

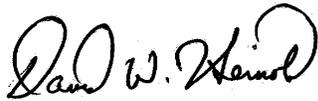
Prepared for:
Hennepin County
Minnesota

Air Dispersion Modeling and Risk Assessment for the Hennepin Energy Recovery Center, Hennepin County, Minnesota (Revised)

ENSR Corporation
June 2007
Document No.: 03433-001-600R

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Prepared By David Heinold



Reviewed By Ishrat Chaudhuri

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Executive Summary

The new ballpark for the Minnesota Twins will be located directly adjacent to the Hennepin Energy Recovery Center (HERC), a mass-burn municipal waste combustor owned by Hennepin County and operated by a subsidiary of Covanta Energy. As required by the Minnesota Urban Ballpark Final Scoping Decision Document (December 2005), Hennepin County Department of Environmental Services (HCDES) retained ENSR Corporation (ENSR) to conduct a dispersion modeling analysis of emissions from HERC to determine if the ballpark could affect the dispersion of HERC emissions in the surrounding area and a risk assessment to determine if HERC emissions could affect the health of ball players, ballpark staff and fans. The analysis conducted by ENSR establishes that the ballpark will not adversely affect dispersion of pollutants from the HERC stacks and that ballpark users will not be exposed to any health risks associated with HERC emissions.

The first part of the dispersion modeling analysis addressed the effect of the ballpark structure on local turbulence and the corresponding effect on the dispersion of pollutants emitted from the two HERC stacks. This analysis was accomplished by applying the U.S. Environmental Protection Agency's (USEPA) guideline air dispersion model, AERMOD, to estimate dispersion factors for two configurations, one without the new ballpark and one with the new ballpark. Dispersion factors are used in dispersion modeling to quantify the degree to which any constituent of stack emissions is diluted before it reaches the ground. Hourly dispersion factors were modeled at an array of receptor locations as close as 100 meters and as far away as 10 kilometers from the HERC stacks using over 5 years of meteorological data (1986-1990) provided by the Minnesota Pollution Control Agency (MPCA). To determine if the presence of the ballpark affected dispersion at any location the maximum 1-hour value and the 5-year average at each receptor were compared. The comparison indicated that the location and magnitude of the maximum dispersion factors will not change as a result of the ballpark and the highest ground-level concentrations associated with HERC emissions will not be affected.

The second part of the analysis addressed whether there could be any exposure to Ballpark users, which includes the players, staff and ticket holders, above levels of concern. Levels of concern in the context of risk assessment are established benchmarks, or risk calculation thresholds defined as a cancer risk range of 1 in 10,000 to 1 in a million and a noncancer Hazard Index (HI) of 1. This was done by applying AERMOD to estimate the air concentrations of specific air compounds emitted from HERC that would occur during periods when players, fans, and staff would be at the ballpark. AERMOD was also applied to estimate the rate of deposition of particulate-bound compounds onto the open ballpark surfaces where 1) fans could inadvertently ingest dust accumulated from deposition of particles in the air, 2) fans could eat food that has been exposed to the open air, and 3) players could ingest soil from the playing field. Compounds evaluated included those for which HERC has specific limits and others which MPCA has identified as being important in evaluating the potential health risk of resource recovery facilities. Two sets of emission rates were applied in the assessment, one set corresponding to actual HERC emissions based on stack measurements and another corresponding to the permit limits. For all compounds, the actual emissions are much lower than the permit limits. Three types of health-risk evaluations were made: 1) compounds for which U.S. EPA and MPCA have established ambient air quality standards, 2) compounds which are known or suspect human carcinogens, and 3) compounds that could be associated with short-term or other long-term health effects. The results indicate that the health risks associated with HERC emissions for each of these types of compounds are below levels of concern.

In summary, the analysis establishes that the ballpark will not adversely affect dispersion of pollutants from HERC stacks and that the estimated risk to ballpark users from exposure to HERC emissions is below levels of concern.

1.0 Introduction

The Hennepin Energy Recovery Center (HERC) facility is a mass-burn municipal waste combustor capable of burning 1,212 tons per day of municipal solid waste in two identical combustion units. Steam produced from combustion is used to turn a 39-megawatt turbine/generator. The facility sells approximately 35 megawatts of electricity (the power usage of approximately 26,000 single family homes) to Xcel Energy. The HERC facility has operated continuously since startup in October 1989. The facility was designed and built by Blount Projects and includes the following major equipment for combustion and air emissions control: two excess-air grate-fired waterwall furnaces, two dry scrubbers for acid gas neutralization, a reverse-air fabric filter baghouse to capture particulates, select non-catalytic reduction of nitrogen oxides, and an activated carbon injection system for capture of gaseous mercury. The HERC facility is owned by Hennepin County and operated by a subsidiary of Covanta Energy, Inc.

The Hennepin County and the Minnesota Ballpark Authority are in the process of building a new 40,000 seat open-air ballpark for the Minnesota Twins at a site adjacent to HERC and one block northwest of the Target Center between 5th Street North and 7th Street North on the edge of the Warehouse District in Downtown Minneapolis, Hennepin County, Minnesota. On behalf of Hennepin County, ENSR Corporation (ENSR) has conducted this study to determine (1) the potential impact of the new ballpark on the dispersion from HERC stacks, and (2) the potential health risk impact of HERC emissions on ballpark users. These tasks were conducted in accordance with the Minnesota Urban Ballpark Final Scoping Decision Document (December, 2005), which outlines issues to be addressed in the Environmental Impact Statement (EIS) for the new ballpark.

Potential impact of the ballpark structure on HERC stack dispersion

The new Minnesota Twins ballpark is proposed to have three levels of seating. The height of the ballpark is about 200 feet, and portions of the park will be within 1000 feet of the HERC stack. It is possible, therefore, that the new structure could slightly alter the local turbulence patterns that affect dispersion of HERC emissions. The impact of the ballpark on HERC dispersion was determined by first applying EPA's Building Profile Input Program (BPIP-Prime) to generate the wind-direction specific building dimensions and then modeling dispersion with and without the ballpark structure with AERMOD. Dispersion modeling indicated that the change in modeled concentrations is inconsequential.

Potential health risk impacts of HERC emissions on ballpark users

AERMOD was then applied to estimate air concentrations of toxic compounds potentially emitted by HERC and estimate deposition on the playing field and in the stands of the ballpark for a variety of receptors including ballplayers, staff and season ticket holders. Two sets of emissions were applied, one corresponding to upper limits provided in the Title V permit and another corresponding to realistic emissions based on the average of recent source tests. Modeling was used to estimate exposure point concentrations and to conduct a health risk assessment to determine potential health risks. The risk assessment indicated that estimated risks to ballpark users are below levels of concern.

2.0 Potential Impact of the Ballpark Structure on HERC Stack Dispersion

2.1 Modeling Considerations

2.1.1 Building Downwash

The analysis used to evaluate the potential for building downwash is referred to as a “Good Engineering Practice” (GEP) stack height analysis. ENSR obtained pertinent source information for the HERC stacks including stack parameters, permitted emission rates of criteria and hazardous air pollutants, and building dimensions for all existing and future structures within a distance of 5 x the lesser of structure height or width from the HERC stacks. Plan view and cross sections were provided for the new ballpark based on present design, including the footprint, height and dimensions of the field and stands. Figure 2-1 (a and b) provides far-field and near-field views of the stack locations and all of the buildings and structures that could result in aerodynamic downwash.

The potential effect of the ballpark on HERC dispersion patterns depends on several factors, such as the ballpark’s height and shape, the distance from the HERC stacks and the height of the HERC stacks. The first step is to determine if the ballpark will have any effect on HERC dispersion. This was accomplished by applying EPA’s Building Profile Input Program (BPIP-Prime) to generate the wind-direction specific building dimensions that are applied in AERMOD for the HERC stacks. BPIP -Prime was applied for two configurations; for the existing configuration and for the ballpark configuration. Comparing the BPIP-Prime output files for the two cases indicates slight differences in effective building dimensions for wind vectors toward the northwest. That is for most directions from HERC (north, northeast, east, southeast, south, southwest and west) the dispersion from HERC would be the same whether or not the new ballpark is present. For the sector to the northwest of HERC, BPIP-Prime indicates there could be some effect on modeled concentrations. Therefore a dispersion model was applied to determine the significance, if any, of this change in downwash parameters.

To evaluate the extent that modeled ground-level concentrations could change, AERMOD (04300) was applied using a 1 g/sec emission rate for both sets of BPIP-Prime input files. The following source parameters used in the most recent modeling conducted for the facility (*Air Quality Modeling Results for Nitrogen Dioxide - Hennepin Energy Resource Company - Municipal Solid Waste Resource Recovery Facility*, April 1995) were used for this assessment.

Height: 65.84 m, Temperature: 399.82° K, Velocity: 20.8 m/sec, Diameter: 2.13 m.

2.1.2 Meteorological Data

MPCA has processed 5 years of hourly National Weather Service (NWS) surface meteorological data (1986-1990) from Minneapolis/Saint Paul airport and twice daily upper air sounding data from Saint Cloud, Minnesota (<ftp://files.pca.state.mn.us/pub/airModel2>). Given the location close to downtown Minneapolis, the dispersion environment was characterized as urban. MPCA’s website has unprocessed (“raw”) surface and upper air meteorological data files which were processed for input to AERMOD. This data was also used for the dispersion and deposition modeling for the risk assessment although deposition was not included in the comparative air quality impact assessment.

The surface meteorological data available on the MPCA website are SAMSON data format which also contain hourly precipitation data required for wet deposition modeling. AERMET (Version 04300), USEPA’s meteorological pre-processor for AERMOD, was used to process the hourly surface and precipitation data, and upper air data. The five years of processed meteorological data were combined into a single meteorological data file for input to AERMOD to compute five-year averages of air concentrations and deposition rates. In addition to the raw meteorological data, site characteristics including surface, albedo and

Bowen ratio are required for the AERMET processing. ENSR used the monthly site characteristics developed by MPCA as provided on the MPCA website.

2.1.3 Ground-Level Receptor Grid

Receptors were placed in concentric rings, centered on a point between the adjacent HERC stacks, in 10° radials and the following distances: 100-m spacing out to 1 km, 200-m spacing out to 2 km, 500-m spacing out to 5 km and 1-km spacing out to 10 km. Receptor terrain elevations and receptor information required by AERMOD was developed through application of the receptor/terrain processor AERMAP (Version 04300). AERMAP was applied with Digital Elevation Model (DEM) data (30-meter resolution) from USGS.

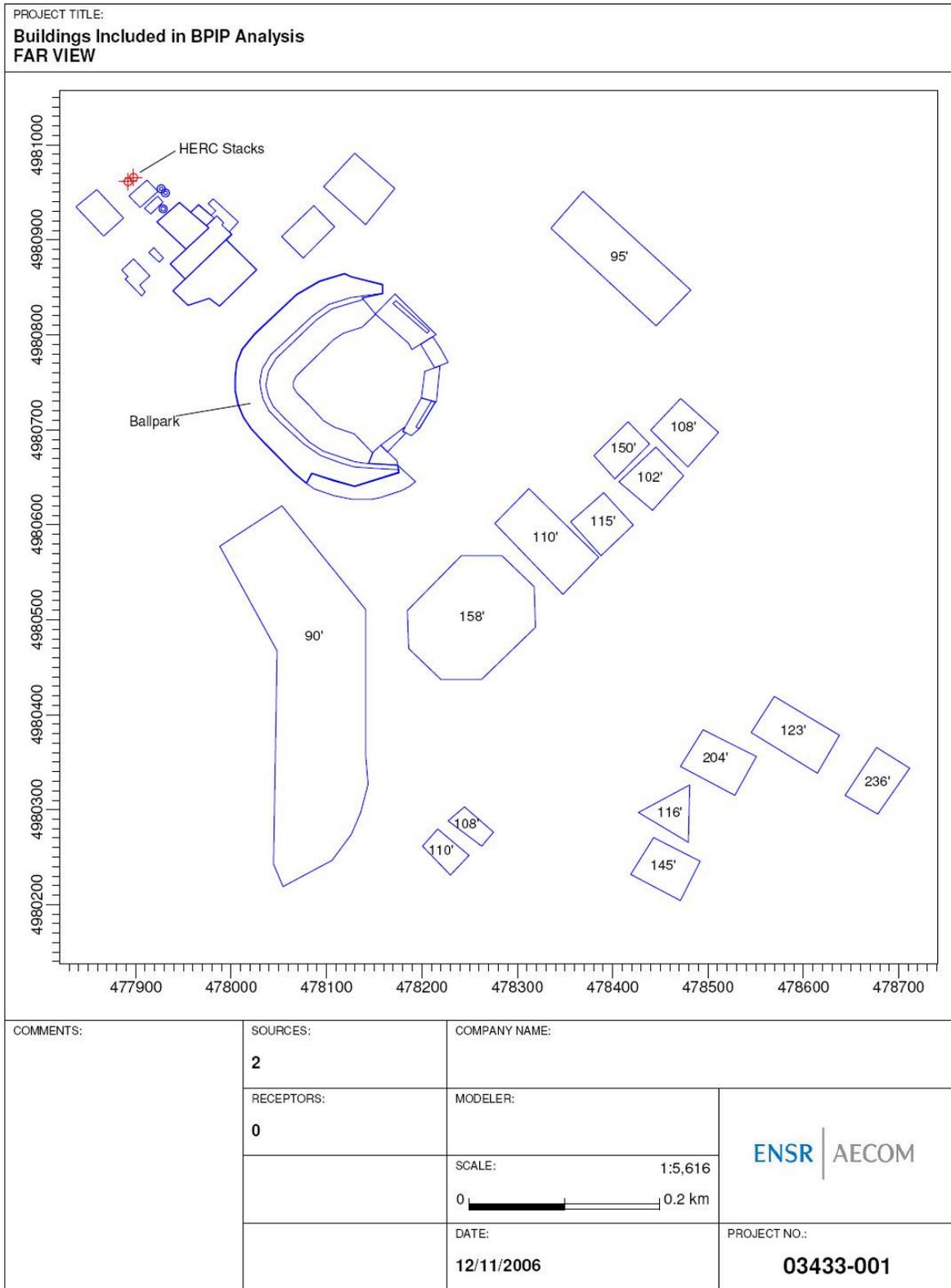
2.2 Results of the Comparative Analysis

A dispersion factor is defined as the modeled concentration associated with a unit (i.e., 1 g/sec) emission of any pollutant and is commonly expressed in units of $\mu\text{g}/\text{m}^3$ per g/sec. That is, if the emission rate of a pollutant, expressed in units of g/sec, is multiplied by the dispersion factor the result would be the modeled ambient concentration of that pollutant in units of $\mu\text{g}/\text{m}^3$. AERMOD was applied for a total 1 g/sec emission rate (0.5 g/sec per stack) for this comparative assessment. Previous modeling for HERC, which formed the basis of the Title V permit, has well established that the facility is designed and operated such that resultant ground level concentrations are well below concentration levels that protect public health and welfare. The purpose of this comparative analysis is to verify that the presence of the ballpark will not substantially affect the modeled maximum concentrations. Figures displaying the one-hour and five-year average modeled dispersion factors prior to and after completion of the ballpark are provided in Figures 2-2 (a, b, c, d). Figure 2-2(a), without the ballpark, and Figure 2-2(b), with the ballpark, show that there is no difference in the maximum one-hour dispersion pattern in all directions except in the sector northwest of HERC. Comparison of Figure 2-2(c), without the ballpark, and Figure 2-2(d), with the ballpark, shows that the five-year average dispersion patterns are virtually identical.

To quantitatively determine if the presence of the ballpark will affect dispersion, the dispersion factors at each receptor location, with and without the ballpark, were compared. Details of this comparison are provided in Appendix A. The comparison indicated that the dispersion factors increased at a small number of receptor locations within about 2 km northwest of HERC. The resultant dispersion factors at these locations were still much less than the maximum dispersion factors, which were at locations where the ballpark will have no effect. Thus, the magnitude and locations of the maximum modeled concentrations are not affected by the ballpark.

In summary, comparison of modeled impacts from the present buildings and with the ballpark indicate that only a small area would be affected and that the ballpark will not affect maximum modeled concentrations. Therefore, the effect of the ballpark on ambient air quality associated with HERC emissions will be inconsequential.

Figure 2-1(a) Buildings Included in Downwash Analysis (far-field view) Showing Building Height (distances in meters)



ISC-AERMOD View - Lakes Environmental Software

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Figure 2-2(a) Maximum 1-Hour Dispersion Factor without the Ballpark

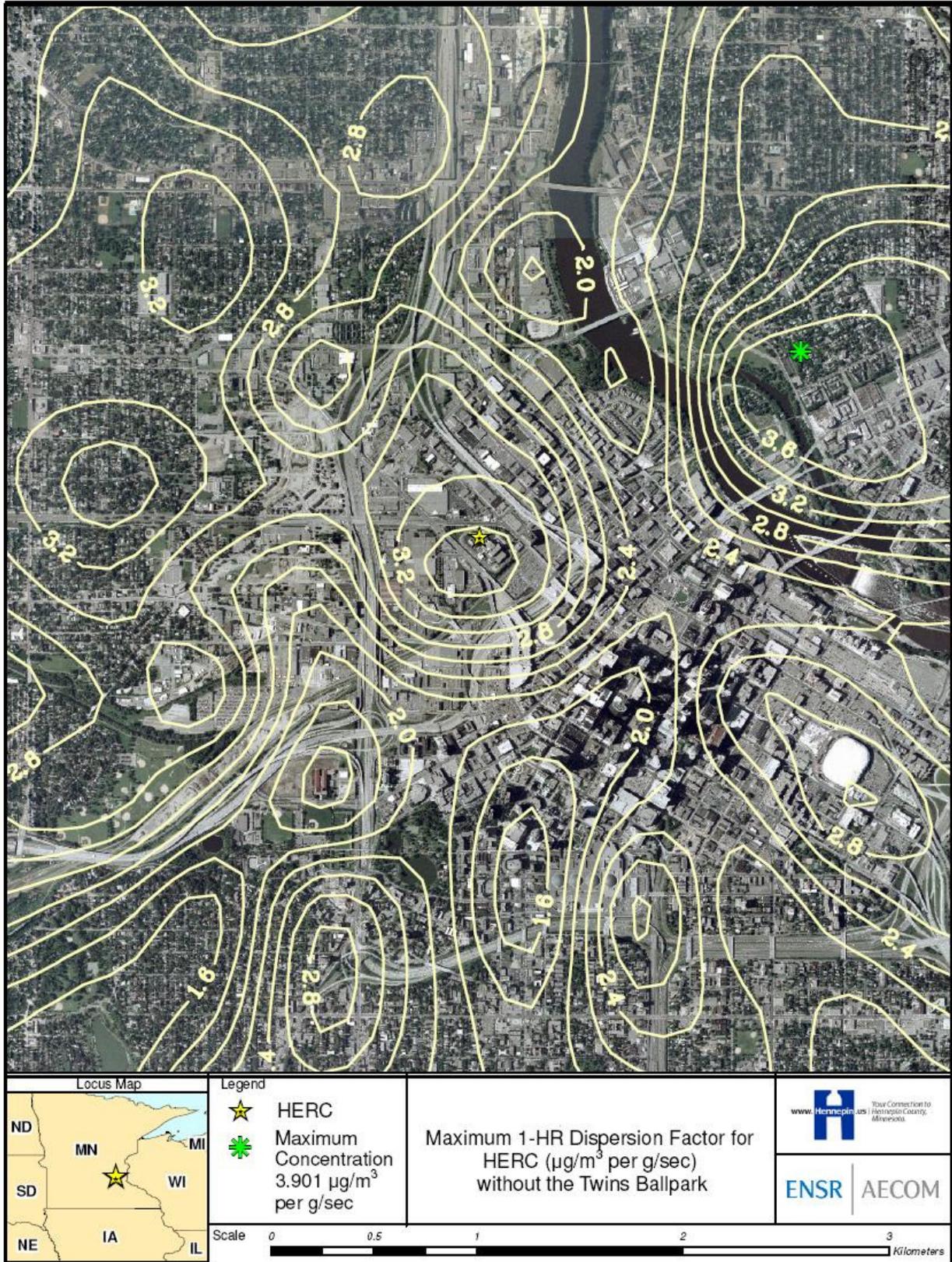


Figure 2-2(b) Maximum 1-Hour Dispersion Factor with the Ballpark

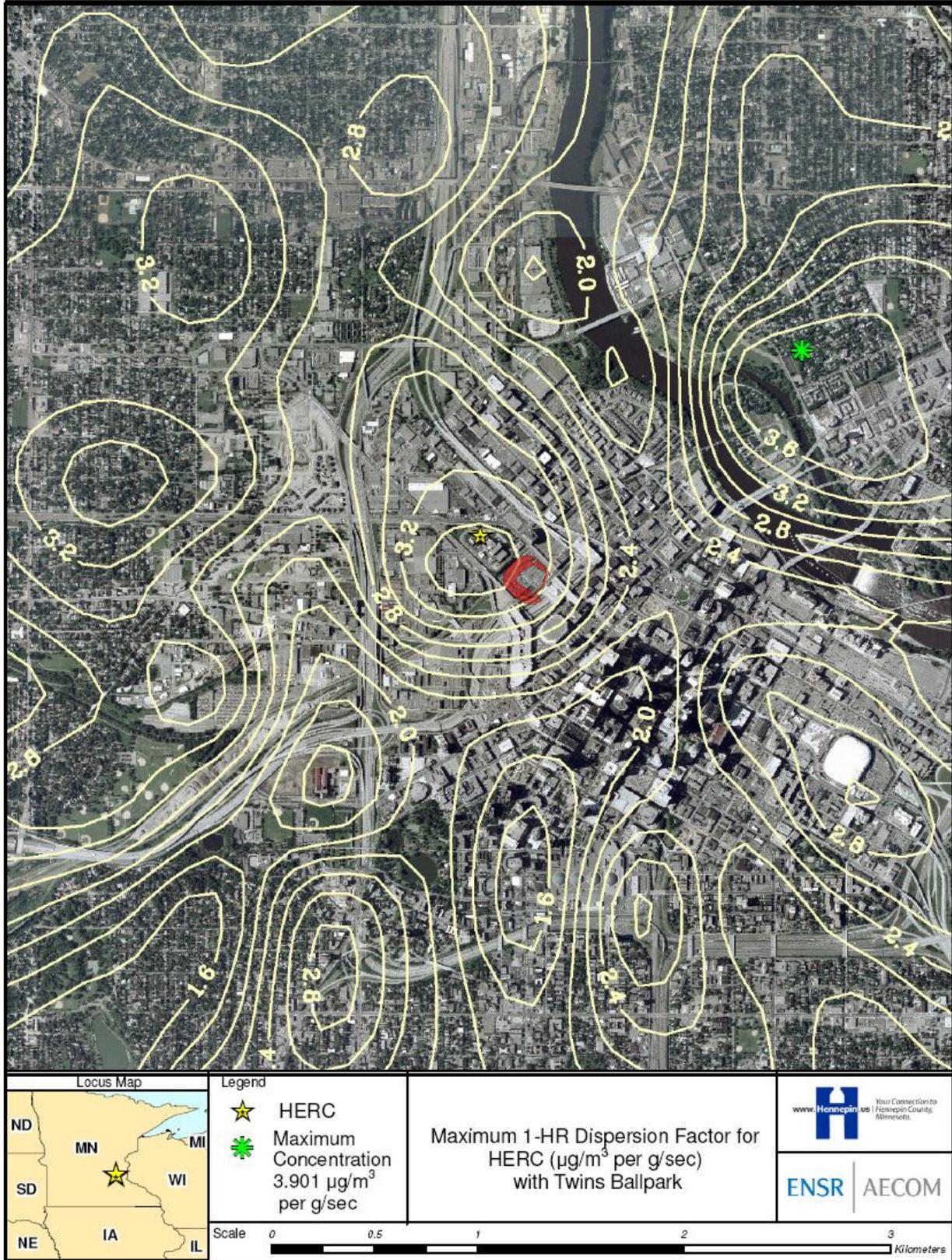


Figure 2-2(c) Five-Year Average Dispersion Factor without the Ballpark

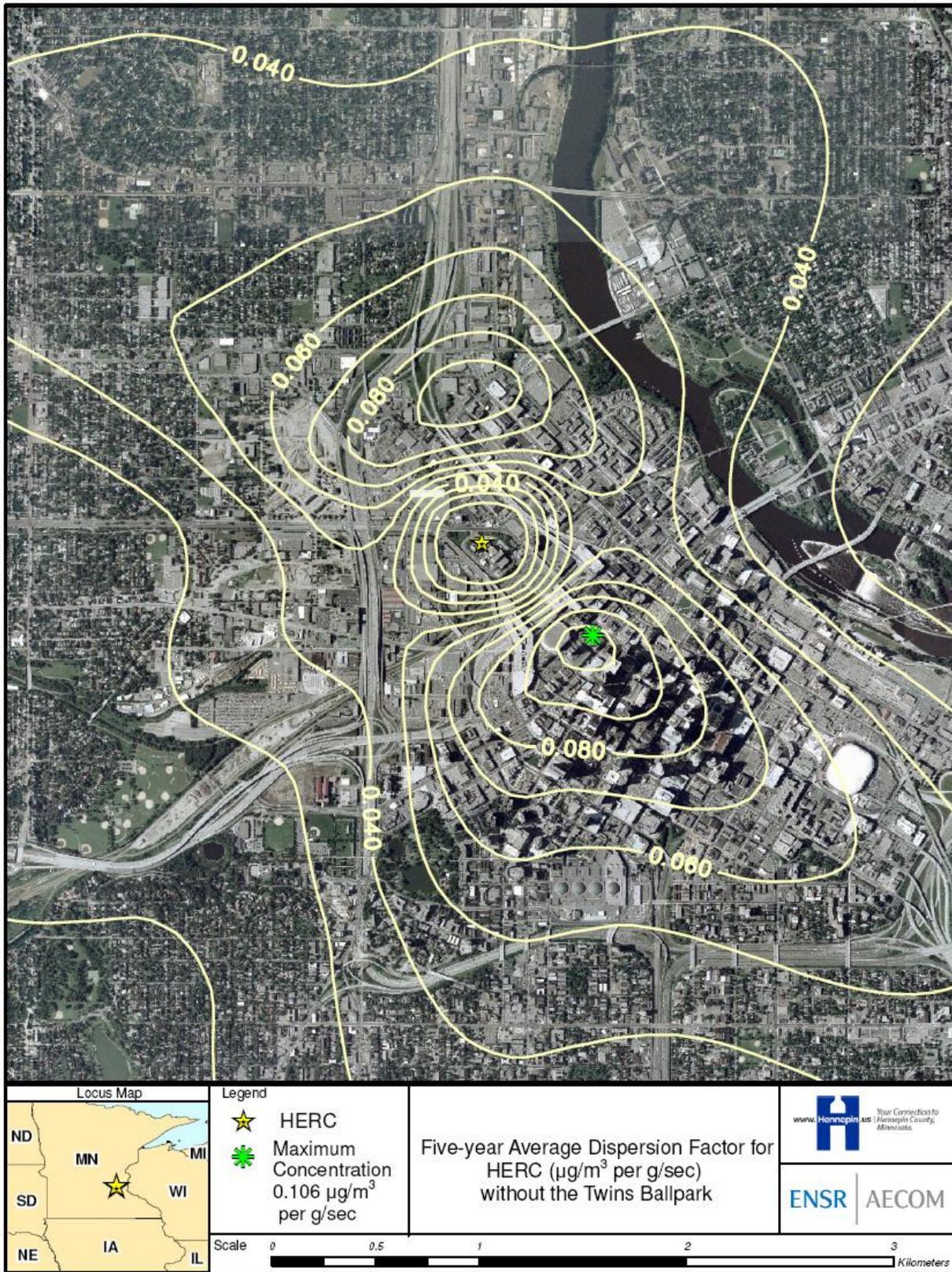
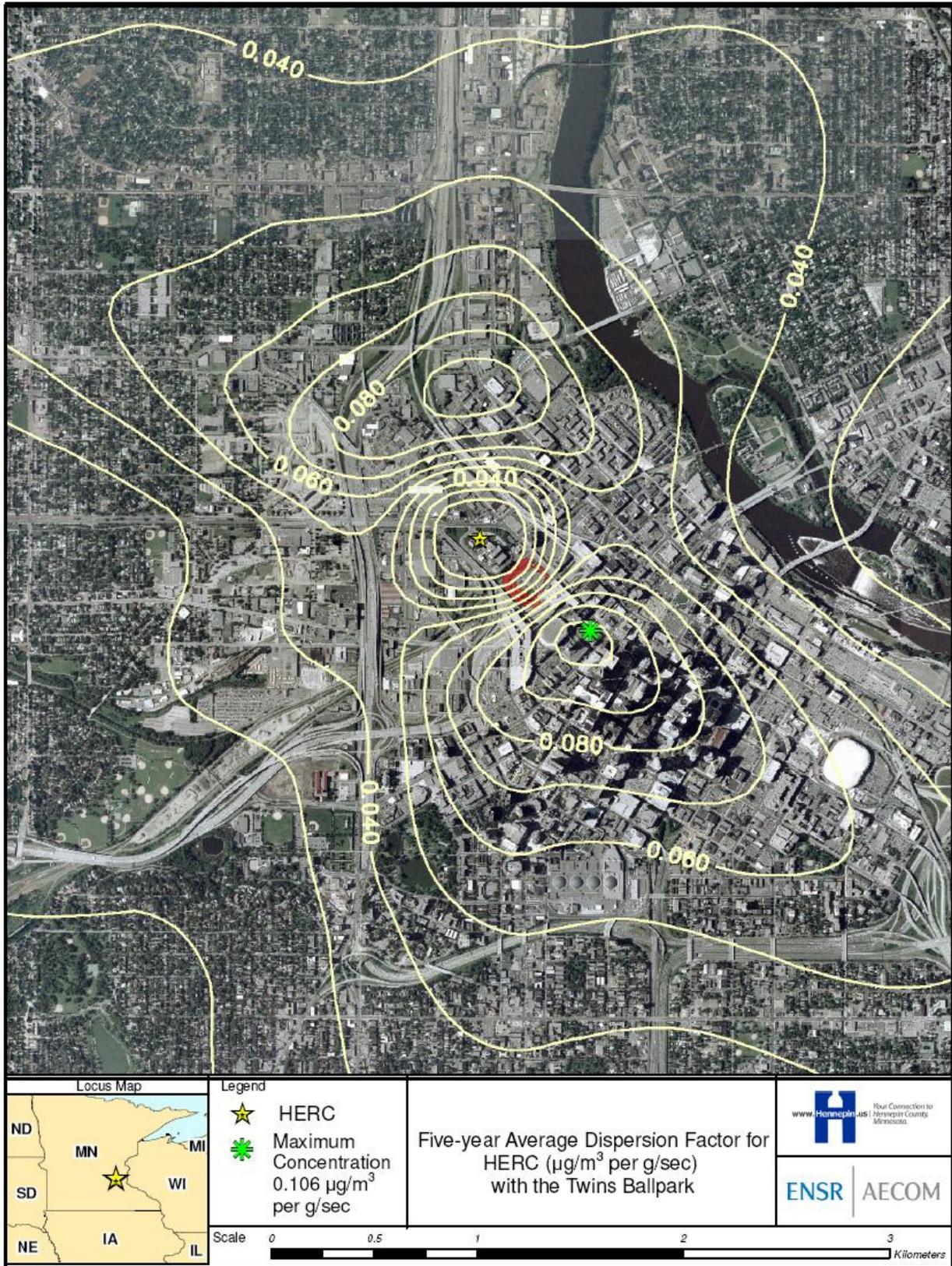


Figure 2-2(d) Five-Year Average Dispersion Factor with the Ballpark



3.0 Potential health risk impacts of HERC emissions on ballpark users

A human health risk assessment (HHRA) was conducted to determine potential risks of HERC emissions on ballpark users who are likely to have the highest exposure. The HHRA was generally conducted in accordance with USEPA's Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (HHRAP; USEPA, 2005a). However, many of the default migration and exposure pathways recommended in HHRAP (such as bioaccumulation in fish and cows, and produce uptake) do not apply to this HHRA, therefore ENSR conducted site-specific risk calculations in accordance with several of the following guidance documents:

- Risk Assessment Guidance for Superfund (RAGS): Volume 1 - Human Health Evaluation Manual (Part A) (USEPA, 1989).
- Exposure Factors Handbook (USEPA, 1997).
- Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites (USEPA, 2002).

The following steps were conducted for the HHRA:

- Hazard identification
- Air dispersion and deposition modeling
- Toxicity assessment
- Exposure assessment
- Risk Characterization

3.1 Hazard Identification

The purpose of the hazard identification step is to identify compounds of potential concern (COPCs) for quantitative evaluation in the HHRA, and to generate emissions estimates for acute and long-term exposures to the selected COPCs.

3.1.1 Identification of COPCs

COPCs were selected based on (1) compounds listed in the MPCA Title V Air Emission Permit #05300400-002 (www.pca.state.mn.us/air/permits/issued/05300400-002-aqpermit.pdf), and (2) additional compounds listed by MPCA in a table entitled "Preliminary Emission Estimates for Calendar Year 2005" (MPCA, 2005).

3.1.1.1 Compounds with Permit Limits

The compounds listed in the MPCA Title V Air Emission Permit include:

Criteria pollutants

- Carbon monoxide
- Sulfur dioxide
- Nitrogen dioxide
- Lead
- TSP
- PM-10
- PM-2.5

These compounds were evaluated by comparing estimated annual average and short-term air concentrations against Minnesota Ambient Air Quality Standards (MAAQS; www.pca.state.mn.us/publications/manuals/nsrtm-chapter06.pdf). Health effects from most of these compounds are mainly through the inhalation route, therefore the comparison with MAAQS is an appropriate approach. In addition to the MAAQS comparison, nitrogen dioxide was also evaluated in the acute inhalation risk assessment. Lead was evaluated for other exposure pathways using USEPA's recommended risk assessment methodology, which is different than the risk assessment approach used for other compounds. Other compounds included in the MPCA Title V Air Emission Permit are:

- Hydrochloric acid
- Cadmium
- Mercury
- Polychlorinated dibenzo-p-dioxins/polychlorinated dibenzo-p-furans (dioxins)

Of these compounds, hydrochloric acid was evaluated through the inhalation pathway. Cadmium, mercury and dioxins were evaluated for both the oral and inhalation exposure pathways.

3.1.1.2 Additional compounds listed by MPCA

In a table provided by MPCA (MPCA, 2005), emissions estimates for additional compounds are provided. A small portion of the compounds are associated with municipal waste combustion, and the majority of the compounds are associated with natural gas usage. The compounds associated with natural gas usage were not included as COPCs because compound emissions arising from solely natural gas usage are likely to be negligible. Natural gas is used sporadically for very short periods. According to information provided by Hennepin County Environmental Services, records of monthly natural gas usage are included in the Annual Air Pollutant Emissions Inventory Reports that HERC submits to the MPCA. Yearly natural gas figures are also included in the Annual Mercury Emissions Reports that HERC submits to the MPCA. HERC burns natural gas at the rate of 850 therms or 85,000 CF per hour to dry out wet fuel, while bringing a unit down, and while bringing a unit back on line. It is rare that two units have come down at the same time, and HERC does not bring two units on line at the same time. HERC could bring one unit up or down while drying out fuel in the other unit. It takes about 2 hours to bring a unit down and 6 to 8 hours to bring a unit back on line. In 2005, HERC used 27,500,000 CF of natural gas or about 22,917 therms/month. At 850 therms/hr this would mean HERC burned natural gas for about 27 hours per month, the equivalent of 2.7 shut down/start up cycles. Because of the short periods of natural gas usage and low emissions, the long-term impacts of compound emissions associated with natural gas are negligible. MPCA has indicated that short-term impacts of nitrogen dioxide should be evaluated during natural gas usage. However, because oxides of nitrogen emissions from natural gas combustion during start-up are much less than both the actual and permitted

levels from HERC when combusting refuse, a separate nitrogen dioxide analysis for gas combustion is not warranted.

A number of compounds are listed as being associated with municipal waste combustion. Some of these compounds (cadmium, hydrochloric acid, mercury and dioxins) are already included as COPCs since they have permit limits. The emissions for these compounds listed in MPCA (2005) are the measured emissions reported by Covanta.

The remaining compounds associated with municipal waste combustion are included as COPCs:

- Arsenic
- Chromium
- Nickel

Arsenic, chromium and nickel were evaluated for both the oral and inhalation pathways.

MPCA also requested that sulfuric acid be included as a COPC. It was necessary to make some assumptions about emissions for sulfuric acid, since there are no permitted emission levels or measurements for sulfuric acid in the HERC. The Environmental Assessment Worksheet (see <http://www.pca.state.mn.us/news/eaw/owef-eaw.pdf>) for the Olmsted County municipal waste incinerator indicated that sulfuric acid emissions are 3% of the SO₂ emissions. This factor has been applied in to estimate sulfuric acid emissions from HERC.

3.1.2 Emissions Estimates

Both chronic and acute emissions estimates were developed for the various COPCs. Details of the emissions estimation are provided in Appendix B in the form of an Excel spreadsheet.

3.1.2.1 Actual Emissions

Measured air emissions were based on the three most recent emission tests conducted in 2004, 2005 and 2006 (see separate tabs for each unit and COPC, e.g., Unit1_Hg, Unit_Hg, Unit1_Cd, etc. in Appendix B). This HHRA considers two averaging periods representing chronic and acute exposure. For chronic exposure, in this case corresponding to up to 20 years, a long-term average emission rate is required. The long-term average emission rates were based on the arithmetic average of the three measurement programs. For acute exposure, the maximum measured emission rate among all nine of the measurement runs was applied. This method of estimating maximum 1-hour concentrations is highly conservative because it inherently assumes that the highest measured emission rate corresponds to the worst-case meteorological condition.

Not all COPCs are tested because several COPCs are not subject to permit limits. There are some COPCs which MPCA has determined to be associated with municipal waste combustion (arsenic, chromium, nickel, PM_{2.5} and sulfuric acid) that do not have permit limits or testing requirements. For these COPCs conservative emission estimates developed by MPCA (MPCA, 2005) were applied (contained in an MPCA spreadsheet: 2705300400_emis.xls). MPCA calculated these emission rates using software developed by USEPA, called Regional Air Pollutant Inventory Development System (RAPIDS; <http://www.glc.org/air/rapids/rpdsover.html>). For these COPCs that were not tested, the maximum 1-hour emission rate was estimated as recommended by MPCA by multiplying the long-term rate by a factor of factor of 1.17, based on the average number of online hours reported in 2003, 2004 and 2005.

For criteria pollutants evaluated in this assessment, the Air Pollutants Emissions Inventory Reports for HERC submitted to MPCA for 2003, 2004 and 2005 were applied. Emission rates for AERMOD

modeling (g/sec) were estimated by dividing the annual emissions by the number of hours of operation per year listed in these reports.

There are 210 individual dioxin/furan congeners, and among these, 17 congeners are routinely assessed in risk assessment as they are considered to pose the greatest risks. These 17 congeners may be expressed as toxic equivalents (TEQ) of 2,3,7,8-tetrachlorodibenzodioxin (2,3,7,8-TCDD) using toxicity equivalency factors (TEF) that have been developed by the World Health Organization (Van den Berg, 2006). The sum of TEQ among all measured congeners was used in this HHRA.

To compute emissions in terms of mass emission rates, the measured stack gas concentrations were multiplied by the full load flow rate corrected for temperature of the exhaust gas. The resultant measured long-term and short term emission rate for each of the COPCs is provided in Tables 3-2 and 3-3, respectively.

3.1.2.2 Permitted Emissions

Permit limits documented in MPCA Title V Air Emission Permit #05300400-002 were applied. For COPCs for which there are no permit limits, MPCA emission estimates were applied. To compute emissions in terms of mass emission rates, the limits, which are principally listed in terms of stack gas concentration, were multiplied by the full load flow rate corrected for actual percent oxygen and temperature of the exhaust gas. As recommended by MPCA, for COPCs that only have long-term emission limits, the long-term emission rate was multiplied by a factor of 1.17 to estimate maximum short-term emissions, based on the average number of online hours reported in 2003, 2004 and 2005.

The permit limits are for total dioxin/furan emissions rather than total 2,3,7,8-TCDD TEQ emissions which are required for the HHRA. To estimate the permitted total TEQ emissions, the permitted dioxin/furan emission rate was multiplied by the average ratio of total TEQ to total dioxin/furan (0.037) as determined from the 2004, 2005 and 2006 emission tests. The calculation of this ratio is shown in Table 3-1.

3.2 Air Dispersion/Deposition Modeling

Air dispersion and deposition modeling was conducted to estimate air concentrations and deposition rates to support the HHRA. Modeling was conducted with the recently promulgated AERMOD model (Version 04300) in accordance with USEPA Guideline on Air Quality Models (GAQM; as incorporated in Appendix W of 40 CFR Part 51).

The AERMOD modeling analysis was conducted with guidance provided in the AERMOD *User's Guide* (USEPA, 2004) and Addendum (USEPA, 2003a), including USEPA recommendations for conducting modeling in support of health risk assessment as provided in HHRAP (USEPA, 2005a).

The source parameters, building downwash parameters and meteorological data applied for the HHRA modeling are the same as described for the comparative modeling in Section 2, above. Other aspects of the modeling varied as outlined in this section.

3.2.1 Source Data

The modeling was performed for 0.5 g/sec emissions from each stack such that the total is a unit (1 g/sec) emission rate. Emission rates for specific pollutants were applied with post-processing spreadsheets to estimate concentration and impacts from the facility.

In addition to the physical stack parameters and exhaust stack parameters, particle size data on stack emission are required to perform deposition modeling.

Actual data on the size distribution of particulate emissions for HERC are not available. For a recent risk assessment conducted on behalf of MPCA for the Olmsted waste-to-energy facility, ENSR reviewed available particle size data and data from a facility in Wurzburg, West Germany, with a fabric filter used. The same particle size distribution was applied in this assessment. In accordance with the HHRAP, two different particle size distributions were modeled. The distribution of particles by mass was used to represent all particulate species with high boiling points (cadmium, chromium and nickel). For dioxin/furan, a semi-volatile organic and low boiling point metals (arsenic and mercury) that tend to vaporize during combustion and condense on the surface of emitted fly-ash the size distribution is represented by the surface area rather than the mass of particulate. Guidance provided in the HHRAP (in Section 3.2.3 of the HHRAP - http://www.epa.gov/earth1r6/6pd/rcra_c/protocol/volume_1/vol1pro.htm), assuming constant density spherical particles (particle density of 1 g/cm³), has been used to estimate the surface area-weighted distribution from the mass weighted distribution. The distributions are provided in Table 3-6.

3.2.2 Model Receptors

AERMOD requires specification of receptor locations, within a defined study area, at which the model computes air concentrations and deposition rates. Model receptors were placed throughout the ballpark and segregated into two groups representing the playing field and the grandstands. Receptor locations are shown in Figure 3-1.

3.2.3 Model Options

AERMOD was applied with the “TOXICS” option to facilitate computation of particle deposition (“WDEP” and DDEP”, wet and dry components) and vapor (gaseous) deposition (wet and dry components). The “URBANOPT” option was specified with a population of 382,618 (2000 Census data) and the default surface roughness of 1.0 m.

Five years of processed meteorological data were combined into a single meteorological data file for input to AERMOD to compute five-year averages of air concentrations and deposition rates. As such, AERMOD was applied with the “PERIOD” averaging time option. The use of this option facilitates obtaining long-term average deposition rates and air concentrations when using the multi-year meteorological data files. The only exception was for modeling annual average impacts of criteria pollutants where each of the 5 years was modeled separately.

For particle deposition, “Method 1” specified in the User’s Guide Addendum was used (USEPA, 2003a). Method 1 is recommended for particle size distributions where the mass of particles greater than or equal to 10 µm exceeds 10 percent as is the case for the proposed distribution.

3.2.4 Derivation of Exposure Point Concentrations through AERMOD Dispersion and Deposition Modeling

AERMOD was applied to determine ambient air concentrations to evaluate criteria pollutants and exposure point concentrations for evaluating health risk of COPC. Exposure point concentrations included the maximum 1-hour and average concentrations in air, concentrations of deposited material in soil and accumulated dust-fall in the stands, and concentrations of deposited material on food consumed by people in the stands. AERMOD was run separately to evaluate criteria pollutants and estimate the various types of exposure point concentrations.

Criteria Pollutants

Modeling for criteria pollutants was based on all 5 years of meteorological data and the highest modeled value among field and elevated grandstand receptors for appropriate averaging times of 1-hour, 3-hour, 8-hour, 24-hour, 1-month (as a conservative surrogate for 3 month) were applied. For the annual average the five years were run separately and the maximum annual concentration was applied.

Airborne Concentrations for Ballpark Users

Air modeling was conducted for periods from 1986-1990 (coincident with the meteorological data). This was achieved by obtaining the Twins schedule for these years and turning on the emissions only during specified hours on days when a game was played. This included the scheduled 81 games per season from early April through September/early October, depending on the year. For each year, 11 additional games were added to the end of the regular playing season assuming that the Twins play the maximum number of home games for the 5-game Division Series (3 games at home), the 7-game American League Championship Series (4 home games) and the 7-game World Series (4 home games). This extended the season through October. Day and night games were distributed according to information provided by the Twins indicating 62 night games and 19 day games over the 81 game schedule. Post season games were assumed to take place at night. Fans were assumed to spend 6 hours at the ballpark and players and staff were assumed to spend 9 hours at the ballpark. Table 3-7a and 3-7b lists the average and highest modeled long-term and maximum 1-hour concentrations of COPCs for each type of receptor assuming measured emissions and permit limits, respectively. The average concentration is the arithmetic average over all receptors of the same type and the highest is the highest concentration among these receptors. For receptors on the playing field representing players, the highest 1-hour concentration is only about 10% higher than the average and the highest long-term concentration is about 50% higher than the average. For the receptors in the stands (representing concession workers and fans) the highest 1-hour concentration is about 30% higher than the average and the highest long term concentration is more than double the average concentration.

Deposition onto Food Consumed at the Ballpark

It is assumed that during the course of a game that a fan will eat food that is exposed to dry deposition of particulate-borne HERC pollutants for four hours. Wet deposition is not considered because it is very unlikely that food would not be covered. During each game the food and beverage consumed is assumed to have the equivalent area of a standard 11-in x 15-in cafeteria-style tray. (To put this in context this could correspond, for example, to 2 pretzels, 2 hot dogs, 1 pizza slice, 1 order French fries and a large drink cup.) The average deposition onto this area during a 4 hour period is computed by modeling the total deposition that would occur over the 5 seasons while fans are at the ballpark (6 hours per game x 92 games/year x 5 years = 2760 hours), computing the hourly deposition rate by dividing the 5 season deposition by 2760 and then multiplying by the 4 hour duration of food exposure. Table 3-8 lists the highest and average food deposition among fans in the ballpark stands. The average deposition is the arithmetic mean of the deposition over all of the receptors in the stands shown in Figure 3-1. The highest deposition is the highest modeled deposition among all of these receptors and coincides with the location of the highest modeled long-term concentration.

Deposition on Ballpark Surfaces

It is assumed that fans in the stands ingest a prescribed mass of accumulated dust from surfaces to which they come in contact. The concentration of COPCs in the dust has been estimated by dividing the modeled rate of deposition associated with HERC emissions during the baseball

season to the ambient deposition due to particulate already present in the air. The rate of HERC deposition was modeled by simulating the entire playing season, extending from April through October for each of the 5 years. Deposition onto the open stands included dry and wet deposition and deposition onto covered stands (near the top of the upper deck) included only dry deposition. Deposition during the off-season was not considered because it is assumed that the park surfaces will be cleaned in the spring, prior to opening day.

The rate of deposition from the ambient air was based on ambient measurements of TSP (Total Suspended Particulate defined as particles less than 30 μm in aerodynamic diameter), PM_{10} (particles less than 10 μm) and $\text{PM}_{2.5}$ (particles less than 2.5 μm). The average concentrations of the particulate constituents were then used to estimate the average concentration of large particles from 10 μm to 30 μm , coarse particles from 2.5 μm to 10 μm , and fine particle less than 2.5 μm . The rate of deposition of the ambient particulate was conservatively underestimated by considering only dry deposition and ignoring the contribution of wet deposition. The rate of dry deposition was computed by multiplying the concentration in each particle size category by the corresponding deposition velocity (m/sec) determined from Sehmel (1984). The calculation of background deposition is shown in Table 3-9 and the resultant COPC concentration (mg/Kg) in deposition on ballpark surfaces is provided in Table 3-10.

Deposition on the Playing Field

It is assumed that players on the field ingest a prescribed amount of soil. Dry and wet deposition of COPCs to the playing field was computed for the entire 5-year period. Because it is possible that runoff from the stands could be used for field irrigation, it is conservatively assumed that all of the deposition on the open portions of the stands are distributed uniformly on the field surface. It is also assumed that the deposited material collects on the field and dugout surface with no attenuation due to run-off or other removal mechanisms. Because most of the ingestion will take place from contact with the grass and surface dust in the dugout, the effective depth of the soil layer to which players are being exposed is conservatively assumed to be 0.02 cm, which is one percent of the 2 cm depth recommended by USEPA (USEPA, 2005a) for deposition in soil. Given that it is assumed that a player will play for 20 years, the soil concentration after 10 years is an estimate of the average concentrations to which the player would be exposed. The 10-year soil concentration was computed by doubling the modeled soil concentration accumulated after 5 years. The result of this calculation is provided in Table 3-11.

3.3 Exposure Assessment

The purpose of the exposure assessment is to estimate the magnitude and frequency of potential human exposure to COPCs being emitted. The first step in the exposure assessment process is determining potential receptors (i.e., people who may contact the impacted environmental media of interest). Potential exposure scenarios identifying appropriate environmental media and exposure pathways for current and potential future site uses and receptors are then developed. In this case, the use of the site as a ballpark and activities within the ballpark were considered. The HHRA considered that COPCs from HERC are emitted and particulate-bound COPCs are deposited onto the playing field and other surfaces that people can contact. Particles can also deposit onto food and drink that are prepared in open air areas of the ballpark.

To estimate the potential risk to human health that may be posed by the presence of COPCs in environmental media in the study area, it is first necessary to estimate the potential exposure dose of each of the COPCs for each receptor. The exposure doses are combined with the toxicity values to estimate potential cancer and noncancer risks for each receptor.

3.3.1 Identification of receptors and exposure pathways

While there are various types of receptors who could be exposed at a ballpark, the ones that are likely to receive the highest exposures were considered in this HHRA. Three sets of receptors were considered based on their exposure potential; these are (1) ballpark staff, (2) ballplayers, and (3) season ticket holders. Table 3-12 lists exposure assumptions for these receptors. The receptors and selection of exposure assumptions are discussed below. It is also possible that HERC COPCs could accumulate on ballpark surfaces over time, and that there could be run-off from rain, snow, washing the decks, and watering the field. These kinds of exposure pathways are discussed qualitatively.

3.3.1.1 Ballpark Staff

Ballpark staff include full time staff, such as groundskeepers, food service vendors and maintenance workers. They could be exposed to COPCs emitted from HERC through inhalation of COPCs present in air. They could also be exposed through incidental ingestion of soil. Dermal contact with COPCs in soil or other surfaces could occur; however, exposure through dermal contact is likely to be minimal compared to other exposure pathways. Therefore, the dermal contact pathway was not evaluated quantitatively. This approach is consistent with HHRAP (USEPA, 2005a), which does not quantitatively evaluate dermal exposure. Ballpark staff could also be exposed through food and drink; however, it is likely that season ticket holders could have greater exposure through food ingestion, and therefore this exposure pathway is evaluated only for season ticket holders.

Table 3-12 lists exposure assumptions for ballpark staff. The exposure assumptions listed are conservative default assumptions that USEPA has developed for outdoor workers. It is assumed that the exposure frequency for ballpark staff is 225 days per year (USEPA, 2002). This value is based on data from the U.S. Census Bureau and represents the average number of days worked per year by male and female workers engaged in activities likely to be similar to those of the outdoor worker receptor (USEPA, 2002). It is assumed that ballpark staff have an exposure duration of 25 years, which is the 95th percentile value for job tenure for men in the manufacturing sector (USEPA, 1991; 2002). The exposure time is assumed to be an average of 8 hours per day over the course of the year. It is possible that certain days could be longer (such as days when games are being played), however an average of 8 hours per day throughout the year is reasonable. To evaluate the soil ingestion exposure pathway, it was assumed that the ballpark staff has a soil ingestion rate of 100 mg/day. This rate is recommended in USEPA's Soil Screening Levels Guidance (USEPA, 2002) for an outdoor worker.

3.3.1.2 Ballplayer

Potential exposure to a long-term Minnesota Twins ballplayer was evaluated. Similar to the ballpark staff, it was assumed that a ballplayer could be exposed through inhalation and incidental ingestion of soil.

Table 3-12 lists exposure assumptions for a ballplayer. As an upper limit, it was assumed that a ballplayer is present at the ballpark for 92 days out of the year, during the season running from April through September and assuming that the team makes it to the World Series. It is assumed that the ballplayers have an exposure duration of 20 years. The exposure time is assumed to be an average of 8 hours per day. To evaluate the soil ingestion exposure pathway, it was assumed that the ballplayer has a soil ingestion rate of 100 mg/day. This rate is recommended in USEPA (2002) for an outdoor worker.

3.3.1.3 Season ticket holder

It was assumed that season ticket holders (adult and child) could be present at games throughout the season. They could be exposed to COPCs emitted from HERC through inhalation of COPCs present in air. They could also be exposed through incidental ingestion of dust collecting on the seats and other surfaces. It was also assumed that season ticket holders could be exposed through COPCs deposited on food and drink.

Table 3-12 lists exposure assumptions for adult and child season ticket holders. The exposure assumptions listed are conservative default assumptions that USEPA has developed for residential exposure. For residential exposure scenarios, exposure is typically evaluated for a child of 0 to 6 years of age, and an adult. It is possible that this child age group may be young for typical baseball game attendants; however, use of this age group results in a conservative assessment of potential risks to children. As an upper limit, it was assumed that season ticket holders are present at the ballpark for 92 days out of the year, during the season running from April through September and assuming that the team makes it to the World Series. It is assumed that the child has an exposure duration of 6 years and the adult has an exposure duration of 24 years. This is a combined exposure duration of 30 years, which is the national upper-bound (90th percentile) time at one residence (USEPA, 1989). The exposure time is assumed to be an average of 4 hours per day during a game. To evaluate the dust ingestion exposure pathway, the default soil ingestion rates for children and adults of 200 mg/day and 100 mg/day, respectively, were used (USEPA, 2002).

The pathway involving deposition onto food surfaces is not a typical exposure pathway that is evaluated in USEPA risk assessments. The food pathways evaluated in HHRAP (USEPA, 2005a) involve uptake and bioaccumulation in food, such as fish, beef and dairy milk that are grown in the area. However, it is possible to model deposition onto food surfaces using air dispersion modeling and conservative assumptions regarding the mass of compound that could deposit onto food surfaces. In order to evaluate this pathway, it was necessary to assume that COPCs could deposit onto a surface that is covered with food (such as hotdogs, fries, drinks, etc.). Rather than evaluating individual food items, it was assumed that COPCs could deposit onto the surface of a standard size cafeteria tray that is covered with food. A standard tray is approximately 15 in. x 11 in (165 in² or 0.11 m²). It was conservatively assumed that food items for a child and adult could cover the whole tray. Deposition onto the tray could occur over the 4 hours that the season ticket holder is assumed to stay at the ballpark.

3.3.2 Quantification of Potential Exposures

To estimate the potential risk to human health that may be posed by the presence of COPCs at the site, it is first necessary to estimate the potential exposure dose of each of the COPCs. The exposure dose is defined as the amount of COPC taken into the receptor. The air dispersion and deposition modeling results were used in conjunction with exposure equations to develop exposure doses. Appendix D contains risk spreadsheets for the various receptors and exposure pathways.

Exposure doses are defined differently for potential carcinogenic and noncarcinogenic effects. The Chronic Average Daily Dose (CADD) is used to estimate a receptor's potential intake from exposure to COPCs with noncarcinogenic effects. According to USEPA (1989), the CADD should be calculated by averaging the dose over the period of time for which the receptor is assumed to be exposed. Therefore, the averaging period is the same as the exposure duration. For COPCs with potential carcinogenic effects, however, the Lifetime Average Daily Dose (LADD) is employed to estimate potential exposures. In accordance with USEPA (1989) guidance, the LADD is calculated by averaging exposure over the receptor's assumed lifetime (70 years). Therefore, the averaging period is the same as the receptor's assumed lifetime. The standardized equations for estimating a receptor's average daily dose (both lifetime and chronic) are presented below. For air inhalation

risk estimates, the equation is different in that the air concentration is used directly with the inhalation toxicity values, therefore there is no need to calculate average daily doses.

3.3.2.1 Estimating Potential Exposure from Ingestion of Soil and Dust

Average Daily Dose (Lifetime and Chronic) Following Incidental Ingestion of Soil and Dust (mg/kg-day):

$$ADD = \frac{CS \times IR \times EF \times ED \times CF}{BW \times AT}$$

where:

ADD	=	Average Daily Dose (mg/kg-day)
CS	=	Soil concentration (mg/kg soil)
IR	=	Ingestion rate (mg soil/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (year)
CF	=	Unit conversion factor (kg soil/10 ⁶ mg soil)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

3.3.2.2 Estimating Potential Exposure from Ingestion of Food and Drink

The exposure dose from ingesting COPCs present in food and drink was calculated using a modification of the soil ingestion equation. Rather than evaluating individual food items, it was assumed that COPCs could deposit onto the surface of a standard size cafeteria tray that is covered with food. A standard tray is approximately 15 in. x 11 in (165 in² or 0.11 m²). Deposition onto the tray could occur over the 4 hours that the season ticket holder is assumed to stay at the ballpark. The modeling results provided the mass (mg) of the COPCs that could be ingested per day.

Average Daily Dose (Lifetime and Chronic) Following Incidental Ingestion of Food and Drink (mg/kg-day):

where:

$$ADD = \frac{MC \times EF \times ED}{BW \times AT}$$

ADD	=	Average Daily Dose (mg/kg-day)
MC	=	Mass of COPC ingested (mg of COPC/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (year)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

3.3.2.3 Estimating Potential Exposure via Inhalation

For inhalation risk estimates, the equation is different in that an adjusted air concentration is used directly with the relevant cancer or noncancer toxicity values, and it is not necessary to calculate average daily doses. The air concentration was modified to account for exposure duration (exposure frequency is already accounted for in the modeling).

Air concentration adjusted for receptor's exposure duration (µg/m³):

$$AAC = \frac{AC \times ED}{AT}$$

where:

AAC	=	Adjusted Air Concentration ($\mu\text{g}/\text{m}^3$)
AC	=	Air concentration estimated from deposition modeling ($\mu\text{g}/\text{m}^3$)
ED	=	Exposure duration (year)
AT	=	Averaging time (days)

3.3.2.4 Exposure Point Concentrations

Concentrations of COPCs in air, soil, dust and food were estimated using the air dispersion modeling based on Permit Limits and measured emissions. Section 3.2 discusses the methods used to estimate these concentrations, and the accompanying tables show the calculation of exposure point concentrations for the various receptors. As shown in Section 3.2, two types of ballpark staff were evaluated – groundskeepers and food service vendors. The groundskeepers were assumed to be exposed to air and dust on the playing field, and the food service vendors were assumed to be exposed to air and dust in the stands. Since the exposure point concentrations for the food service vendors were generally higher, this receptor was evaluated as the ballpark staff receptor. Tables 3-7 a and b and 3-8 a and b also show highest and average concentrations for various types of receptors. Because the risk calculations used the highest concentration the risk is overestimated for the average player and fan. For receptors on the playing field the highest modeled values are about 25% greater than the average over the entire playing field. Given that players are on the field about half the time and the other half either in the dugout, at bat or on the bases, the average over the entire field is more representative than the highest receptor value. Likewise for the stands the highest value is more than twice the average value. Tables 3-13 through 3-15 show exposure point concentrations for ballpark staff, ballplayers and season ticket holders based on HERC emissions at the permit limits and Tables 3-16 through 3-18 show exposure point concentrations based on actual measured HERC emissions. The air concentrations for sulfuric acid were estimated assuming that sulfuric acid emissions are 3% of the SO_2 emissions as described in Section 3.2.4. These tables do not include exposure concentrations for lead because lead is evaluated separately in accordance with USEPA recommendations (USEPA, 2005).

3.3.3 Pathways Related to Accumulation and Run-off

It is also possible that HERC COPCs could accumulate on ballpark surfaces over time, and that there could be run-off from rain, snow, washing the decks, and watering the field. The soil and dust concentrations calculated in the deposition modeling assume that deposition is happening over a period of 5 years. According to information provided by HOK Sport (who are in charge of developing the ballpark), stadiums can go for 10 years or more without resodding. Therefore, the soil and dust exposure point concentrations used for the various receptors account for accumulation over time.

According to information provided by HOK Sport, run-off from rain, snow, washing the decks and watering the field goes into stormwater drains, and then piped into some type of basin, cistern or tank. It is possible that some of the water could be used in the following ways, but this has not been decided: wash the concourses and seating areas, or water the grass. Any water not re-used on site will be discharged to the Minneapolis storm sewer system. Any impact of HERC emissions on the storm sewer system and ultimate discharge to the Mississippi is likely to be negligible.

3.4 Toxicity Assessment

The purpose of the toxicity assessment is to identify the types of health effects a compound may potentially cause, and to define the relationship between the dose of that compound and the likelihood or magnitude of a health effect (response).

Chronic health effects are characterized by USEPA as potentially carcinogenic or noncarcinogenic. Combining the results of the toxicity assessment with information on the magnitude of potential human exposure (discussed in Section 3.3, Exposure Assessment) provides an estimate, usually conservative, of potential health risk.

In addition to chronic health effects, acute inhalation of maximum short-term air concentrations was also evaluated. Chronic and acute toxicity values were identified for the relevant COPC at this facility. As noted earlier, the criteria pollutants modeled in this HHRA (carbon monoxide, sulfur dioxide, nitrogen dioxide, lead, PM-10 and PM-2.5) were evaluated by comparing them against the MAAQS. Nitrogen dioxide was also evaluated for acute risks. Toxicity values were identified for the remaining COPCs.

3.4.1 Chronic Toxicity Values

Both potentially carcinogenic and noncarcinogenic health effects were evaluated in the HHRA. Published toxicity values used in this HHRA were selected in accordance with recommendations from the MPCA and Minnesota Department of Health (MDH), and generally followed the following hierarchy:

- Minnesota Department of Health (MDH),
- USEPA's Integrated Risk Information System (IRIS) (USEPA, 2006), and
- California EPA (CalEPA 2005a and 2005b).

3.4.1.1 Inhalation Toxicity Values

The toxicity values used to evaluate potential carcinogenic health effects resulting from long-term inhalation exposure to COPCs are called unit risk factors, and are expressed in units of the inverse of micrograms of the compound per cubic meter of air ($\mu\text{g}/\text{m}^3$)⁻¹. Unit risk refers to the upper bound excess cancer risk from a continuous lifetime exposure to a compound at one microgram per cubic meter ($1 \mu\text{g}/\text{m}^3$) in air. The typical toxicity value used to evaluate potential noncarcinogenic health effects resulting from long-term inhalation exposure to COPCs is called a reference concentration (RfC) and is expressed in units of $\mu\text{g}/\text{m}^3$. The RfC is defined as an estimate, with uncertainty spanning perhaps an order of magnitude, of a continuous inhalation exposure to the human population, including sensitive subgroups, that is likely to be without an appreciable risk or deleterious effects during a lifetime. It can be derived from a no observed adverse effects level (NOAEL), lowest observed adverse effects level (LOAEL), or benchmark concentration, with uncertainty factors generally applied to reflect limitations on the scientific data available. MDH has also developed chronic health risk values (HRV); the HRV is defined as the concentration of a compound or defined mixture of compounds in ambient air, at or below which the compound is unlikely to cause an adverse health effect to the general public when exposure occurs daily throughout a person's lifetime (<http://www.health.state.mn.us/divs/eh/air/rules.htm>).

Table 3-19 lists inhalation toxicity values for chronic cancer and noncancer effects for the COPCs that are evaluated for the inhalation pathway. For dioxins, MPCA uses an inhalation unit risk factor of $400 (\mu\text{g}/\text{m}^3)^{-1}$ for 2,3,7,8-TCDD toxic equivalents based on extrapolation from the oral slope factor, and assuming an inhalation rate of $20 \text{ m}^3/\text{day}$ and body weight of 70 kg (<http://www.health.state.mn.us/divs/eh/risk/guidance/dioxinmemo2.html>).

3.4.2 Oral Toxicity Values

The toxicity values used to evaluate potential carcinogenic health effects resulting from long-term oral exposure to COPCs are called Cancer Slope Factors (CSFs). A CSF is generally defined as an upper bound, approximating a 95 percent confidence limit, on the increased cancer risk from a lifetime exposure to a compound or defined mixture of compounds. This estimate, usually

expressed in units of proportion (of a population) affected per mg/kg/day, is generally reserved for use in the low-dose region of the dose-response relationship, that is, for exposures corresponding to risks less than one in 100. This number is derived from a mathematical extrapolation model that uses toxicologic data specific to each carcinogen.

The CSF for the ingestion route is expressed in units of the inverse of milligrams of the compound or defined mixture of compounds per kilogram of body weight per day (mg/kg-day)⁻¹ (<http://www.health.state.mn.us/divs/eh/air/rules.htm>).

The typical toxicity value used to evaluate potential noncarcinogenic health effects resulting from long-term oral exposure to COPCs is called a reference dose (RfD). The RfD is defined as an estimate, with uncertainty spanning perhaps an order of magnitude, of a daily oral exposure to the human population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a no observed adverse effects level (NOAEL), lowest observed adverse effects level (LOAEL), or benchmark dose, with uncertainty factors generally applied to reflect limitations of the scientific data available. The RfD is expressed in units of milligrams of the compound or defined mixture of compounds per kilogram of body weight per day (mg/kg-day). MDH has also developed multimedia health risk values (MHRV), which are defined as the total daily dose of a compound or defined mixture of compounds that results from an emission to ambient air, at or below which is unlikely to cause an adverse health effect to the general public over a lifetime exposure. Total daily dose is the sum of the exposure doses calculated from applicable inhalation or non-inhalation exposure pathways. The MHRV is expressed in units of mg/kg-day (<http://www.health.state.mn.us/divs/eh/air/rules.htm>). It is noted that the oral CSF for 2,3,7,8-TCDD developed by MDH of 1.4×10^6 /mg/kg-day is 10-fold higher (i.e., 10-fold more conservative) than USEPA's CSF of 1.5×10^5 /mg/kg-day. If the USEPA CSF were used, then the cancer risk estimate would be 10-fold lower.

Table 3-20 lists oral toxicity values for chronic cancer and noncancer effects for the COPCs that are being evaluated for the oral pathway.

3.4.3 Toxicity Assessment for Lead

U.S. EPA has not derived toxicity values for lead due to uncertainties about the health effects and dose-response associated with exposures to lead. Based on findings that neurobehavioral effects in young children occur at exposure levels below those that have caused cancer in laboratory animals, an Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children has been developed by USEPA (US EPA, 1994; the latest IEUBK software (IEUBKwin v1.0 build 263) is dated December, 2005 (USEPA, 2005b)). Young children represent the most sensitive receptor for potential lead exposures. USEPA's HHRAP guidance (USEPA, 2005a) recommends the IEUBK model to evaluate potential risks to human health associated with combustion facility emissions of lead. USEPA has also developed an adult lead biokinetic model for assessing adult exposures to lead in multiple environmental media (air, soil, and water) in an industrial/commercial setting (USEPA, 2003b); however, the IEUBK model generally yields more conservative results.

Several recent combustor facility risk studies have yielded extremely low incremental concentrations of lead in the modeled environmental media. Those concentrations are often so low that their media concentrations can not be input into the publicly available version of the IEUBK model (due to threshold format restrictions). As a conservative approach, the USEPA benchmark (USEPA, 1994) of less than 5 percent of children having blood lead concentrations exceeding 10 ug/dL has been used by the agency to calculate proportional concentrations that were used in the HHRA.

Based on the IEUBK model, the target soil lead concentration is 400 mg/kg. The USEPA incorporates a margin of safety by assuming that only 25% of the allowable threshold lead level would be assigned to the specific facility. That leads to a target soil concentration of 100 mg/kg. Similarly, USEPA has derived a target ambient air concentration for lead of 0.2 ug/m³. This value assumes that the target equals 25% of the quarterly average air concentration of 1.5 ug/m³ specified by the National Ambient Air Quality Standards (NAAQS) adjusted on an annual basis to 0.9 ug/m³.

3.4.4 Acute Toxicity Values

Potential risks due to short-term inhalation exposure (such as respiratory or irritant health effects), in addition to the more commonly evaluated chronic risks to human health discussed above, were evaluated in the HHRA. A screening level evaluation of short-term health effects was conducted by comparing predicted maximal short-term air concentrations against acute benchmarks. Acute benchmarks used in the HHRA are acute health risk values (acute HRVs) developed by MDH and acute Reference Exposure Levels (RELs) developed by CalEPA (2000). The acute HRV is defined as the concentration of a compound, at or below which the compound is unlikely to cause an adverse health effect to the general public when exposure occurs over a prescribed time. Acute HRVs are compared to one-hour averaged concentrations of compounds in air (<http://www.health.state.mn.us/divs/eh/air/rules.htm>). The acute REL values are also generally compared against a 1-hour concentration in air.

Table 3-21 lists the acute benchmarks for the COPCs. Acute benchmarks are available for five COPCs.

3.5 Risk Characterization

The Risk Characterization combines the results of the Exposure Assessment with the results of the Toxicity Assessment to derive an estimate of potential risk to human health. In this HHRA, the potential for occurrence of carcinogenic and noncarcinogenic health effects was evaluated for various receptors under the exposure scenarios identified. In addition, acute inhalation risks were also evaluated.

3.5.1 Approach for Risk Characterization

Carcinogenic Risk Characterization - The purpose of carcinogenic risk characterization is to estimate the upper-bound likelihood, over and above the background cancer rate, that a receptor will develop cancer in his or her lifetime as a result of exposure to a compound in environmental media at the site. This likelihood is a function of the dose of a compound (described in the Exposure Assessment) and the Cancer Slope Factor (CSF) (described in the Toxicity Assessment) for that compound. The Excess Lifetime Cancer Risk (ELCR) is the likelihood over and above the background cancer rate that an individual will contract cancer in his or her lifetime. The risk value is expressed as a probability (e.g., 10⁻⁶, or one in one million). For oral cancer risk estimates, a Lifetime Average Daily Dose (LADD) is calculated that averages a receptor's exposure dose over a lifetime. The relationship between the ELCR and the estimated LADD of a compound may be expressed as:

$$\text{ELCR} = 1 - e^{-(\text{CSF} \times \text{LADD})}$$

When the product of the CSF and the LADD is much greater than 1, the ELCR approaches 1 (i.e., 100 percent probability). When the product is less than 0.01 (one chance in 100), the equation can be closely approximated by:

$$\text{ELCR} = \text{LADD (mg/kg-day)} \times \text{CSF (mg/kg-day)}^{-1}$$

The product of the CSF and the LADD is unitless, and provides an upper-bound estimate of the potential carcinogenic risk associated with a receptor's exposure to that compound via that pathway.

For inhalation cancer risk estimates, an adjusted lifetime air concentration was calculated for each receptor accounting for the specific exposure frequency and duration for that receptor. This concentration is multiplied by the unit risk factor, as shown in the following equation:

$$\text{ELCR} = \text{Adjusted lifetime air concentration } (\mu\text{g}/\text{m}^3) \times \text{unit risk factor } (\mu\text{g}/\text{m}^3)^{-1}$$

The ELCR is compared to the cancer risk guideline defined by USEPA (2005a) and MPCA of 1×10^{-5} (1 in 100,000).

Non-carcinogenic Risk Characterization – For oral noncancer risk estimates, a Chronic Average Daily Dose (CADD) is calculated that averages a receptor's exposure dose over the exposure duration. The potential risk of adverse non-carcinogenic health effects is estimated for each receptor by comparing the CADD for each compound with the RfD for that compound. The resulting ratio, which is unitless, is known as the Hazard Quotient (HQ) for that compound. The HQ is calculated using the following equation:

$$\text{HQ} = \text{CADD (mg/kg – day)} / \text{RfD (mg/kg-day)}$$

For inhalation noncancer risk estimates, an adjusted chronic air concentration is estimated which represents the air concentration that the receptor could inhale averaged over the exposure duration, and accounts for the specific exposure frequency and duration of that receptor. This concentration is divided by the Reference Concentration (RfC), as shown in the following equation:

$$\text{HQ} = \text{Adjusted chronic air concentration } (\mu\text{g}/\text{m}^3) / \text{RfC } (\mu\text{g}/\text{m}^3)$$

The total Hazard Index (HI) for each receptor is calculated by summing the HQs for each pathway and compound within that pathway. The total HI is compared to the acceptable HI defined by USEPA and MPCA of 1.

When risk characterization results for all COPCs are below both the USEPA's acceptable cancer risk level and noncarcinogenic HI, no further analysis is presumed to be necessary. If the initial predictions exceed one or both benchmarks, there is not necessarily a significant risk of health effects. Rather, the results would indicate the need to look more carefully at site-specific conditions to refine the evaluation of potential health risk associated with exposure to facility related emissions.

Acute Risk Characterization - Potential acute (maximum 1-hour) risks to human health were evaluated only for the inhalation exposure pathway, as recommended in USEPA guidance (USEPA, 2005a). Acute risks to human health were evaluated for the maximum one hour air concentration for all COPCs. Acute hazard quotients were calculated using the following equation:

$$\text{HQ} = \frac{\text{AC } (\mu\text{g}/\text{m}^3)}{\text{acute benchmark } (\mu\text{g}/\text{m}^3)}$$

Where:

HQ = hazard quotient

AC = estimated 1-hour maximum air concentration

Acute benchmark = compound-specific acute benchmark or toxicity value.

3.5.2 Risk Characterization Results

The following sections discuss the chronic cancer and non-cancer results for the various receptors, and the acute risk results.

3.5.2.1 Chronic Cancer and Noncancer Risk Results

ELCR and noncarcinogenic HI were calculated for the various receptors assuming exposure to emissions based on Permit Limits and actual emissions.

Risk Estimates based on Permit Limits

The total ELCR and noncarcinogenic HI for each of the receptors are summarized in Tables 3-22 to 3-25. The risk calculation spreadsheets are in Appendix D.

Ballpark staff - As shown in Table 3-22, the total ELCR for the ballpark staff is 3×10^{-6} , which is less than USEPA and MPCA's cancer risk level of 1×10^{-5} . The compound and exposure pathway responsible for most of the risk is the ingestion of 2,3,7,8-TCDD TEQ in dust. It is noted that the oral CSF for 2,3,7,8-TCDD developed by MDH of 1.4×10^6 /mg/kg-day is 10-fold higher (i.e., 10-fold more conservative) than USEPA's CSF of 1.5×10^5 /mg/kg-day. If the USEPA CSF were used, then the ELCR would be 10-fold lower. However, even with the use of the more conservative MDH CSF for 2,3,7,8-TCDD the ELCR is less than acceptable levels.

As shown in Table 3-22, the total HI is 0.02, which is less than the acceptable HI defined by USEPA and MPCA of 1.

Ballplayers - As shown in Table 3-23, the total ELCR for the ballplayers is 2×10^{-6} , which is less than USEPA and MPCA's cancer risk level of 1×10^{-5} . Similar to the results for ballpark staff, ingestion of 2,3,7,8-TCDD in soil is the compound and exposure pathway responsible for most of the cancer risk. As shown in Table 3-23, the total HI is 0.02, which is less than the acceptable HI defined by USEPA and MPCA of 1.

Child season ticket holders - As shown in Table 3-24, the total ELCR for child season ticket holders is 3×10^{-6} , which is less than USEPA and MPCA's cancer risk level of 1×10^{-5} . Similar to the results for the other receptors, ingestion of 2,3,7,8-TCDD in dust is responsible for most of the cancer risk. The exposure pathways involving inhalation and food ingestion resulted in minimal risk. As shown in Table 3-24, the total HI is 0.07, which is less than the acceptable HI defined by USEPA and MPCA of 1.

Adult season ticket holders - As shown in Table 3-25, the total ELCR for adult season ticket holders is 1×10^{-6} , which is less than USEPA and MPCA's cancer risk level of 1×10^{-5} . Similar to the results for the other receptors, ingestion of 2,3,7,8-TCDD in dust is responsible for most of the cancer risk. The exposure pathways involving inhalation and food ingestion resulted in minimal risk. As shown in Table 3-25, the total HI is 0.009, which is less than the acceptable HI defined by USEPA and MPCA of 1.

Risk Estimates based on Actual Emissions

The total ELCR and noncarcinogenic HI for each receptor is summarized in Tables 3-26 to 3-29. The risk calculation spreadsheets are in Appendix D.

Ballpark staff - As shown in Table 3-26, the total ELCR for the ballpark staff is 4×10^{-7} , which is less than USEPA and MPCA's cancer risk level of 1×10^{-5} . The compound and exposure pathway responsible for most of the risk is the ingestion of 2,3,7,8-TCDD TEQ in dust, although the estimated 2,3,7,8-TCDD TEQ levels in dust are almost an order of magnitude lower than the levels

estimated based on Permit Limits. As shown in Table 3-26, the total HI is 0.001, which is less than the acceptable HI defined by USEPA and MPCA of 1.

Ballplayers - As shown in Table 3-27, the total ELCR for the ballplayers is 3×10^{-7} , which is less than USEPA and MPCA's cancer risk level of 1×10^{-5} . Similar to the results for ballpark staff, ingestion of 2,3,7,8-TCDD in soil is the compound and exposure pathway responsible for most of the cancer risk. As shown in Table 3-27, the total HI is 0.001, which is less than the acceptable HI defined by USEPA and MPCA of 1.

Child season ticket holders - As shown in Table 3-28, the total ELCR for child season ticket holders is 3×10^{-7} , which is less than USEPA and MPCA's cancer risk level of 1×10^{-5} . Similar to the results for the other receptors, ingestion of 2,3,7,8-TCDD in dust is responsible for most of the cancer risk. The exposure pathways involving inhalation and food ingestion resulted in minimal risk. As shown in Table 3-28, the total HI is 0.005, which is less than the acceptable HI defined by USEPA and MPCA of 1.

Adult season ticket holders - As shown in Table 3-29, the total ELCR for adult season ticket holders is 2×10^{-7} , which is less than USEPA and MPCA's cancer risk level of 1×10^{-5} . Similar to the results for the other receptors, ingestion of 2,3,7,8-TCDD in dust is responsible for most of the cancer risk. The exposure pathways involving inhalation and food ingestion resulted in minimal risk. As shown in Table 3-29, the total HI is 0.0008, which is less than the acceptable HI defined by USEPA and MPCA of 1.

3.5.2.2 Criteria Pollutants

The criteria pollutants evaluated in this HHRA (carbon monoxide, sulfur dioxide, nitrogen dioxide, lead, TSP, PM-10 and PM-2.5) were evaluated by adding the maximum modeled concentrations to the background concentrations as measured by MPCA over the past three years (2003-2005) and then comparing with Minnesota Ambient Air Quality Standards (MAAQS). Compliance with MAAQS indicates that there is no health risk associated with these pollutants. For lead an additional assessment has been made to ensure that any risks associated with incidental ingestion of deposited material are below levels of concern.

Permit Limits

Table 3-30 shows estimated air concentrations based on emissions at the Permit Limits and compares them against the MAAQS. All of the air concentrations are lower than the MAAQS.

Actual Emissions

Table 3-31 shows estimated air concentrations based on actual emissions and compares them against the MAAQS. All of the air concentrations are lower than the MAAQS.

3.5.2.3 Risk Estimates for Lead

A screening level risk assessment was conducted for lead by comparing estimated air and soil/dust concentrations against screening levels recommended in USEPA (2005a). As shown in Table 3-8b, the highest long-term air concentration for lead estimated at the stands is 1.8×10^{-4} ug/m³. This value is significantly lower than the threshold concentration of 0.2 ug/m³ recommended in USEPA (2005a). As shown in Tables 3-10b and 3-11b, the highest estimated lead concentrations in dust on ballpark surfaces is 3 mg/kg in dust, and the highest estimated lead concentrations in soil on the field is 6 mg/kg. These concentrations are significantly lower than the threshold concentration of 100 mg/kg in soil recommended by USEPA (2005a). As shown in Table 3-8b, the highest estimated food deposition of lead is 6.5×10^{-3} ug/day. This value is significantly lower than the average dietary lead intake for U.S. children, which ranges from 5.5 to 7 ug/day (USEPA, 1994).

These average dietary lead intake values are incorporated into the IEUBK model. These results indicate that lead emissions at either the Permit limits or measured amounts are below levels of concern for all of the receptors.

3.5.2.4 Acute Risk Results

The maximum 1-hour air concentrations anywhere in the ballpark were estimated using dispersion modeling.

Permit Limits

Table 3-32 shows the maximum 1-hour air concentrations based on emissions at the Permit Limits, acute inhalation toxicity values and the resulting HQs. Several of the compounds do not have acute benchmarks or toxicity values, therefore it was not possible to calculate an HQ for these compounds. The sum of the HQs is below the HI of 1. These results show that people continuously present at the location of maximum 1-hour air concentrations have estimated acute health risks that are below levels of concern. For nitrogen dioxide, both HERC-related air concentrations and background concentrations are available. These were compared separately against the acute inhalation toxicity value for nitrogen dioxide. Even when the background nitrogen dioxide air concentration was included, the resultant acute health risks are below levels of concern.

Actual Emissions

Table 3-33 shows the maximum 1-hour air concentrations based on actual emissions, acute inhalation toxicity values and the resulting HQs. The sum of the HQs is below the HI of 1. These results show that people continuously present at the location of maximum 1-hour air concentrations have estimated acute health risks that are below levels of concern.

3.5.2.5 Summary of Risk Results

For emissions based on both Permit Limits and measurements, the chronic carcinogenic and noncarcinogenic risk results for the various receptors are less than USEPA and MPCA's cancer risk level of 1×10^{-5} and HI of 1.

The criteria pollutants evaluated in this HHRA (carbon monoxide, sulfur dioxide, nitrogen dioxide, lead, TSP, PM-10 and PM-2.5) were evaluated by comparing estimated annual average and short-term air concentrations against MAAQS. All of the estimated air concentrations are lower than their respective MAAQS.

A comparison to screening concentrations of lead in air and soil based on the IEUBK model for lead (USEPA, 1994) indicated that lead emissions at either the Permit Limits or measurements are below levels of concern.

An acute risk evaluation was conducted for inhalation exposure to maximum 1-hour air concentrations emitted from the HERC facility. For emissions based on both Permit Limits and actual emissions, the air concentrations were all below acute benchmarks.

These results show that even if the emissions from HERC are at the Permit Limits, the potential health risks to people working at and visiting the ballpark are below levels of concern. The risk estimates are even lower if actual emissions data are used.

3.5.3 Uncertainty Analysis

There are many assumptions involved in risk assessment. In general, conservative assumptions are made throughout the risk assessment to account for uncertainties in the risk assessment

process. For the compounds assessed, when all of the assumptions are combined, it is much more likely that actual risks are overestimated rather than underestimated.

The assumptions that introduce the greatest amount of uncertainty in this risk assessment are discussed in this section. They are discussed in general terms, because for most of the assumptions there is not enough information to assign a numerical value that can be factored into the calculation of risk.

3.5.3.1 Hazard Identification

During the Hazard Identification step, compounds are selected for inclusion in the quantitative risk assessment from a list of all compounds known or expected to be emitted from municipal waste combustion. Uncertainty is introduced in three principal areas during this step: (1) selection of COPCs; (2) estimation of emission rates; and (3) air quality modeling.

Selection of COPCs

COPCs were selected based on compounds that have permit limits and stack testing results. In addition, a table provided by MPCA (MPCA, 2005) included emissions estimates for additional compounds. A small portion of the compounds is associated with municipal waste combustion, and the majority of the compounds are associated with natural gas usage. The additional compounds associated with municipal waste combustion were included as COPCs. Since natural gas usage at this facility is very limited, compounds associated with natural gas usage were not included as COPCs. It is likely that most of the significant compounds have been included as COPCs.

Emission Rates

For COPCs with permit limits, two sets of emissions were evaluated:

- MPCA Title V Air Emission Permit #05300400-002 limits
- 2005 air emission data obtained from Covanta Energy reflecting the permitted capacity of 365,000 tons per year (82.5% of the design capacity).

For the additional compounds listed by MPCA (MPCA, 2005) as being associated with municipal waste combustion (arsenic, chromium, nickel and PM_{2.5}), the emission rates listed by MPCA were used. MPCA calculated these emission rates using software developed by USEPA, called Regional Air Pollutant Inventory Development System (RAPIDS; <http://www.glc.org/air/rapids/rpdsover.html>). These compounds and the MPCA-estimated emission rates were included in the HHRA results for COPCs with permit limits (evaluated at both the permit limits and measured emissions).

Emissions were estimated for dioxins expressed as 2,3,7,8-TCDD toxic equivalents using congener-specific emissions measurement data supplied by Covanta. The fraction of individual congeners considered to be toxic was accounted for when developing emissions based on permit limits and measurement data. The approach of estimating emissions on both the permit limits and measurement data helps to place an upper bound on potential health risks that could occur assuming that the facility emissions are constantly at the permit limits. In reality, actual emissions are likely to be much less than the permit limits.

Air Quality and Deposition Modeling

Air dispersion and deposition modeling was conducted to estimate air concentrations and deposition rates to support the HHRA. The modeling was conducted with the recently promulgated AERMOD model (Version 04300) in accordance with USEPA Guideline on Air Quality Models. AERMOD is a state-of-the-art dispersion model that incorporates modeling improvements over the

Industrial Source Complex (ISC) model and, similar to ISC, is capable of computing particle and vapor deposition in addition to air concentrations.

Dispersion model evaluation studies have shown that models such as AERMOD typically estimate air concentrations to within about a factor of 2 compared to observations. While the uncertainty of particle and vapor deposition rates computed by AERMOD has not yet been established, it is likely to be larger than that for air concentrations because deposition in the model is highly parameterized and difficult to verify. Where possible, upper limit estimates were used which is consistent with guidance in USEPA HHRAP.

3.5.3.2 Exposure Assessment

The exposure assessment involves the selection of receptors, estimation of exposure point concentrations, and calculation of exposure doses. Exposure point concentrations are the estimated concentrations of compounds to which humans may be exposed. Once the concentrations in an environmental medium such as soil, water, or air have been predicted, the calculation of human exposure and dose involves making additional assumptions. The major sources of uncertainty associated with these assumptions are discussed below.

Selection of receptors

While there are various types of receptors who could be exposed at a ballpark, the ones that are likely to receive the highest exposures were considered in this HHRA. Three sets of receptors were considered based on their exposure potential; these are (1) ballpark staff, (2) ballplayers, and (3) season ticket holders.

The exposure assumptions used to evaluate these receptors were based on conservative default values developed by USEPA, and conservative site-specific values based on activities at the ballpark. It was assumed that ballplayers and season ticket holders would attend the maximum number of games per season.

Estimation of air concentrations

The maximum concentrations were used in the risk calculations. Because the risk calculations used the highest concentrations for each type of receptor the risk is overestimated for the average player and fan. For receptors on the playing field the difference between the highest values and average values is about 25% but for the stands the difference more than a factor of 2.

Estimation of soil and dust concentrations

It has been assumed that all selected organic and inorganic compounds once deposited in the soil remain in the top 2-cm layer and do not degrade. This assumption ignores processes that, in reality, result in the loss of compounds from soil. For deposition on the playing field it has been assumed that all of the water runoff from rain and cleaning the stands is used for irrigation. For dust depositing on the stands wet deposition of ambient particulate is not included. These factors will overestimate the exposure point concentration.

Estimation of Compound Intake from Food

Deposition of particles onto food was evaluated using air dispersion modeling and conservative assumptions regarding the mass of compound that could deposit onto food surfaces. In order to evaluate this pathway, it was necessary to assume that a COPC could deposit onto a surface that is covered with food (such as hotdogs, fries, drinks, etc.). Rather than evaluating individual food items, it was assumed that COPCs could deposit onto the surface of a standard size cafeteria tray that is covered with food. Deposition onto the tray could occur over the 4 hours that the season

ticket holder is assumed to stay at the ballpark. It is likely that these conservative assumptions account for receptors likely to receive the highest exposure.

3.5.3.3 Toxicity Assessment

Dose-response values are usually based on limited toxicological data. For this reason, a margin of safety is built into estimates of both carcinogenic and noncarcinogenic risk. The two major areas of uncertainty introduced in the dose-response assessment are: (1) animal to human extrapolation; and (2) high to low dose extrapolation. These are discussed in the following subsections.

Animal to Human Extrapolation

Human dose-response values are often extrapolated, or estimated, using the results of animal studies. Extrapolation from animals to humans introduces a great deal of uncertainty in the risk assessment because in most instances, it is not known how differently a human may react to the compound compared to the animal species used to test the compound. The procedures used to extrapolate from animals to humans involve conservative assumptions and incorporate several uncertainty factors that generally overestimate the adverse effects associated with a specific dose. Thus, it is more likely that the potential for adverse effects is overestimated than underestimated.

High to Low Dose Extrapolation

Predicting potential health effects from combustor emissions requires the use of models to extrapolate the observed health effects from the high doses used in laboratory studies to the anticipated human health effects from low doses experienced in the environment.

The models contain conservative assumptions to account for the large degree of uncertainty associated with this extrapolation (especially for potential carcinogens) and therefore, tend to be more likely to overestimate than underestimate the risks.

3.5.3.4 Risk Characterization

The risk of adverse human health effects depends on estimated levels of exposure and dose-response relationships. Two important additional sources of uncertainty are introduced in this phase of the risk assessment: (1) the evaluation of potential exposure to more than one compound; and (2) the combination of upper-bound exposure assumptions.

Risk from Multiple Compounds

Once exposure to and risk from each of the selected compounds is calculated, the total risk posed by cumulative impacts of facilities in the study area is estimated by combining the health risk contributed by each compound. It is assumed that carcinogenic effects of different compounds may be added together. Noncarcinogenic effects are often summed, as in this report, although this is less appropriate because different compounds may have different health endpoints (e.g., neurotoxicity, liver effects, respiratory irritation). A target endpoint-specific analysis would have been conducted if the combined HI was found to be greater than 1. It is also possible that a mixture of compounds could have antagonistic or synergistic effects. There are few scientific studies that study mixtures. The amount of uncertainty associated with summing the effects varies case-by-case.

Combination of Several Upper-Bound Assumptions

Generally, the goal of risk assessment is to estimate an upper-bound, but reasonable, prediction of potential risk to human health. Most of the assumptions about exposure and toxicity used in this assessment are representative of statistical upper-bounds or even maxima for each of the parameters. The result of combining several such upper-bound assumptions is that the final estimate of potential exposure or potential risk is very conservative.

TABLE 3-1 WHO DIOXIN/FURAN 2,3,7,8-TCDD TOXIC EQUIVALENT FACTORS

Dioxin/Furan Congener	TEF	2004 Unit 1		2005 Unit 1		2006 Unit 1		2004 Unit 2		2005 Unit 2		2006 Unit 2	
		Mass (pg)	TEQ (pg)										
2,3,7,8-TCDD	1	5.69	5.69	3.48	3.48	8.8	8.8	1.26	1.26	2.7	2.7	3.33	3.33
1,2,3,7,8-PeCDD	1	35.9	35.9	30.1	30.1	26.7	26.7	6.84	6.84	16.2	16.2	5.27	5.27
1,2,3,4,7,8-HxCDD	0.1	70.2	7.02	63.5	6.35	22.9	2.29	11	1.1	21.1	2.11	7.9	0.79
1,2,3,6,7,8-HxCDD	0.1	312	31.2	394	39.4	50.5	5.05	35.8	3.58	115	11.5	18.1	1.81
1,2,3,7,8,9-HxCDD	0.1	156	15.6	177	17.7	32.5	3.25	19.5	1.95	53	5.3	11.1	1.11
1,2,3,4,6,7,8-HpCDD	0.01	1900	19	3710	37.1	339	3.39	222	2.22	764	7.64	152	1.52
OCDD	0.0003	5160	1.548	6720	2.016	591	0.1773	220	0.066	868	0.2604	286	0.0858
2,3,7,8-TCDF	0.1	25.4	2.54	16.5	1.65	42.1	4.21	4.97	0.497	13.3	1.33	6.8	0.68
1,2,3,7,8-PeCDF	0.03	48.3	1.449	31.4	0.942	60.6	1.818	10	0.3	22.8	0.684	7.64	0.2292
2,3,4,7,8-PeCDF	0.3	121	36.3	62.6	18.78	84	25.2	19.8	5.94	50.8	15.24	17.7	5.31
1,2,3,4,7,8-HxCDF	0.1	98.9	9.89	57.5	5.75	73.7	7.37	14.5	1.45	39.3	3.93	18.4	1.84
1,2,3,6,7,8-HxCDF	0.1	136	13.6	74.8	7.48	83.9	8.39	15.5	1.55	50.1	5.01	22.1	2.21
2,3,4,6,7,8-HxCDF	0.1	323	32.3	151	15.1	75.4	7.54	27.4	2.74	106	10.6	22.8	2.28
1,2,3,7,8,9-HxCDF	0.1	63.1	6.31	43.7	4.37	22.2	2.22	6.44	0.644	21.9	2.19	6.54	0.654
1,2,3,4,6,7,8-HpCDF	0.01	502	5.02	248	2.48	176	1.76	37.5	0.375	146	1.46	74.7	0.747
1,2,3,4,7,8,9-HpCDF	0.01	176	1.76	119	1.19	29.3	0.293	12.2	0.122	47.7	0.477	8.24	0.0824
OCDF	0.0003	441	0.1323	268	0.0804	66.2	0.01986	28.2	0.00846	84.8	0.02544	32.3	0.00969
Total		9574.49	225.26	12170.58	193.97	1784.80	108.48	692.91	30.64	2422.70	86.66	700.92	27.96
TEQ/Total		0.024		0.016		0.061		0.044		0.036		0.040	
AverageTEQ/Total		0.037											

TABLE 3-2 ACTUAL LONG TERM EMISSION RATES

Constituent	Long-Term Emission Rate (g/sec)			Data Source
	Unit 1	Unit 2	Total	
Arsenic	1.17E-04	1.13E-04	2.29E-04	MPCA
Cadmium	3.35E-05	1.91E-05	5.26E-05	Testing
Chromium	2.40E-04	2.31E-04	4.71E-04	MPCA
Lead	3.84E-04	1.02E-04	4.86E-04	Testing
Mercury	1.97E-04	1.13E-04	3.10E-04	Testing
Nickel	2.10E-04	2.02E-04	4.12E-04	MPCA
Total 2,3,7,8-TCDD-TEQ	1.19E-08	8.33E-10	1.28E-08	Testing
Hydrochloric acid	7.07E-01	4.90E-01	1.20E+00	Testing
Carbon monoxide	9.19E-01	9.62E-01	1.88E+00	Inventory
Sulfur dioxide	2.13E-01	1.85E-01	3.98E-01	Inventory
Sulfuric acid ⁽¹⁾	6.39E-03	5.54E-03	1.19E-02	Inventory
Nitrogen dioxide ⁽²⁾	7.10E+00	7.02E+00	1.41E+01	Inventory
TSP	4.34E-01	2.25E-01	6.59E-01	Inventory
PM ₁₀ ⁽³⁾	0.00E+00	0.00E+00	0.00E+00	Inventory
PM _{2.5}	2.68E-01	8.07E-02	3.49E-01	Inventory

(1) Based on 3% of sulfur dioxide emissions

(2) NOx emissions from firing natural gas at 170,000 ft³/hr is 2.14 g/sec

(3) TSP fraction determined from particle size distribution data 0.622

TABLE 3-3 ACTUAL SHORT-TERM EMISSION RATES

Constituent	Maximum Short-Term Emission Rate (g/sec)			Data Source
	Unit 1	Unit 2	Total	
Arsenic	1.37E-04	1.32E-04	2.68E-04	MPCA
Cadmium	1.24E-04	7.06E-05	1.95E-04	Testing
Chromium	2.80E-04	2.71E-04	5.51E-04	MPCA
Lead	1.43E-03	2.29E-04	1.66E-03	Testing
Mercury	8.27E-04	3.44E-04	1.17E-03	Testing
Nickel	2.45E-04	2.37E-04	4.82E-04	MPCA
Total 2,3,7,8-TCDD-TEQ	9.62E-08	2.62E-09	9.89E-08	Testing
Hydrochloric acid	8.88E-01	8.93E-01	1.78E+00	Testing
Carbon monoxide	1.11E+00	1.07E+00	2.17E+00	Inventory
Sulfur dioxide	2.25E-01	2.03E-01	4.28E-01	Inventory
Sulfuric acid ⁽¹⁾	6.75E-03	6.08E-03	1.28E-02	Inventory
Nitrogen dioxide ⁽²⁾	7.24E+00	7.33E+00	1.46E+01	Inventory
TSP	6.53E-01	4.22E-01	1.07E+00	Inventory
PM ₁₀ ⁽³⁾	4.06E-01	2.63E-01	6.69E-01	Inventory
PM _{2.5}	3.13E-01	9.45E-02	4.08E-01	Inventory

(1) Based on 3% of sulfur dioxide emissions

(2) NOx emissions from firing natural gas at 170,000 ft³/hr is 2.14 g/sec

(3) TSP fraction determined from particle size distribution data 0.622

TABLE 3-4 TITLE V PERMIT LIMITS

Constituent	Permit Limit-Based Emission Rate, per Unit (as listed in permit)			
	Long-Term		Maximum Short-Term	
Arsenic	NA		NA	
Cadmium	4.00E+01 ug/dscm	@ 7% O2	NA	
Chromium	NA		NA	
Lead	4.40E+02 ug/dscm	@ 7% O2	NA	
Mercury	6.00E+01 ug/dscm	@ 7% O2	8.00E+01 ug/dscm	@ 7% O2
Nickel	NA		NA	
Total Dioxin/Furan	3.00E+01 ng/dscm	@ 7% O2	NA	
Hydrochloric acid	2.90E+01 ppm	@ 7% O2	NA	
	43233 ug/dscm	@ 7% O2		
Carbon monoxide	1.00E+02 (ppm)	4 hr block average @ 7% O2	1.00E+02 (ppm)	4 hr block average @ 7% O2
	114519 ug/dscm	@ 7% O2	114519 ug/dscm	@ 7% O2
Sulfur dioxide	1.14E+01 (lbs/hr)	365 day rolling average	7.80E+01 (ppm)	3 hr block average @ 7% O2
			204172 ug/dscm	@ 7% O2
Nitrogen oxides (as NO2)	2.05E+02 (ppm)	24 hr block average @ 7% O2	2.05E+02 (ppm)	24 hr block average @ 7% O2
	385685 ug/dscm	@ 7% O2	385685 ug/dscm	@ 7% O2
TSP	9.80E+01 tons/yr	12-month Rolling Sum	2.00E-02 (grains/dscf)	@ 7% O2
PM2.5	NA		NA	

TABLE 3-5 EMISSION RATES BASED ON PERMIT LIMITS

Permit Limit-Based Emission Rate - <u>Total, Both Units (g/sec)</u>			Data
Constituent	Long-Term	Maximum Short-Term	Source
Arsenic ⁽¹⁾	2.29E-04	2.68E-04	MPCA
Cadmium ⁽¹⁾	4.40E-03	5.15E-03	Title V
Chromium ⁽¹⁾	4.71E-04	5.51E-04	MPCA
Lead ⁽¹⁾	4.84E-02	5.67E-02	Title V
Mercury	6.60E-03	8.80E-03	Title V
Nickel ⁽¹⁾	4.12E-04	4.82E-04	MPCA
Total 2,3,7,8-TCDD-TEQ ^(1,2)	1.21E-07	1.42E-07	Title V
Hydrochloric acid ⁽¹⁾	4.76E+00	5.57E+00	Title V
Carbon monoxide	1.26E+01	1.26E+01	Title V
Sulfur dioxide	2.87E+00	2.25E+01	Title V
Sulfuric acid ⁽⁴⁾	8.62E-02	6.74E-01	MPCA
Nitrogen dioxide	4.24E+01	4.24E+01	Title V
TSP	5.04E+00	5.04E+00	Title V
PM ₁₀ ⁽³⁾	3.13E+00	3.13E+00	Title V
PM _{2.5} ⁽¹⁾	3.49E-01	4.08E-01	MPCA

- (1) No short-term permit limit, used long-term limit increased by: 17%
- (2) Based on ratio of TEQ TCDD emissions to actual dioxin/furan emissions of stack tes 0.037
- (3) Fraction of TSP determined from particle size distribution data 0.622
- (4) Based on 3% of sulfur dioxide emissions

TABLE 3-6 PARTICLE SIZE DISTRIBUTION

Particle Size Diameter (μm)	Mass Fraction	Surface Area Fraction ⁽¹⁾
0.3	5.26E-01	9.57E-01
0.59	1.00E-02	9.25E-03
0.91	5.00E-03	3.00E-03
1.77	2.00E-02	6.17E-03
2.94	3.60E-02	6.68E-03
4.35	1.50E-02	1.88E-03
6.38	1.00E-02	8.55E-04
13.56	3.78E-01	1.52E-02

(1) Assumes particle density of 1 g/cm^3 . Calculation of surface area fraction based on the USEPA recommended approach in Chapter 3, Section 3.2.3 of the HHRAP.

TABLE 3-7A AIRBORNE CONCENTRATIONS FOR PARK USERS, ASSUMING ACTUAL EMISSIONS

Compound	Air Concentrations for Players (mg/m ³)				Air Concentrations for Concession Workers & Fans (mg/m ³)			
	Highest 1-Hour Max	Average 1-Hour Max	Highest Long-Term	Average Long-Term	Highest 1-Hour Max	Average 1-Hour Max	Highest Long-Term	Average Long-Term
Arsenic	2.88E-07	2.62E-07	6.65E-10	4.48E-10	3.58E-07	2.74E-07	8.53E-10	3.80E-10
Cadmium	2.09E-07	1.90E-07	1.53E-10	1.03E-10	2.60E-07	1.99E-07	1.96E-10	8.71E-11
Chromium	5.92E-07	5.37E-07	1.37E-09	9.20E-10	7.35E-07	5.63E-07	1.75E-09	7.79E-10
Hydrogen Chloride	1.91E-03	1.74E-03	3.47E-06	2.34E-06	2.37E-03	1.82E-03	4.45E-06	1.98E-06
Lead	1.78E-06	1.62E-06	1.41E-09	9.49E-10	2.21E-06	1.69E-06	1.81E-09	8.04E-10
Mercury	1.26E-06	1.14E-06	8.99E-10	6.06E-10	1.56E-06	1.20E-06	1.15E-09	5.13E-10
Nickel	5.18E-07	4.70E-07	1.20E-09	8.05E-10	6.43E-07	4.92E-07	1.53E-09	6.82E-10
2,3,7,8-TCDD TEQ	1.06E-10	9.64E-11	3.71E-14	2.50E-14	1.32E-10	1.01E-10	4.75E-14	2.11E-14
Nitrogen Dioxide	1.56E-02	1.42E-02	4.10E-05	2.76E-05	1.94E-02	1.49E-02	5.26E-05	2.34E-05
Sulfuric Acid	1.38E-05	1.25E-05	3.46E-08	2.33E-08	1.71E-05	1.31E-05	4.44E-08	1.97E-08

TABLE 3-7B AIRBORNE CONCENTRATIONS FOR PARK USERS, ASSUMING PERMIT LIMITS

Compound	Air Concentrations for Players (mg/m ³)				Air Concentrations for Concession Workers & Fans (mg/m ³)			
	Highest 1-Hour Max	Average 1-Hour Max	Highest Long- Term	Average Long-Term	Highest 1-Hour Max	Average 1-Hour Max	Highest Long- Term	Average Long-Term
Arsenic	2.88E-07	2.62E-07	6.65E-10	4.48E-10	3.58E-07	2.74E-07	8.53E-10	3.80E-10
Cadmium	5.53E-06	5.02E-06	1.28E-08	8.60E-09	6.87E-06	5.26E-06	1.64E-08	7.29E-09
Chromium	5.92E-07	5.37E-07	1.37E-09	9.20E-10	7.35E-07	5.63E-07	1.75E-09	7.79E-10
Hydrogen Chloride	5.98E-03	5.43E-03	1.38E-05	9.29E-06	7.42E-03	5.69E-03	1.77E-05	7.87E-06
Lead	6.09E-05	5.53E-05	1.40E-07	9.46E-08	7.55E-05	5.79E-05	1.80E-07	8.01E-08
Mercury	9.46E-06	8.59E-06	1.91E-08	1.29E-08	1.17E-05	8.99E-06	2.46E-08	1.09E-08
Nickel	5.18E-07	4.70E-07	1.20E-09	8.05E-10	6.43E-07	4.92E-07	1.53E-09	6.82E-10
2,3,7,8-TCDD TEQ	1.52E-10	1.38E-10	3.51E-13	2.37E-13	1.89E-10	1.45E-10	4.51E-13	2.00E-13
Nitrogen Dioxide	4.56E-02	4.14E-02	1.23E-04	8.29E-05	5.66E-02	4.33E-02	1.58E-04	7.02E-05
Sulfuric Acid	7.24E-04	6.57E-04	2.50E-07	1.68E-07	8.99E-04	6.88E-04	3.21E-07	1.43E-07

TABLE 3-8A HIGHEST AND AVERAGE FOOD DEPOSITION AMONG FANS IN THE BALLPARK STANDS, ASSUMING ACTUAL EMISSIONS

Compound	Grams of Compound Ingested from Food (g)	
	Highest	Average
Arsenic	6.53E-11	3.08E-11
Cadmium	2.39E-10	1.12E-10
Chromium	2.14E-09	9.98E-10
Lead	1.38E-10	6.52E-11
Mercury	8.82E-11	4.16E-11
Nickel	1.87E-09	8.74E-10
2,3,7,8-TCDD TEQ	3.64E-15	1.71E-15

TABLE 3-8B HIGHEST AND AVERAGE FOOD DEPOSITION AMONG FANS IN THE BALLPARK STANDS, ASSUMING PERMIT LIMITS

Compound	Grams of Compound Ingested from Food (g)	
	Highest	Average
Arsenic	6.53E-11	3.08E-11
Cadmium	2.00E-08	9.33E-09
Chromium	2.14E-09	9.98E-10
Lead	1.38E-08	6.50E-09
Mercury	1.88E-09	8.86E-10
Nickel	1.87E-09	8.74E-10
2,3,7,8-TCDD TEQ	3.45E-14	1.63E-14

TABLE 3-9 CALCULATION OF BACKGROUND DEPOSITION

Particulate Size	Mean Conc 2003-2005* (µg/m³)
PM _{2.5}	9.787
PM _{2.5} -PM ₁₀	15.313
PM ₁₀ --TSP	16.033
Total	41.133

* Annual concentrations for TSP and PM_{2.5} from monitor located at 2727 10TH ST, Minneapolis.
 Annual concentrations for PM₁₀ from monitor located at 309 2ND AVE, Minneapolis.
 Data downloaded from EPA AirData <http://www.epa.gov/air/data/>.

Diameter Range (µm)		Geometric Mean Diameter (µm)	Deosition Velocity* (cm/sec)
0.5	2.5	1.1	0.9
2.5	10	5.0	5.8
10	30	17.3	7.7

*Sehmel (1984), Figure 12.7

Particulate Size	April - October 1986-1990, 1070-day deposition (g/m²/1070 days)
PM _{2.5}	8.143
PM _{2.5} -PM ₁₀	82.110
PM ₁₀ --TSP	114.133
Total	204.386

TABLE 3-10A HIGHEST AND AVERAGE COPC CONCENTRATION IN DEPOSITION ON BALLPARK SURFACES, ASSUMING ACTUAL EMISSIONS

Compound	Concentration in Dust (mg/kg-dust)	
	Highest	Average
Arsenic	1.44E-02	5.96E-03
Cadmium	6.36E-02	2.69E-02
Chromium	5.69E-01	2.41E-01
Lead	3.05E-02	1.26E-02
Mercury	1.95E-02	8.06E-03
Nickel	4.98E-01	2.11E-01
2,3,7,8-TCDD TEQ	8.02E-07	3.32E-07

TABLE 3-10B HIGHEST AND AVERAGE COPC CONCENTRATION IN DEPOSITION ON BALLPARK SURFACES, ASSUMING PERMIT LIMITS

Compound	Concentration in Dust (mg/kg-dust)	
	Highest	Average
Arsenic	1.44E-02	5.96E-03
Cadmium	5.32E+00	2.25E+00
Chromium	5.69E-01	2.41E-01
Lead	3.04E+00	1.26E+00
Mercury	4.14E-01	1.72E-01
Nickel	4.98E-01	2.11E-01
2,3,7,8-TCDD TEQ	7.60E-06	3.15E-06

**TABLE 3-11A HIGHEST AND AVERAGE 10-YEAR SOIL CONCENTRATION ON THE FIELD,
ASSUMING ACTUAL EMISSIONS**

Compound	Concentration in Soil (mg/kg-soil)	
	Highest	Average
Arsenic	3.00E-02	2.42E-02
Cadmium	1.38E-01	1.13E-01
Chromium	1.23E+00	1.01E+00
Lead	6.34E-02	5.13E-02
Mercury	4.05E-02	3.27E-02
Nickel	1.08E+00	8.81E-01
2,3,7,8-TCDD TEQ	1.67E-06	1.35E-06

**TABLE 3-11B HIGHEST AND AVERAGE 10-YEAR SOIL CONCENTRATION ON THE FIELD,
ASSUMING PERMIT LIMITS**

Compound	Concentration in Soil (mg/kg-soil)	
	Highest	Average
Arsenic	3.00E-02	2.42E-02
Cadmium	1.15E+01	9.42E+00
Chromium	1.23E+00	1.01E+00
Lead	6.32E+00	5.11E+00
Mercury	8.62E-01	6.97E-01
Nickel	1.08E+00	8.81E-01
2,3,7,8-TCDD TEQ	1.58E-05	1.28E-05

TABLE 3-12 SUMMARY OF POTENTIAL EXPOSURE ASSUMPTIONS

Parameter	Ballpark Staff	References	Players	References	Season ticket holders			
					Child (1-6)	References	Adult	References
Parameters Used in the Food Ingestion Pathway								
Exposure Frequency (days/year)	NA		NA		92	(a)	92	(a)
Exposure Duration (yr)	NA		NA		6	(b)	24	(b)
Body Weight (kg)	NA		NA		15	(c)	70	(c)
Parameters Used in the Soil Ingestion Pathway								
Exposure Frequency (days/year)	225	(b)	92	(a)	92	(a)	92	(a)
Exposure Duration (yr)	25	(b)	20	(d)	6	(b)	24	(b)
Soil Ingestion Rate (mg/day)	100	(b,e)	100	(b,e)	200	(b)	100	(b)
Body Weight (kg)	70	(c)	70	(c)	15	(b)	70	(c)
Parameters Used in the Inhalation Pathway								
Exposure Duration (years)	25	(b)	20	(d)	6	(b)	24	(b)

Notes:

NA - Not Applicable.

(a) Best professional judgement. Assumed exposure during the season April through September.

(b) USEPA, 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. OSWER 9355.4-24, December 2002.

(c) USEPA, 1989. Risk Assessment Guidance for Superfund, Volume 1. Human Health Evaluation Manual (Part A). Office of Emergency and Remedial Response.

(d) Best professional judgement. Assumed that a ballplayer is with one team for 20 years.

(e) Assumed similar soil ingestion as an outdoor worker.

TABLE 3-13 EXPOSURE POINT CONCENTRATIONS - PERMIT LIMITS - BALLPARK STAFF

Compound	Soil/Dust (mg/kg)	Air (ug/m3)
Arsenic	1.44E-02	8.53E-07
Cadmium	5.32E+00	1.64E-05
Chromium (VI), particulates	5.69E-01	1.75E-06
Hydrogen Chloride (as Cl)	NCOPC	1.77E-02
Mercury	4.14E-01	2.46E-05
Nickel	4.98E-01	1.53E-06
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	7.60E-06	4.51E-10
Sulfuric Acid	NCOPC	3.21E-04
<p>Notes:</p> <p>2,3,7,8-TCDD is the toxic equivalent concentration of all dioxin/furan congeners. Toxic equivalents were calculated following WHO, 2006.</p> <p>NCOPC - Not a Compound of Potential Concern.</p> <p>TEQ - Toxic Equivalents.</p>		

TABLE 3-14 EXPOSURE POINT CONCENTRATIONS - PERMIT LIMITS – BALLPLAYERS

Compound	Soil/Dust (mg/kg)	Air (ug/m3)
Arsenic	3.00E-04	6.65E-07
Cadmium	1.15E-01	1.28E-05
Chromium (VI), particulates	1.23E-02	1.37E-06
Hydrogen Chloride (as Cl)	NCOPC	1.38E-02
Mercury	8.62E-03	1.91E-05
Nickel	1.08E-02	1.20E-06
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	1.58E-07	3.51E-10
Sulfuric Acid	NCOPC	2.50E-04
<p>Notes:</p> <p>2,3,7,8-TCDD is the toxic equivalent concentration of all dioxin/furan congeners. Toxic equivalents were calculated following WHO, 2006.</p> <p>NCOPC - Not a Compound of Potential Concern.</p> <p>TEQ - Toxic Equivalents.</p>		

TABLE 3-15 EXPOSURE POINT CONCENTRATIONS - PERMIT LIMITS - SEASON TICKET HOLDERS

Compound	Soil/Dust (mg/kg)	Air (ug/m3)	Food Ingestion (mg/day)
Arsenic	1.44E-02	8.53E-07	6.53E-08
Cadmium	5.32E+00	1.64E-05	2.00E-05
Chromium (VI), particulates	5.69E-01	1.75E-06	2.14E-06
Hydrogen Chloride (as Cl)	NCOPC	1.77E-02	NCOPC
Mercury	4.14E-01	2.46E-05	1.88E-06
Nickel	4.98E-01	1.53E-06	1.87E-06
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	7.60E-06	4.51E-10	3.45E-11
Sulfuric Acid	NCOPC	3.21E-04	NCOPC

Notes:
 2,3,7,8-TCDD is the toxic equivalent concentration of all dioxin/furan congeners. Toxic equivalents were calculated following WHO, 2006.
 NCOPC - Not a Compound of Potential Concern.
 TEQ - Toxic Equivalents.

TABLE 3-16 EXPOSURE POINT CONCENTRATIONS - ACTUAL EMISSIONS - BALLPARK STAFF

Compound	Soil/Dust (mg/kg)	Air (ug/m3)
Arsenic	1.44E-02	8.53E-07
Cadmium	6.36E-02	1.96E-07
Chromium (VI), particulates	5.69E-01	1.75E-06
Hydrogen Chloride (as Cl)	NCOPC	4.45E-03
Mercury	1.95E-02	1.15E-06
Nickel	4.98E-01	1.53E-06
2,3,7,8-Tetrachlorodibenzodioxin TEQ	8.02E-07	4.75E-11
Sulfuric Acid	NCOPC	4.44E-05
<p>Notes:</p> <p>2,3,7,8-TCDD is the toxic equivalent concentration of all dioxin/furan congeners. Toxic equivalents were calculated following WHO, 2006.</p> <p>NCOPC - Not a Compound of Potential Concern.</p> <p>TEQ - Toxic Equivalents.</p>		

**TABLE 3-17 EXPOSURE POINT CONCENTRATIONS – ACTUAL EMISSIONS –
BALLPLAYERS**

Compound	Soil/Dust (mg/kg)	Air (ug/m3)
Arsenic	3.00E-04	6.65E-07
Cadmium	1.38E-03	1.53E-07
Chromium (VI), particulates	1.23E-02	1.37E-06
Hydrogen Chloride (as Cl)	NCOPC	3.47E-03
Mercury	4.05E-04	8.99E-07
Nickel	1.08E-02	1.20E-06
2,3,7,8-Tetrachlorodibenzodioxin TEQ	1.67E-08	3.71E-11
Sulfuric Acid	NCOPC	3.46E-05
<p>Notes: 2,3,7,8-TCDD is the toxic equivalent concentration of all dioxin/furan congeners. Toxic equivalents were calculated following WHO, 2006. NCOPC - Not a Compound of Potential Concern. TEQ - Toxic Equivalents.</p>		

TABLE 3-18 EXPOSURE POINT CONCENTRATIONS - ACTUAL EMISSIONS - SEASON TICKET HOLDER

Compound	Soil/Dust (mg/kg)	Air (ug/m3)	Food Ingestion (mg/day)
Arsenic	1.44E-02	8.53E-07	6.53E-08
Cadmium	6.36E-02	1.96E-07	2.39E-07
Chromium (VI), particulates	5.69E-01	1.75E-06	2.14E-06
Hydrogen Chloride (as Cl)	NCOPC	4.45E-03	NCOPC
Mercury	1.95E-02	1.15E-06	8.82E-08
Nickel	4.98E-01	1.53E-06	1.87E-06
2,3,7,8-Tetrachlorodibenzodioxin TEQ	8.02E-07	4.75E-11	3.64E-12
Sulfuric Acid	NCOPC	4.44E-05	NCOPC

Notes:
 2,3,7,8-TCDD is the toxic equivalent concentration of all dioxin/furan congeners. Toxic equivalents were calculated following WHO, 2006.
 NCOPC - Not a Compound of Potential Concern.
 TEQ - Toxic Equivalents.

TABLE 3-19 CHRONIC INHALATION TOXICITY VALUES HUMAN HEALTH RISK ASSESSMENT

Constituent	CAS	HAP?	VOC?	Cancer Assessment			Chronic Noncancer Assessment		
				Tox Value Source	Unit Risk (ug/m3)-1	Surrogate Name	Tox Value Source	Reference Conc. (ug/m3)	Toxic Endpoint
Arsenic	7440-38-2	YES	NO	HRV	4.30E-03		CAL EPA	3.00E-02	Development; cardiovascular system; nervous system
Cadmium	7440-43-9	YES	NO	HRV	1.80E-03		CAL EPA	2.00E-02	Kidney; respiratory system
Chromium (VI), particulates	18540-29-9	YES	NO	HRV	1.20E-02		IRIS	1.00E-01	Lower respiratory system
Hydrogen Chloride (as Cl)	7647-01-0	YES	NO	NA	NA		HRV	2.00E+01	Upper respiratory system
Mercury	7439-97-6	YES	NO	NA	NA		IRIS	3.00E-01	Neurotoxicity
Nickel	7440-02-0	YES	NO	IRIS	2.40E-04	Nickel refinery dust	CAL EPA	5.00E-02	Respiratory system; hematopoietic system
2,3,7,8-Tetrachlorodibenzodioxin TEQ	1746-01-6	YES	NO	MDH	4.00E+02		CAL EPA	4.00E-05	Alimentary system (liver); reproductive system
Sulfuric Acid	7664-93-9	NA	NA	NA	NA		CAL EPA	1.00E+00	Respiratory system

Notes:
 CalEPA - California Office of Environmental Health Hazard Assessment.
 CAS - Chemical Abstracts Service.
 HAP - Hazardous Air Pollutant.
 HRV - Minnesota Department of Health Risk Value (<http://www.health.state.mn.us/divs/eh/air/rules.htm>).
 IRIS - EPA Integrated Risk Information System.
 MDH - Minnesota Department of Health value (<http://www.health.state.mn.us/divs/eh/risk/guidance/dioxinmemo2.html>).
 NA - Not Available.
 TEQ - Toxic Equivalents.
 VOC - Volatile Organic Compound.
 1. Individual dioxan/furan congeners were expressed as toxic equivalents of 2,3,7,8-TCDD using the World Health Organizations 2006 Toxic Equivalency Factors (TEF's).

TABLE 3-20 CHRONIC ORAL TOXICITY VALUES HUMAN HEALTH RISK ASSESSMENT

Constituent	CAS	RfD (mg/kg-d)	RfD Source	Critical Effect	Oral CSF(mg/kg-d) ⁻¹	Oral CSF Source	Wt. Evidence	Comments
Arsenic	7440-38-2	3.00E-04	IRIS	Hyperpigmentation, keratosis and possible vascular complications	1.50E+00	IRIS	A	
Cadmium	7440-43-9	5.00E-04	mHRV	Renal system	NA	NA	B1	Value for food
Chromium (VI)	18540-29-9	3.00E-03	IRIS	None reported	NA	NA	A	
Hydrogen Chloride (as Cl)	7647-01-0	NA	NA	NA	NA	NA	NA	
Mercury	7439-97-6	3.00E-04	IRIS	Autoimmune effects	NA	NA	C	RfD for mercuric chloride
Nickel	7440-02-0	2.00E-02	mHRV	Decreased body and organ weights	NA	NA	NA	
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	1746-01-6	1.00E-09	HHRAP	Developmental effects	1.40E+06	MDH	B2	

Notes:

CAS - Chemical Abstracts Service.

CSF - Cancer Slope Factor

HHRAP - USEPA Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. September 2005.

IRIS - Integrated Risk Information System. USEPA's on-line database of toxicity information. April, 2006.

MDH - Minnesota Department of Health value (<http://www.health.state.mn.us/divs/eh/risk/guidance/dioxinmemo1.html>).mHRV - Multimedia Health Risk Value developed by MDH (<http://www.health.state.mn.us/divs/eh/air/rules.htm>).

NA - Not available.

RfD - Reference Dose

TEQ - Toxic Equivalents.

TABLE 3-21 ACUTE INHALATION TOXICITY VALUES

Constituent	CAS	HAP?	VOC?	Acute		
				Tox Value Source	Acute Air Conc. (ug/m3)	Toxic Endpoint
Arsenic	7440-38-2	YES	NO	CAL EPA	1.90E-01	Reproductive/ developmental
Cadmium	7440-43-9	YES	NO	NA	NA	
Chromium (VI), particulates	18540-29-9	YES	NO	NA	NA	
Mercury	7439-97-6	YES	NO	CAL EPA	1.80E+00	Reproductive/ developmental
Nickel	7440-02-0	NO	NO	HRV	1.10E+01	Irritant - respiratory system
Hydrogen Chloride (as Cl)	7647-01-0	YES	NO	HRV	2.70E+03	Irritant - eye and respiratory system
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	1746-01-6	YES	NO	NA	NA	
Nitrogen Oxide (NO ₂)	1012-44-0	NO	NO	CAL EPA	470	Respiratory system
Notes:						
CalEPA - California Office of Environmental Health Hazard Assessment.						
CAS - Chemical Abstracts Service.						
HAP - Hazardous Air Pollutants.						
HRV - Minnesota Department of Health Risk Value (http://www.health.state.mn.us/divs/eh/air/rules.htm).						
NA - Not Available.						
TEQ - Toxic Equivalents.						
VOC - Volatile Organic Compound.						

TABLE 3-22 TOTAL RISK ESTIMATES FOR BALLPARK STAFF - PERMIT LIMITS

Compound	Excess Lifetime Cancer Risk			Hazard Indices		
	Inhalation of Air	Ingestion of Dust	Total ELCR	Inhalation of Air	Ingestion of Dust	Total HI
Arsenic	1.31E-09	6.79E-09	8.10E-09	2.84E-05	4.23E-05	7.07E-05
Cadmium	1.05E-08	NC	1.05E-08	8.19E-04	9.37E-03	1.02E-02
Chromium (VI)	7.51E-09	NC	7.51E-09	1.75E-05	1.67E-04	1.85E-04
Hydrogen Chloride (as Cl)	NC	NC	NC	8.85E-04	NC	8.85E-04
Mercury	NC	NC	NC	8.19E-05	1.22E-03	1.30E-03
Nickel	1.31E-10	NC	1.31E-10	3.07E-05	2.19E-05	5.26E-05
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	6.44E-08	3.35E-06	3.41E-06	1.13E-05	6.70E-03	6.71E-03
Sulfuric Acid	NC	NC	NC	3.21E-07	NC	3.21E-07
Total ELCR/HI:	8.E-08	3.E-06	3.E-06	2.E-03	2.E-02	2.E-02

Notes:

ELCR - Excess Lifetime Cancer Risk.

HI - Hazard Indices.

TEQ - Toxic Equivalentents.

TABLE 3-23 TOTAL RISK ESTIMATES FOR BALLPLAYERS - PERMIT LIMITS

Compound	Excess Lifetime Cancer Risk			Hazard Indices		
	Inhalation of Air	Ingestion of Soil	Total ELCR	Inhalation of Air	Ingestion of Soil	Total HI
Arsenic	8.17E-10	4.62E-11	8.64E-10	2.22E-05	3.60E-05	5.81E-05
Cadmium	6.57E-09	NC	6.57E-09	6.38E-04	8.30E-03	8.93E-03
Chromium (VI)	4.68E-09	NC	4.68E-09	1.37E-05	1.48E-04	1.62E-04
Hydrogen Chloride (as Cl)	NC	NC	NC	6.90E-04	NC	6.90E-04
Mercury	NC	NC	NC	6.38E-05	1.03E-03	1.10E-03
Nickel	8.20E-11	NC	8.20E-11	2.39E-05	1.94E-05	4.33E-05
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	4.01E-08	2.28E-08	6.29E-08	8.78E-06	5.70E-03	5.70E-03
Sulfuric Acid	NC	NC	NC	2.50E-04	NC	2.50E-04
Total ELCR/HI:	5.E-08	2.E-08	8.E-08	2.E-03	2.E-02	2.E-02

Notes:

ELCR - Excess Lifetime Cancer Risk.

HI - Hazard Indices.

TEQ - Toxic Equivalentents.

TABLE 3-24 TOTAL RISK ESTIMATES FOR CHILD SEASON TICKET HOLDERS - PERMIT LIMITS

Compound	Excess Lifetime Cancer Risk				Hazard Indices			
	Inhalation of Air	Ingestion of Dust	Food Ingestion	Total ELCR	Inhalation of Air	Ingestion of Dust	Food Ingestion	Total HI
Arsenic	3.15E-10	6.22E-09	1.41E-10	6.68E-09	2.84E-05	1.61E-04	3.66E-06	1.93E-04
Cadmium	2.53E-09	NC	NC	2.53E-09	8.19E-04	3.58E-02	6.71E-04	3.73E-02
Chromium (VI)	1.80E-09	NC	NC	1.80E-09	1.75E-05	6.38E-04	1.20E-05	6.67E-04
Hydrogen Chloride (as Cl)	NC	NC	NC	NC	8.85E-04	NC	NC	8.85E-04
Mercury	NC	NC	NC	NC	8.19E-05	4.64E-03	1.05E-04	4.83E-03
Nickel	3.15E-11	NC	NC	3.15E-11	3.07E-05	8.37E-05	1.57E-06	1.16E-04
2,3,7,8-Tetrachlorodibenzodioxin TEQ	1.54E-08	3.07E-06	6.95E-08	3.15E-06	1.13E-05	2.56E-02	5.79E-04	2.61E-02
Sulfuric Acid	NC	NC	NC	NC	3.21E-04	NC	NC	3.21E-04
Total ELCR/HI:	2.E-08	3.E-06	7.E-08	3.E-06	2.E-03	7.E-02	1.E-03	7.E-02

Notes:

ELCR - Excess Lifetime Cancer Risk.

HI - Hazard Indices.

TEQ - Toxic Equivalents.

TABLE 3-25 TOTAL RISK ESTIMATES FOR ADULT SEASON TICKET HOLDERS - PERMIT LIMITS

Compound	Excess Lifetime Cancer Risk				Hazard Indices			
	Inhalation of Air	Ingestion of Dust	Food Ingestion	Total ELCR	Inhalation of Air	Ingestion of Dust	Food Ingestion	Total HI
Arsenic	1.26E-09	2.67E-09	1.21E-10	4.05E-09	2.84E-05	1.73E-05	7.83E-07	4.65E-05
Cadmium	1.01E-08	NC	NC	1.01E-08	8.19E-04	3.83E-03	1.44E-04	4.79E-03
Chromium (VI)	7.21E-09	NC	NC	7.21E-09	1.75E-05	6.83E-05	2.56E-06	8.84E-05
Hydrogen Chloride (as Cl)	NC	NC	NC	NC	8.85E-04	NC	NC	8.85E-04
Mercury	NC	NC	NC	NC	8.19E-05	4.98E-04	2.25E-05	6.02E-04
Nickel	1.26E-10	NC	NC	1.26E-10	3.07E-05	8.97E-06	3.37E-07	4.00E-05
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	6.18E-08	1.31E-06	5.96E-08	1.44E-06	1.13E-05	2.74E-03	1.24E-04	2.87E-03
Sulfuric Acid	NC	NC	NC	NC	3.21E-04	NC	NC	3.21E-04
Total ELCR/HI:	8.E-08	1.E-06	6.E-08	1.E-06	2.E-03	7.E-03	3.E-04	1.E-02

Notes:

ELCR - Excess Lifetime Cancer Risk.

HI - Hazard Indices.

TEQ - Toxic Equivalents.

TABLE 3-26 TOTAL RISK ESTIMATES FOR BALLPARK STAFF - ACTUAL EMISSIONS

Compound	Excess Lifetime Cancer Risk			Hazard Indices		
	Inhalation of Air	Ingestion of Dust	Total ELCR	Inhalation of Air	Ingestion of Dust	Total HI
Arsenic	1.31E-09	6.79E-09	8.10E-09	2.84E-05	4.23E-05	7.07E-05
Cadmium	1.26E-10	NC	1.26E-10	9.79E-06	1.12E-04	1.22E-04
Chromium (VI)	7.51E-09	NC	7.51E-09	1.75E-05	1.67E-04	1.85E-04
Hydrogen Chloride (as Cl)	NC	NC	NC	2.23E-04	NC	2.23E-04
Mercury	NC	NC	NC	3.85E-06	5.71E-05	6.10E-05
Nickel	1.31E-10	NC	1.31E-10	3.07E-05	2.19E-05	5.26E-05
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	6.79E-09	3.53E-07	3.60E-07	1.19E-06	7.06E-04	7.08E-04
Sulfuric Acid	NC	NC	NC	4.44E-05	NC	4.44E-05
Total ELCR/HI:	2.E-08	4.E-07	4.E-07	4.E-04	1.E-03	1.E-03

Notes:

ELCR - Excess Lifetime Cancer Risk.

HI - Hazard Indices.

TEQ - Toxic Equivalents.

TABLE 3-27 TOTAL RISK ESTIMATES FOR BALLPLAYERS - ACTUAL EMISSIONS

Compound	Excess Lifetime Cancer Risk			Hazard Indices		
	Inhalation of Air	Ingestion of Soil	Total ELCR	Inhalation of Air	Ingestion of Soil	Total HI
Arsenic	1.02E-09	4.62E-11	1.07E-09	2.22E-05	3.60E-05	5.81E-05
Cadmium	9.81E-11	NC	9.81E-11	7.63E-06	9.91E-05	1.07E-04
Chromium (VI)	5.85E-09	NC	5.85E-09	1.37E-05	1.48E-04	1.62E-04
Hydrogen Chloride (as Cl)	NC	NC	NC	1.74E-04	NC	1.74E-04
Mercury	NC	NC	NC	3.00E-06	4.86E-05	5.16E-05
Nickel	1.02E-10	NC	1.02E-10	2.39E-05	1.94E-05	4.33E-05
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	5.29E-09	2.40E-09	7.70E-09	9.26E-07	6.01E-04	6.02E-04
Sulfuric Acid	NC	NC	NC	3.46E-05	NC	3.46E-05
Total ELCR/HI:	1.E-08	2.E-09	1.E-08	3.E-04	1.E-03	1.E-03

Notes:

ELCR - Excess Lifetime Cancer Risk.

HI - Hazard Indices.

TEQ - Toxic Equivalents.

TABLE 3-28 TOTAL RISK ESTIMATES FOR CHILD SEASON TICKET HOLDERS - ACTUAL EMISSIONS

Compound	Excess Lifetime Cancer Risk				Hazard Indices			
	Inhalation of Air	Ingestion of Dust	Food Ingestion	Total ELCR	Inhalation of Air	Ingestion of Dust	Food Ingestion	Total HI
Arsenic	3.15E-10	6.22E-09	1.41E-10	6.68E-09	2.84E-05	1.61E-04	3.66E-06	1.93E-04
Cadmium	3.02E-11	NC	NC	3.02E-11	9.79E-06	4.27E-04	8.02E-06	4.45E-04
Chromium (VI)	1.80E-09	NC	NC	1.80E-09	1.75E-05	6.38E-04	1.20E-05	6.67E-04
Hydrogen Chloride (as Cl)	NC	NC	NC	NC	2.23E-04	NC	NC	2.23E-04
Mercury	NC	NC	NC	NC	3.85E-06	2.18E-04	4.94E-06	2.27E-04
Nickel	3.15E-11	NC	NC	3.15E-11	3.07E-05	8.37E-05	1.57E-06	1.16E-04
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	1.63E-09	3.23E-07	7.33E-09	3.32E-07	1.19E-06	2.70E-03	6.11E-05	2.76E-03
Sulfuric Acid	NC	NC	NC	NC	4.44E-05	NC	NC	4.44E-05
Total ELCR/HI:	4.E-09	3.E-07	7.E-09	3.E-07	4.E-04	4.E-03	9.E-05	5.E-03

Notes:

ELCR - Excess Lifetime Cancer Risk.

HI - Hazard Indices.

TEQ - Toxic Equivalents.

TABLE 3-29 TOTAL RISK ESTIMATES FOR ADULT SEASON TICKET HOLDERS - ACTUAL EMISSIONS

Compound	Excess Lifetime Cancer Risk				Hazard Indices			
	Inhalation of Air	Ingestion of Dust	Food Ingestion	Total ELCR	Inhalation of Air	Ingestion of Dust	Food Ingestion	Total HI
Arsenic	1.26E-09	2.67E-09	1.21E-10	4.05E-09	2.84E-05	1.73E-05	7.83E-07	4.65E-05
Cadmium	1.21E-10	NC	NC	1.21E-10	9.79E-06	4.58E-05	1.72E-06	5.73E-05
Chromium (VI)	7.21E-09	NC	NC	7.21E-09	1.75E-05	6.83E-05	2.56E-06	8.84E-05
Hydrogen Chloride (as Cl)	NC	NC	NC	NC	2.23E-04	NC	NC	2.23E-04
Mercury	NC	NC	NC	NC	3.85E-06	2.34E-05	1.06E-06	2.83E-05
Nickel	1.26E-10	NC	NC	1.26E-10	3.07E-05	8.97E-06	3.37E-07	4.00E-05
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	6.52E-09	1.39E-07	6.28E-09	1.51E-07	1.19E-06	2.89E-04	1.31E-05	3.03E-04
Sulfuric Acid	NC	NC	NC	NC	4.44E-05	NC	NC	4.44E-05
Total ELCR/HI:	2.E-08	1.E-07	6.E-09	2.E-07	4.E-04	5.E-04	2.E-05	8.E-04

Notes:

ELCR - Excess Lifetime Cancer Risk.

HI - Hazard Indices.

TEQ - Toxic Equivalents.

TABLE 3-30 COMPARISON OF MAXIMUM MODELED CONCENTRATIONS OF CRITERIA POLLUTANTS TO MINNESOTA AMBIENT AIR QUALITY STANDARDS (MAAQS) BASED ON ACTUAL EMISSIONS

Pollutant	Avg Period	MAAQS		Emissions	Max Chi/Q	Impact	Background**	Total	Percent of MAAQS	
		Primary (µg/m ³)	Secondary (µg/m ³)	(g/sec)	µg/m ³ per g/sec	µg/m ³	µg/m ³	µg/m ³	Primary	Secondary
SO2	1-hour	1300	None	4.28E-01	1.29604	5.54E-01	351	351	27%	NA
	3-hour	None	1300	4.28E-01	1.11517	4.77E-01	283	283	NA	22%
	24-hour	365	None	4.28E-01	0.33167	1.42E-01	157	157	43%	NA
	Annual	80	60	3.98E-01	0.02558	1.02E-02	10	10	12%	17%
TSP	24-hour	260	150	1.07E+00	0.33167	3.56E-01	105	105	41%	70%
	Annual	75	60	6.59E-01	0.02558	1.69E-02	41	41	55%	69%
PM10	24-hour	150	150	6.69E-01	0.33167	2.22E-01	65	65	43%	43%
	Annual	50	50	4.10E-01	0.02558	1.05E-02	25	25	50%	50%
PM2.5	24-hour	35	35	4.08E-01	0.33167	1.35E-01	28	28	81%	81%
	Annual	15	15	3.49E-01	0.02558	8.92E-03	10	10	65%	65%
NO2	Annual	100	100	1.41E+01	0.02558	3.61E-01	23	24	24%	24%
CO	1-hour	35,000	35,000	2.17E+00	1.29604	2.82E+00	5382	5385	15%	15%
	8-hour	10,000	10,000	2.17E+00	0.71235	1.55E+00	2634	2635	26%	26%
Lead*	Quarterly	1.5	1.5	4.86E-04	0.05465	2.65E-05	0.032	0.03	2%	2%

* Modeled impact conservatively based on 1-month averaging period.

** 2003-2005 MPCA ambient monitoring data: SO2, CO - site 954, Lead - site 965, NO2 - site 1002, TSP, PM2.5 - site 963, PM10 - site 966

TABLE 3-31 COMPARISON OF MAXIMUM MODELED CONCENTRATIONS OF CRITERIA POLLUTANTS TO MINNESOTA AMBIENT AIR QUALITY STANDARDS (MAAQS) BASED ON PERMITTED EMISSIONS

Pollutant	Avg Period	MAAQS		Emissions	Max Chi/Q	Impact	Background**	Total	Percent of MAAQS	
		Primary (µg/m ³)	Secondary (µg/m ³)	(g/sec)	µg/m ³ per g/sec	µg/m ³	µg/m ³	µg/m ³	Primary	Secondary
SO2	1-hour	1300	None	2.25E+01	1.29604	2.91E+01	351	380	29%	NA
	3-hour	None	1300	2.25E+01	1.11517	2.51E+01	283	308	NA	24%
	24-hour	365	None	2.25E+01	0.33167	7.45E+00	157	165	45%	NA
	Annual	80	60	2.87E+00	0.02558	7.35E-02	10	10	13%	17%
TSP	24-hour	260	150	5.04E+00	0.33167	1.67E+00	105	107	41%	71%
	Annual	75	60	5.04E+00	0.02558	1.29E-01	41	41	55%	69%
PM10	24-hour	150	150	3.13E+00	0.33167	1.04E+00	65	66	44%	44%
	Annual	50	50	3.13E+00	0.02558	8.01E-02	25	25	50%	50%
PM2.5	24-hour	35	35	4.08E-01	0.33167	1.35E-01	28	28	81%	81%
	Annual	15	15	3.49E-01	0.02558	8.92E-03	10	10	65%	65%
NO2	Annual	100	100	4.24E+01	0.02558	1.09E+00	23	24	24%	24%
CO	1-hour	35,000	35,000	1.26E+01	1.29604	1.63E+01	5382	5399	15%	15%
	8-hour	10,000	10,000	1.26E+01	0.71235	8.98E+00	2634	2643	26%	26%
Lead*	Quarterly	1.5	1.5	4.84E-02	0.05465	2.65E-03	0.032	0.03	2%	2%

* Modeled impact conservatively based on 1-month averaging period.

** 2003-2005 MPCA ambient monitoring data: SO2, CO - site 954, Lead - site 965, NO2 - site 1002, TSP, PM2.5 - site 963, PM10 - site 966

TABLE 3-32 ACUTE RISK SUMMARY - PERMIT LIMITS

Compound	Acute Air Concentration (ug/m³) (a)	Acute Toxicity Value (ug/m³)	Acute Inhalation Hazard Quotient
Arsenic	3.58E-04	1.90E-01	1.88E-03
Cadmium	6.87E-03	NA	NC
Chromium (VI), particulates	7.35E-04	NA	NC
Mercury	1.17E-02	1.80E+00	6.52E-03
Nickel	6.43E-04	1.10E+01	5.84E-05
Hydrogen Chloride (as Cl)	7.42E+00	2.70E+03	2.75E-03
2,3,7,8-Tetrachlorodibenzodioxin TEQ	1.89E-07	NA	NC
Nitrogen Oxide (NO ₂)	5.66E+01	4.70E+02	1.20E-01
Acute Hazard Index for HERC:			1.E-01
Measured Background Nitrogen Oxide (NO ₂):	1.09E+02	4.70E+02	2.E-01
Acute Hazard Index including Nitrogen Oxide (NO ₂) Background:			4.E-01

Notes:

NA - Not Available.

NC - Not Calculated.

TEQ - Toxic Equivalents.

(a) This is the maximum estimated 1-hr air concentration based on permit limits.

TABLE 3-33 ACUTE RISK SUMMARY - ACTUAL EMISSIONS

Compound	Acute Air Concentration (ug/m³) (a)	Acute Toxicity Value (ug/m³)	Acute Inhalation Hazard Quotient
Arsenic	3.58E-04	1.90E-01	1.88E-03
Cadmium	2.60E-04	NA	NC
Chromium (VI), particulates	7.35E-04	NA	NC
Mercury	1.56E-03	1.80E+00	8.67E-04
Nickel	6.43E-04	1.10E+01	5.84E-05
Hydrogen Chloride (as Cl)	2.37E+00	2.70E+03	8.79E-04
2,3,7,8-Tetrachlorodibenzopdioxin TEQ	1.32E-07	NA	NC
Nitrogen Oxide (NO ₂)	1.94E+01	4.70E+02	4.13E-02
Acute Hazard Index for HERC:			4.E-02
Measured Background Nitrogen Oxide (NO ₂):	1.09E+02	4.70E+02	2.E-01
Acute Hazard Index including Nitrogen Oxide (NO ₂) Background:			3.E-01

Notes:

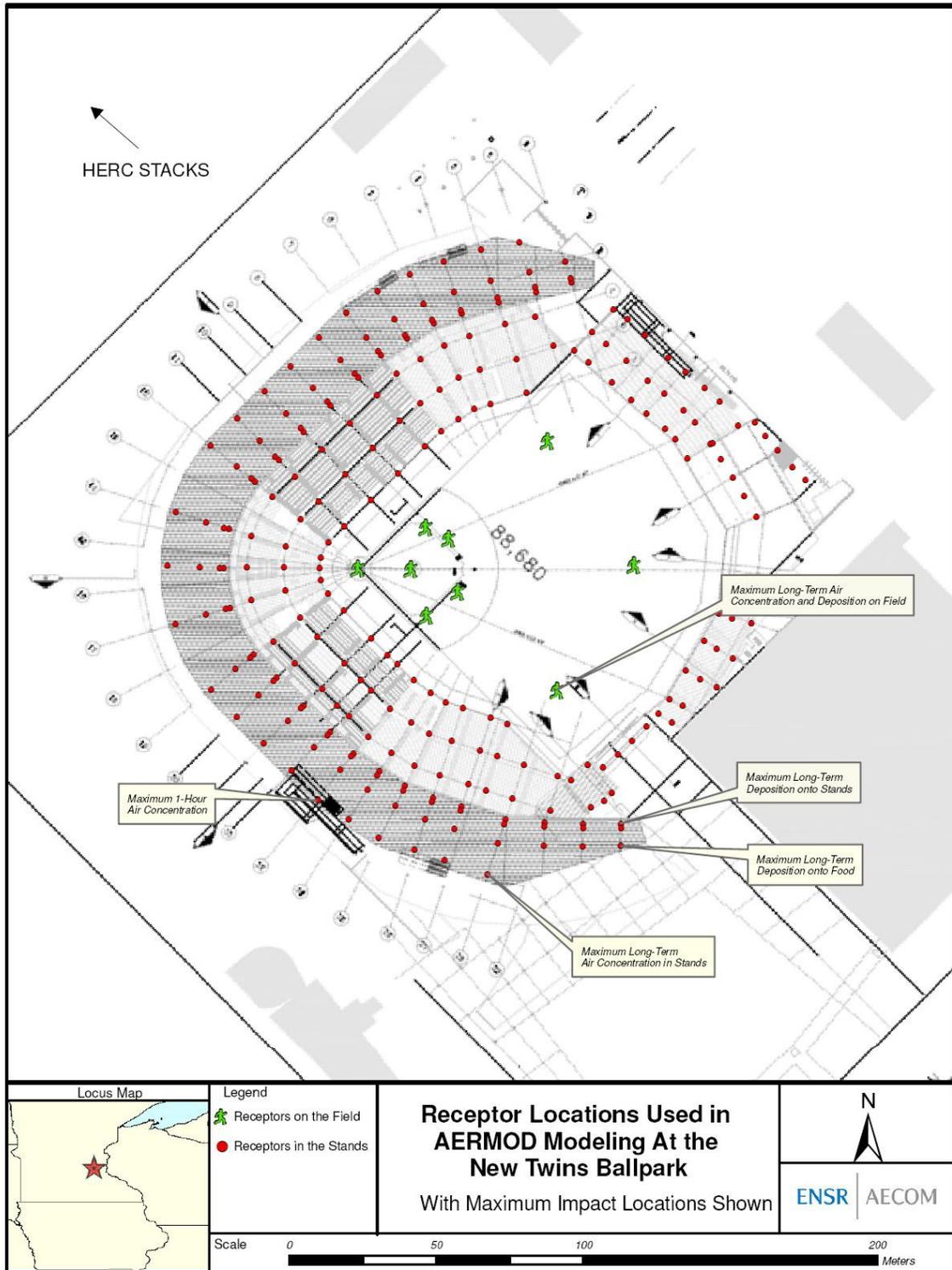
NA - Not Available.

NC - Not Calculated.

TEQ - Toxic Equivalents.

(a) This is the maximum estimated 1-hr air concentration based on measured emissions.

Figure 3-1 Ballpark Model Receptors Locations



4.0 Summary and Conclusions

Hennepin County and the Minnesota Ballpark Authority are building a new 40,000-seat, open-air baseball park at a site one block northwest of the Target Center between 5th Street North and 7th Street North on the edge of the Warehouse District in Downtown Minneapolis, Hennepin County, Minnesota. On behalf of Hennepin County, ENSR Corporation (ENSR) has conducted this study to determine (1) the potential impact of the new Minnesota Twins ballpark on the Hennepin Energy Recovery Center (HERC) stack dispersion patterns, and (2) the potential health risk impact of HERC emissions on ballpark users.

Potential impact of the ballpark structure on HERC stack dispersion

The new Minnesota Twins ballpark is proposed to have three levels of seating. The height of the ballpark is about 200 feet, and portions of the park will be within 1000 feet of the HERC stacks. It is possible, therefore, that the new structure could alter the local turbulence patterns that affect dispersion of HERC emissions. The potential for the ballpark to affect HERC dispersion was first evaluated by applying EPA's Building Profile Input Program (BPIP-Prime) for two configurations, one as it exists today and another with ballpark in-place. Because BPIP analysis indicated that some changes in building downwash could occur, the importance of these changes was then evaluated through dispersion modeling with EPA's advanced model AERMOD. This dispersion analysis showed that slight changes to dispersion will occur to the northwest of HERC but the ballpark would not affect the magnitude or location of the maximum modeled concentrations associated with HERC.

Potential health risk impacts of HERC emissions on ballpark users

AERMOD was also used to estimate air concentrations of compounds potentially emitted by HERC and estimate deposition on the playing field and in the stands of the ballpark for a variety of receptors including ballplayers, staff and season ticket holders. Two sets of emissions were applied, corresponding to (1) upper limits provided in the Title V permit and (2) more realistic emissions based on the average of recent source measurement tests. These estimated concentrations and rates of deposition were used in a health risk assessment to determine potential health risks to ballpark users. Both long-term (carcinogenic and noncarcinogenic) and acute risks were evaluated following USEPA and MPCA guidance.

Although many receptors are possible, the ones likely to receive the highest impacts were selected for evaluation in the HHRA. These receptors included ballpark staff, ballplayers and child and adult season ticket holders. It was assumed that these receptors could be exposed through inhalation of compounds in air and ingestion of soil and dust. For the season ticket holders, ingestion of compounds in food was also evaluated.

The risk calculations showed that for emissions based on both Permit Limits and measurements, the chronic carcinogenic and noncarcinogenic risk results for the various receptors are less than USEPA and MPCA's cancer risk level of 1×10^{-5} and HI of 1. The criteria pollutants evaluated in this HHRA (carbon monoxide, sulfur dioxide, nitrogen dioxide, lead, PM-10 and PM-2.5) were evaluated by comparing estimated annual average and short-term air concentrations against MAAQS. Nitrogen dioxide was also evaluated for acute health effects. Health effects from most of these compounds are mainly through the inhalation route, therefore the comparison with MAAQS is an appropriate approach. All of the estimated air concentrations are lower than their respective MAAQS.

An acute risk evaluation was conducted for inhalation exposure to maximum 1-hour air concentrations emitted from the HERC facility. For emissions based on both Permit Limits and measurements, the air concentrations were all below acute benchmarks.

These results show that even if the emissions from HERC are at the Permit Limits, the potential health risks to people working at and visiting the ballpark are below levels of concern. The risk estimates are even lower if measured emissions data are used.

5.0 References

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Appendices

(provided in electronic format on enclosed CD)

Appendix A Comparative Dispersion Factor Analysis

“Appendix A - Effect of Ballpark on HERC Stack.xls”

Appendix B Emissions Calculations

“Appendix B - Emissions.xls”

Appendix C Computation of Exposure Point Concentrations

“Appendix C1 – Dispersion Factor in Stands.xls”

“Appendix C2 – Dispersion Factor on Field.xls”

“Appendix C3 - Deposition onto Food.xls”

“Appendix C4 - Long Term Deposition on Field.xls”

“Appendix C5 - Seasonal Deposition on Stands.xls”

“Appendix C6 - Summary of Background Deposition.xls”

Appendix D Risk Calculations (“pdf” file)