with operation of the proposed MME facility are likely to have been overestimated. Even though application of the HHRA process indicates that the operation of the new facility will be accomplished without appreciable additional risk to the surrounding community, actual risks should be even lower than those demonstrated in this assessment.

9.0 SUMMARY AND CONCLUSIONS

Mid-Michigan Energy is proposing to construct and install a new coal-fired facility in Midland, Michigan. The Michigan Department of Environmental Quality (MDEQ) has requested that a human health risk assessment (HHRA) be conducted as part of the permitting process for the new facility. The resulting HHRA focuses on mercury, lead and 2,3,7,8-TCDD emissions and evaluates potential human health impacts associated with operation of the new facility.

The HHRA has been based on current federal risk assessment guidance, titled Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (USEPA, 2005), although it has also incorporated several requirements from the MDEQ. The migration of emitted mercury, lead and 2,3,7,8-TCDD from their release into the atmosphere at the MME facility up to their ultimate interaction with receptors involves a series of complex modeling processes. USEPA's AERMOD was used to evaluate the atmospheric movement of emitted chemicals, resulting in air concentration and deposition flux estimates at 5,536 predefined grid locations.

At the request of MDEQ, a subset of USEPA default receptor scenarios were evaluated in the HHRA for both direct and indirect exposures to mercury, lead and 2,3,7,8-TCDD. The default receptor scenarios included:

- Resident;
- Resident Child;
- Farmer;
- Farmer Child;
- Fisher; and
- Fisher Child.

Consistent with current USEPA guidance, the Resident and Fisher receptors were assumed to live at the same location, identified as the residential area of highest potential impact through evaluation of air modeling outputs, consideration of existing zoning and examination of digitized aerial photographs. Potential exposure to emitted lead and mercury through inhalation, incidental ingestion of surface soil and ingestion of homegrown plants/vegetables was evaluated for all four default receptor scenarios at the selected residence location. In addition, the Fisher and Fisher Child were evaluated for exposure to mercury, lead and 2,3,7,8-TCDD through the ingestion of fish from the Kiewassee Lake. Kiewassee Lake was selected for inclusion in the HHRA following a screening process that evaluated impacts on the Tittabawassee River, Kiewassee Lake and an unnamed lake located to the southeast of the MME facility. Farmer and Farmer Child receptors were assumed to live in

agricultural areas selected through reconnaissance of candidate locations as well as an evaluation of aerial photographs and air modeling results.

The terrestrial and aquatic fate-and-transport of mercury, lead and 2,3,7,8-TCDD involve a complex series of linked algorithms developed by USEPA in the HHRA guidance. The first step involves estimating the concentration of the COPCs in various exposure media, including: air, soil (both tilled and untilled), homegrown plants, animal products, surface water and fish. The second step involves estimating the daily intake of COPCs through exposure to those media. Once an exposure level has been estimated, USEPA methods are used to determine if that exposure was sufficiently high to pose a threat to human health. Potential impacts from the various mercury species and 2,3,7,8-TCDD are evaluated through comparison of exposure levels against either a reference dose (RfD) or cancer slope factor (CSF) for ingestion or a reference concentration (RfC) or unit risk factor (URF) for inhalation.

Potential impacts from lead exposure were evaluated in a different manner. In contrast to the various forms of mercury, lead is not believed to exhibit threshold behavior; as a consequence, USEPA has not developed a RfD or RfC. The MDEQ recommended that the potential health impacts from lead be evaluated using the Integrated Exposure Uptake Biokinetic (IEUBK) model which focuses on potential impacts on children during the first seven years of life. IEUBK integrates lead intake from multiple exposure pathways and then estimates a blood lead level. The Centers for Disease Control (CDC) have established a blood lead level of 10 µg/dL as the level of concern; according to the agency, blood lead concentrations less than that are “not considered to be lead poisoned”.

The HHRA results indicate that incremental exposure to emitted 2,3,7,8-TCDD (the sole carcinogenic COPC in the current HHRA) should not pose a carcinogenic threat to individuals residing within the study area. Potential carcinogenic impacts from 2,3,7,8-TCDD were quantified through generation of a Lifetime Incremental Cancer Risk (LICR); direct and indirect exposure levels were compared against the appropriate toxicity criterion and the resulting LICR values were summed across pathways and COPCs for each receptor group. From Table 7-19, the highest combined LICR was associated with the Farmer (1E-08) followed by the Farmer Child (3E-09), Fisher (6E-10), Fisher Child (2E-10) and Resident/Resident Child (7E-11). This pattern is not surprising given the consumption of animal products by the Farmer and Farmer Child. Again, the combined LICR estimates for all four receptor groups indicated that the incremental 2,3,7,8-TCDD impacts should not present a human health concern.

The MDEQ recommended a target combined target LICR level equal to or less than 1E-05. For all four receptor scenarios, the combined LICR was well below that target level.

The HHRA results also indicate that incremental exposure to emitted mercury or 2,3,7,8-TCDD should not pose a noncarcinogenic threat to individuals residing within the study area. Potential noncarcinogenic impacts from the various mercury species and 2,3,7,8-TCDD were quantified through generation of a hazard index (HI); direct and indirect exposure levels were compared against the appropriate toxicity criterion and the resulting ratio values were summed across pathways and COPCs for each receptor group. From Table 7-20, the highest combined HI was associated with the Fisher (4E-02) followed by the Fisher Child (3E-02), Farmer Child (6E-04), Farmer (4E-04), Resident Child (3E-04) and Resident (2E-04). This pattern is not surprising given the consumption of fish by the two Fisher groups. Again, the combined HI estimates for all four receptor groups indicated that the incremental mercury and 2,3,7,8-TCDD impacts should not present a human health concern. Typically, a combined HI ≤ 1 indicates an exposure level that can be tolerated by all individuals, including sensitive subpopulations, throughout a lifetime without any adverse effects. As mercury exposures unrelated to operation of the proposed MME facility can occur, the MDEQ recommended an alternate target level (i.e., combined HI ≤ 0.25). For all four receptor scenarios, the combined HI was well below that target level.

The incremental increases in media lead levels resulting from operation of the proposed MME facility were added to region-specific background lead levels in those media. The resulting media lead concentrations were evaluated using USEPA's IEUBK model, which integrates multiple exposure pathways and predicts blood lead levels in children. As there was uncertainty associated with the potential contribution of lead-based paint to dust levels in area homes, two different residential scenarios were evaluated. In the first, the assumption was made that no lead-based paint was present; all lead in household dust was assumed to result from the tracking of soil into the homes and the deposition of airborne lead. The IEUBK-modeled geometric mean blood lead level under that background scenario was 2.401 $\mu\text{g}/\text{dL}$. Under the second scenario, the assumption was made that lead-based paint was present in area homes, however, the paint was in good condition. USEPA believes that dust lead levels under that scenario could be as high as 200 mg/kg. The IEUBK-modeled geometric mean blood lead level under the second background scenario was 3.495 $\mu\text{g}/\text{dL}$.

When the impact of MME emissions was evaluated in children living in homes with no lead-based paint, the IEUBK model predicted a slight increase in the geometric mean blood levels (i.e., from 2.401 to 2.404 $\mu\text{g}/\text{dL}$). A similar pattern was predicted for children living in homes with lead-based paint in good condition where the IEUBK model again predicted a modest increase to 3.498 $\mu\text{g}/\text{dL}$ in the geometric mean blood levels (i.e., from 3.495 to 3.498 $\mu\text{g}/\text{dL}$). Under both scenarios, background lead levels would not be predicted to result in blood lead levels of concern in area children and the incremental impact of the MME facility emissions above those background levels would not be significant.

Given the level of conservatism inherent in USEPA's HHRA guidance, the results of the HHRA indicate that the incremental impact of mercury, lead and 2,3,7,8-TCDD emissions resulting from operation of the proposed MME facility should not present a human health threat to individuals living in the surrounding area.

10.0 REFERENCES

Agency for Toxic Substances Disease registry (ATSDR), 2005, *Minimal Risk Levels for Hazardous Substances*, Centers for Disease Control, Atlanta, GA [<http://www.atsdr.cdc.gov/mrls.html>]

City of Midland (Midland), 2006, *2006 Drinking Water Quality Report*.

Centers for Disease Control (CDC), 1991, *Preventing Lead Poisoning in Young Children*, U.S. Department of Health and Human Services, Centers for Disease Control, Atlanta, GA.

California Environmental Protection Agency (CalEPA), 2002, *Chronic Reference Exposure Levels*. Office of Environmental Health Hazard Assessment, Sacramento, CA [http://www.oehha.ca.gov/air/chronic_rels/index.html]

Department of Energy (DOE), 2006, *Large Scale Testing of Enhanced Mercury Removal at W.A. Parish Unit 8, Hoot Lake Unit 3, and Hawthorn Unit 5*, U.S. DOE NETL, October 2006.

Geraghty, J.J., Miller, D.W., Van Der Leeden, F. and F.L. Troise, 1973, *Water Atlas of the United States*, Water Information center, Port Washington, NY.

Michigan Department of Environmental Quality (MDEQ), 2005, *2005 Annual Air Quality Report*, MDEQ, Air Quality Division, Lansing, MI.

Michigan Department of Environmental Quality (MDEQ), 2006a, *Soil Erosion and Sediment Control Training Manual*, MDEQ, Water Bureau, Lansing, MI.

Michigan Department of Environmental Quality (MDEQ), 2006b, *Table 2. Soil: Residential and Commercial I Part 201 Generic Cleanup Criteria and Screening Levels (RBSLs)*, Remediation and Redevelopment Division, Lansing, MI.

National Oceanographic and Atmospheric Administration (NOAA), 2006, *Annual Climatological Survey* [<http://hurricane.ncdc.noaa.gov/ancsum/ACS>]

United States Environmental Protection Agency (USEPA), 1986, *Guidelines for Carcinogen Risk Assessment*, Federal Register, 51:33992-34012.

United States Environmental Protection Agency (USEPA), 1989, *Risk Assessment Guidance for Superfund, Human Health Evaluation Manual, Part A*, Office of Emergency and Remedial Response, Washington, DC. [EPA-540/1-89-002]

United States Environmental Protection Agency (USEPA), 1994, *Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children*, Offices of Emergency and Remedial Response, Washington, DC. [EPA-540/R-93-081]

United States Environmental Protection Agency (USEPA), 1995, *Compilation of Air Pollutant Emission Factors, Volume I: Stationary Point and Area Sources, Fifth Edition, [Supplement E-September 1999]*, Office of Air Quality Planning and Standards, Research Triangle Park, NC. [EPA-540/R-93-081]

United States Environmental Protection Agency (USEPA), 1997a, *Mercury Study Report to Congress, Volume III: Fate and Transport of Mercury in the Environment*, Offices of Air Quality Planning and Standards and Research and Development, Washington, DC. [EPA-452/R-97-005]

United States Environmental Protection Agency (USEPA), 1997b, *Health Effects Assessment Summary Table (HEAST). Fiscal Year 1997 Update*, Environmental Criteria and Assessment Office, Cincinnati, OH. [EPA-540/R-97-036]

United States Environmental Protection Agency (USEPA), 2001, *Database of Sources of Environmental Release of Dioxin-like Compounds in the United States, Version 3.0* [EPA-600/C-01-012]

United States Environmental Protection Agency (USEPA), 2002, *User's Guide for the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) Windows Version-32 Bit Version*, Office of Solid waste and Emergency Response, Washington, DC. [EPA-540/K-01-005]

United States Environmental Protection Agency (USEPA), 2004, *Memorandum Regarding Human Health Toxicity Values in Superfund Risk Assessments*, From: Michael B. Cook, Director, Office of Superfund Remediation and Technologies Innovation (OSRTI). To: Superfund National Policy Managers, Regions 1-10. [OSWER Directive 9285.7-53]

United States Environmental Protection Agency (USEPA), 2005, *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Peer Review Draft*, Solid Waste and Emergency Response, Washington, DC. [EPA-530/R-05-006]

United States Environmental Protection Agency (USEPA), 2007, *Integrated Risk Information System (IRIS)*, National Center for Exposure Assessment, Cincinnati, OH.