

# Letter Health Consultation

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POPE DOUGLAS SOLID WASTE MANAGEMENT

ALEXANDRIA, POPE AND DOUGLAS COUNTIES, MINNESOTA

**Prepared by the  
Minnesota Department of Health**

SEPTEMBER 25, 2009

Prepared under a Cooperative Agreement with the  
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Agency for Toxic Substances and Disease Registry  
Division of Health Assessment and Consultation  
Atlanta, Georgia 30333

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LETTER HEALTH CONSULTATION

POPE DOUGLAS SOLID WASTE MANAGEMENT  
ALEXANDRIA, POPE AND DOUGLAS COUNTIES, MINNESOTA

Prepared By:

Minnesota Department of Health  
Under Cooperative Agreement with  
U.S. Department of Health and Human Services  
Agency for Toxic Substances and Disease Registry

August 18, 2009

Heather Magee-Hill  
Minnesota Pollution Control Agency (MPCA)  
520 Lafayette Road  
Saint Paul, Minnesota 55155-4194

Dear Ms. Magee-Hill,

This letter summarizes the results of the review conducted, at your request, of the Human Health Risk Assessment Report “Working Draft” (HHRAR; Wenck Associates Inc., 2009) submitted by the Pope Douglas Solid Waste Management (Pope/Douglas). The HHRAR was submitted as part of the application for a Permit to increase the waste combustion capacity of this waste-to-energy facility. The health risk assessment was conducted using US Environmental Protection Agency (EPA) guidance *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities* (HHRAP; EPA, 2005a), supplemented or superseded by MPCA requirements. The Industrial Risk Assessment (computer) Program – Human Health (IRAP-h; Lakes Environmental Software) was used to calculate risks and hazards.

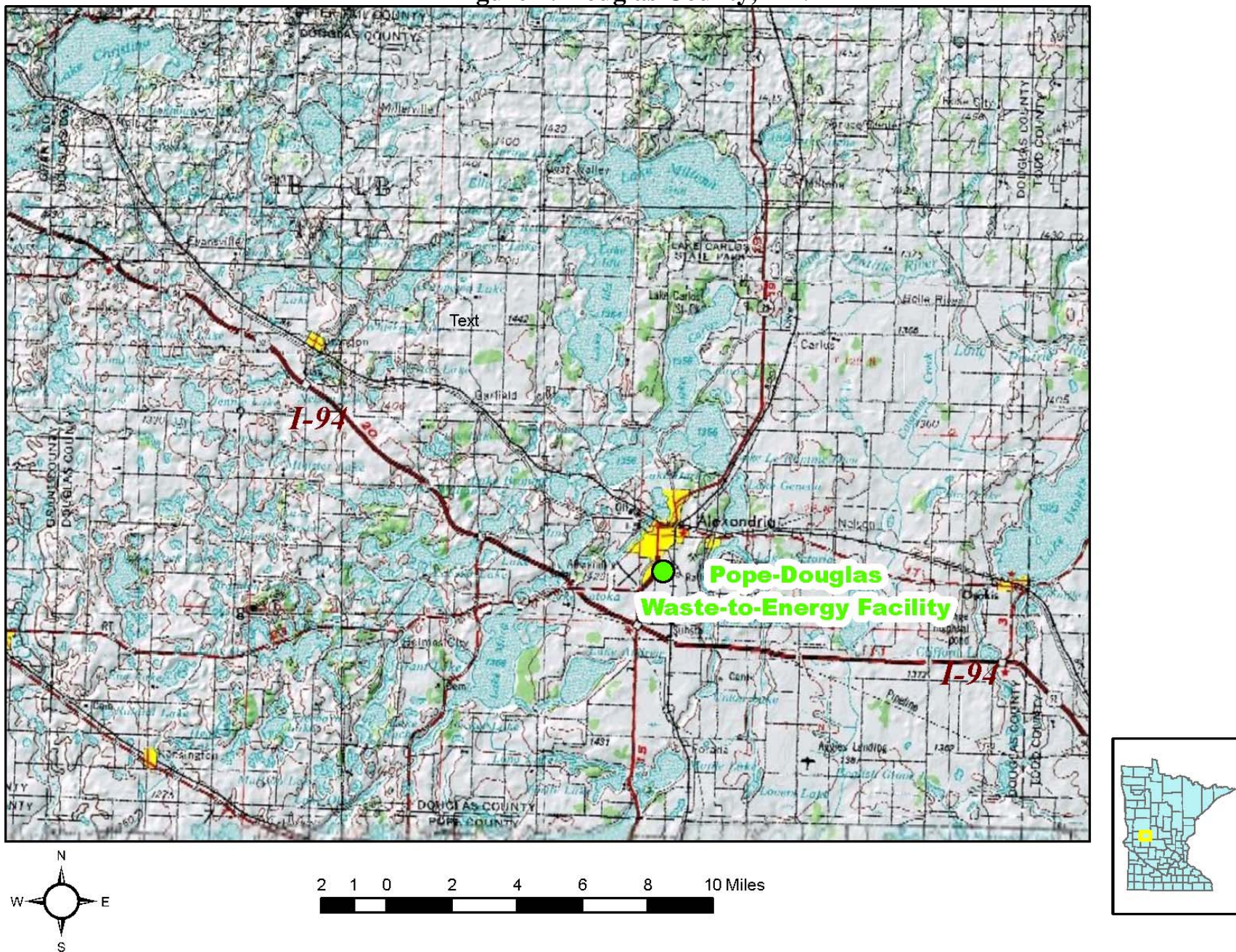
Previous work by the Minnesota Department of Health (MDH) included participating in a site visit to the facility in December 2008 and commenting on the Draft HHRAP in early February 2008. On June 22, 2009 MDH was asked to comment on the HHRAR. The issues addressed in this letter were first discussed with the MPCA on July 9, 2009. MPCA responses were presented to the company and me during a telephone conference call on July 15, 2009, and recorded in the notes of that call (Attachment 1, pages 7-8).

### **Background and Site History**

The Pope/Douglas waste-to-energy plant is located in the city of Alexandria in west-central Minnesota, about 120 miles northwest of Minneapolis (Figure 1). The facility, which opened in 1987, has 2 - 60 ton per day separate but similar waste combustors (Units 1 and 2). These combustors provide steam that serves district heating customers in properties adjacent to the facility. Customers include a 3M Company facility and the Douglas County Hospital. Remaining steam is used to power a 500-kilowatt steam turbine generator. The proposed addition will add a 120 ton per day combustor (Unit 3), which can provide additional steam generating capacity for serving new customers which may include the Alexandria Technical College.

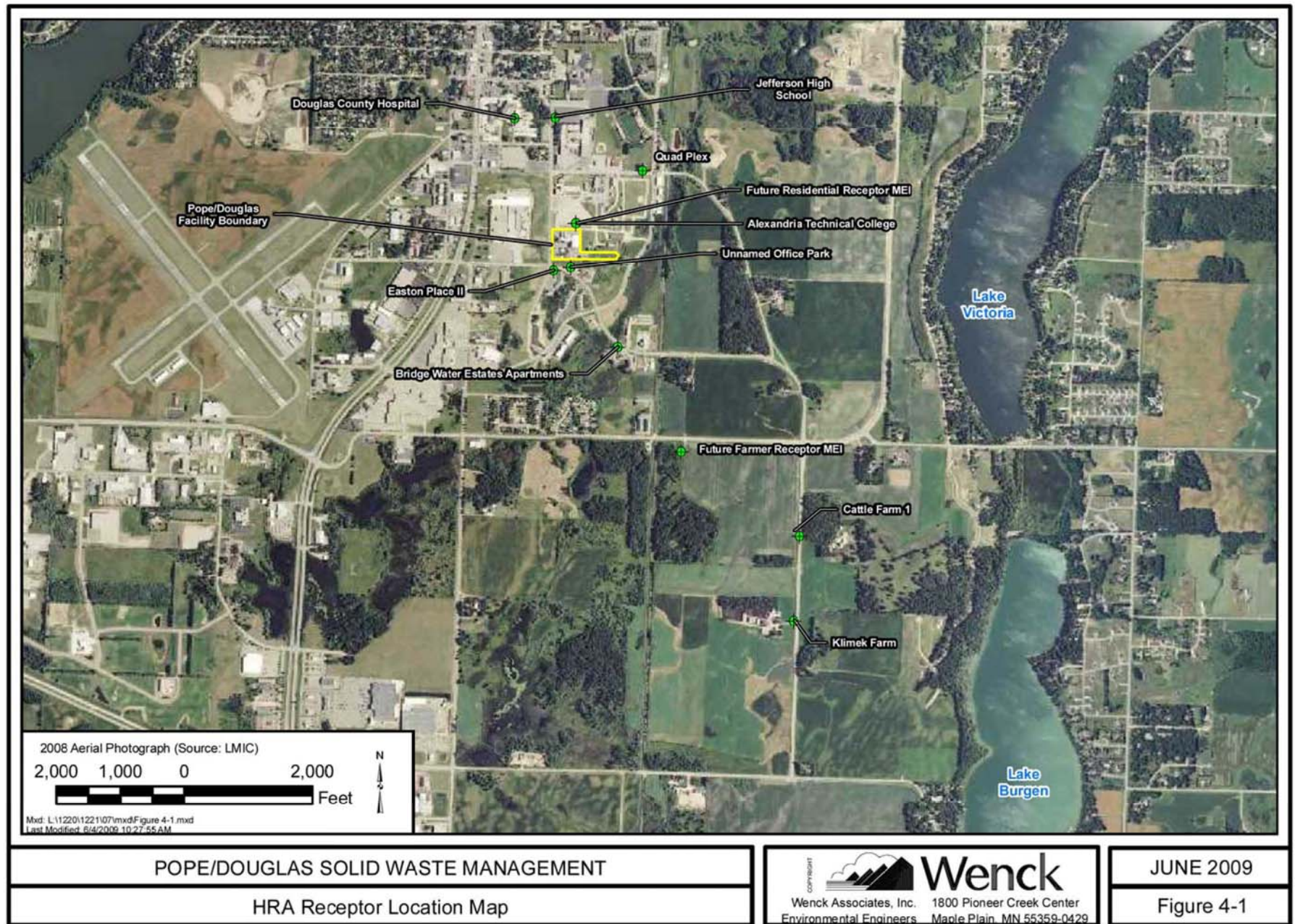
The current facility, as well as the planned expansion, use carbon injection coupled with baghouses to remove much of the mercury, as well as dioxin and other organic compounds, from exhaust gases prior to emission.

Figure 1: Douglas County, MN





**Figure 2: Pope/Douglas Waste-to-Energy Facility**



## **MDH Human Health Risk Assessment Report (HHRAR) Review**

The IRAP models the dispersion of emissions from the stack and the potential health risks and hazards from individual chemicals to people at various locations, including homes and businesses, near the facility. Figure 2 shows the locations modeled. Exposure scenarios included workplace and school exposures (Unnamed Office Park, Alexandria Technical College), residences (Future Residential Receptor – Most Exposed Individual [MEI], Bridgewater Estates Apartments), farming (Cattle Farm 1), farming including dairy (Future Farmer Receptor – Most Exposed Individual), fishers (Lake Victoria and Lake Burgen). Fisher exposures (and consequent risks) are calculated for the fish consumption pathway and may be added to various residential and farming exposures, depending on the fishers assumed residence location.

The total mass of emissions, speciated dioxin-like emissions, and mercury emissions were measured during stack testing at Pope/Douglas. Chemical composition of the stack emissions for all chemicals except dioxin-like compounds and total mercury were estimated using emission factors from a similar facility (Olmsted County Incinerator) in Minnesota.

The MPCA identified some errors in the modeling parameters used in the IRAP for this facility. These included the use of 20 years of facility use/deposition instead of the typical 30 years; failure to use the receptor with the highest risk for chronic exposure to all forms of chemicals (wet, dry, particulate, vapor) in the risk calculations; and inappropriate lake depth information. As a result, the final hazards and risks for the proposed project are likely to be somewhat different than the risks calculated in the HHRAR.

MDH comments on the HHRAR are related to Appendices E-G and Tables in the main document that summarize data in these appendices (e.g. Tables 4-1, 4-2, 4-3). Appendix E of the HHRAR shows the calculated adult and child cancer risks and non-cancer hazards from individual chemicals through 3 different scenarios (above ground vegetables, inhalation, soil), 4 different exposure locations (Bridgewater Estates Apartments, Quadplex, Easton Place II, and Zoned Residential MEI). Appendix F shows the calculated adult and child cancer risks and non-cancer hazards from individual chemicals (through above ground vegetables, inhalation, beef, chicken, eggs, pork, milk, soil) at the location of Cattle Farm 1 (see Figure 2) and at the Zoned Agricultural MEI (Future Farmer Receptor MEI – Figure 2). Appendix G shows the calculated adult and child cancer risks and non-cancer hazards from subsistence and recreational fish consumption from Lake Victoria and Lake Burgen.

Upon review of Appendices E-G, MDH identified the following issues or inconsistencies in the cancer risks and non-cancer hazards:

1. No inhalation hazard was identified for lead, and yet lead hazard and cancer risks were determined for consumption of lead from above ground vegetables and soil.

2. Childhood cancer risk in the HHRAR is less than adult cancer risk for all carcinogens. Yet for carcinogens where the effects of early and later-life exposures have been studied (animal and human studies), early life exposure often increases the cancer risk.
3. Cancer risk from polycyclic aromatic hydrocarbons (PAHs) was determined from data on 8 carcinogenic polycyclic aromatic hydrocarbons (cPAHs). However, MDH recommends using the extended list of cPAHs for evaluating cancer risk from PAHs.
4. The use of the term “Current risk” when describing soil exposure is confusing. The permit is for incremental risk from the facility and does not include an evaluation of current exposure to previous deposition of facility emissions.

## Discussion

### *Lead Inhalation Hazard*

The HHRAR reported the incremental addition from facility emissions to the noncancer hazard quotients from ingestion of lead in soil and ingestion of lead in food as well as the incremental cancer risk from lead inhalation, soil ingestion and the ingestion of farm produce. Cancer risk was determined using the lead unit risk from the MPCA Risk Assessment Spreadsheet model (RASS; <http://www.pca.state.mn.us/publications/aq9-22.zip>) ( $1.2E-5$  ( $\mu\text{g}/\text{m}^3$ )<sup>-1</sup>). (The source of this toxicity value is the California Office of Environmental Health Hazard Assessment (OEHHA)). However, the greatest health impact from a facility emitting lead into the air is likely from inhalation. There is a National Ambient Air Quality Standard (NAAQS) for lead, set to be protective of human health, that was updated in October 2008 to a 3-month rolling average standard of  $0.15$   $\mu\text{g}/\text{m}^3$ .

Lead emissions from the facility in the January 12, 2009 MPCA Emissions Calcs spreadsheet were estimated at about 0.1 pounds per hour (lb/hr) and 0.39 tons per year (tpy). At this rate, assuming conservative default stack height of 28 meters (m), the RASS suggests that the lead concentration at 15 meters from the stack may exceed the NAAQS for lead by 7 times (MDH analysis). (It is not clear from the MPCA documentation whether the RASS calculates exceedance of a quarterly limit of  $0.15\mu\text{g}/\text{m}^3$  or whether the RASS calculates exceedance of a 3 month rolling average of  $0.15$   $\mu\text{g}/\text{m}^3$ .)

The emission rates were preliminary estimates; stack heights and distance to fence line were estimated for use in the RASS. The RASS model is more conservative than the IRAP model which was used to model facility emissions of other chemicals. The IRAP model accepts site-specific data.

### *Childhood cancer risk*

The HHRAR shows the childhood cancer risk to be less than the adult cancer risk for all chemicals. However, as noted in the EPA Supplemental Guidance (EPA, 2005b), OEHHA Technical Support Document For Cancer Potency Factors (OEHHA, 2009), and MDH Health Risk Limit Guidance (MDH, 2008), carcinogens are, generally, more potent to children than they are to adults.



One reason why childhood cancer risk in the HHRAR is less than adult cancer risk is that the childhood risk is calculated for 6 years and averaged over a full 70 year lifetime, and the adult risk is calculated for 30 years and averaged over 70 years. In addition, the IRAP does not include age-adjusted cancer potencies.

Table 1 shows the cancer risk for an individual living the first 30 years of their life (0-29 years-old) at the location of the most exposed farmer, recalculated with 2 different adjustments. Cancer risks quantified in Table 1 are: HHRAR adult and childhood risks; HHRAR-modified risk calculated using 6 years of HHRAR Childhood Risk and 24 years of HHRAR Adult Risk (with no potency adjustment); and age-adjusted cancer risk calculated for all carcinogens, except 2,3,7,8-tetrachlorinated dibenzo-p-dioxin-like dioxins and furans (dioxins), by increasing the cancer potency by a factor of 10 for exposures from ages 0-1 years-old, and by a factor of 3 for ages 2-15. (At this time, MDH does not recommend a potency adjustment for childhood dioxin exposure. See <http://www.health.state.mn.us/divs/eh/risk/guidance/dioxinmemo1.html> )

**Table 1: Most Exposed Farmer Non-dioxin Cancer Risk**

HHRAR Adult Risk	HHRAR Childhood Risk	HHRAR-modified (childhood 0-5, adult 6-29)	Age-adjusted Risk (0-29 years old)
4.37E-06	8.69E-07	4.37E-06	1.08E-05

Note that the 30 year risk calculated from the HHRAR data is identical to the HHRAR Adult Risk. This suggests that the annual HHRAR Childhood Risk is the same as the annual HHRAR Adult Risk. When potency is adjusted for age, the 30 year total non-dioxin cancer risk is 2.46 times the adult risk noted in the HHRAR.

*Cancer risk from polycyclic aromatic hydrocarbons*

Data from the HHRAR indicate 2.53E-6 adult cancer risk from 8 cPAHs (benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, indeno(1,2,3-cd) pyrene, naphthalene). When these data are age-adjusted for increased early-life potency, the total cancer risk is 6.44E-6. This chemical group-specific risk is over 1/2 of the total non-dioxin cancer risk (see Table 1, above).

General MDH guidance on evaluation of the cancer risk from PAHs recommends evaluating an extended list of 25 cPAHs (MDH, 2001). The cPAH cancer risk could be significantly higher if the extended list of cPAHs is analyzed.

Dioxin-like chemical and mercury data are the only chemical-specific stack test data available for Pope/Douglas. Speciation of other chemicals emitted from the current Pope/Douglas stacks has not been conducted. The facility is using data acquired from another waste-to-energy facility (Olmsted County) for most chemicals. Olmsted County data on cPAHs is limited to the 8 cPAHs listed above. MPCA staff have noted that there is considerable difference between the dioxin emissions per mass total emissions from the

Olmsted County facility and the Pope/Douglas facility (personal communication, Heather Magee-Hill). Similar to dioxin, most of the PAHs emitted are products of combustion and not chemicals originating in the products that are burned. Therefore, there may be significant differences between the 2 facilities in species and amounts of PAHs formed. It may be prudent to perform a stack test at Pope/Douglas for the extended list of cPAHs.

*Use of the term “Current Risk”*

The HHRAR uses the term “current risk” to describe the risk from soil in 20 years if Pope/Douglas does not expand, and “future risk” to describe risk in 20 years if the facility expands. This is somewhat confusing. It would be better if the HHRAR compared the “future risk” for each of the 2 options.

## **Conclusions**

The Human Health Risk Assessment Report for Pope Douglas Solid Waste Management is an assessment of the future impacts of the current Pope/Douglas facility and also an assessment of the potential future impacts of an additional proposed incineration unit. The exposure modeling relies heavily on chemical-specific emission factors from a similar facility in Minnesota for characterizing current emissions, as well as potential future emissions. Dioxin-like chemical emissions, which have been estimated using site-specific data, provide the greatest cancer health risk from the facility. Carcinogenic PAH emissions are also likely to be significant. Acute and chronic non-cancer hazards, except for lead, are adequately evaluated in the Report and are not commented on in this letter.

Specific MDH conclusions:

- Conservative modeling of lead concentrations in air near the Pope/Douglas facility suggests that lead emissions from this facility may be an inhalation concern.
- The HHRAR does not combine childhood and adult cancer risk in a single risk calculation for a child that grows into adulthood.
- The HHRAR does not adjust the cancer risk for young people to reflect increased early life susceptibility to cancer.
- The HHRAR calculates the total PAH cancer risk from only a few carcinogenic PAHs, and emission factors for the remaining cPAHs on the extended list were not available from the data set used for calculating cPAH cancer risk.
- “Current risk” in the HHRAR means future risk from the facility operating under current conditions.

## **Recommendations and Outcomes**

**MDH recommended** that total (facility and background) lead concentration in ambient air near the facility should be characterized and compared with the NAAQS.

**Outcome:**

- In response to a request from MPCA, Pope-Douglas modeled lead emissions in IRAP and determined that, when site-specific parameters were used to model

dispersion and exposure, the concentration of lead in air near the facility, including background air concentrations, should not exceed the NAAQS. The memo containing the modeling results is attached to this letter (Attachment 2).

- The MPCA intends to update their emission risk guidance to include evaluating lead inhalation.

**MDH recommended** that the potencies (cancer slope factors) for all carcinogens, except dioxin-like carcinogens, be increased by a default factor of 10 for ages 0-1, and by a factor of 3 for ages 2-15. This guidance is consistent with guidance on the MDH Health Risk Limits (MDH, 2008). Adjustment of dioxin-like carcinogens is not recommended at this time (<http://www.health.state.mn.us/divs/eh/risk/guidance/dioxinmemo1.html>). In addition, MDH has offered to review the appropriateness of adjusting the potency for carcinogenic risk-drivers with established modes of action that may exclude increased early-life susceptibility.

**Outcome:**

- MPCA noted that the incremental dioxin risk from this facility (approximately 5E-5) is considerably larger than the non-dioxin risk. MPCA has proposed that Pope/Douglas lower their dioxin emission limits to decrease dioxin risks.
- MDH and MPCA will begin discussions on incorporating age-adjusted potencies into cancer risk evaluations within different program areas.
- In future projects MPCA will be considering including a post-IRAP analysis to make childhood and adult risks more easily compared.

**MDH recommended** evaluating cPAH cancer risk from an extended list of PAHs.

**Outcome:**

- MPCA is looking into the feasibility of conducting PAH stack testing for the extended list of cPAHs at Pope/Douglas.

**MDH recommended** that the HHRAR describe the “future risk” for each of the 2 options instead of using “current risk” to describe the risk in the future from the current facility.

**Actions taken:**

- MPCA agrees with the comment and anticipates that the final HHRAR will clarify these options.

Sincerely,



Carl Herbrandson, PhD  
Toxicologist  
Minnesota Department of Health

## References

- U.S. Environmental Protection Agency (2005a). Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Final, Office of Solid Waste and Emergency Response, Washington, DC. EPA530-R-05-006, September 2005. <http://www.epa.gov/waste/hazard/tsd/td/combust/risk.htm#hhrad>
- U.S. Environmental Protection Agency (2005b). Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. Final, Risk Assessment Forum, Washington, DC. EPA/630/R-03/003F, March 25, 2005. [http://www.epa.gov/raf/publications/cancer\\_guidelines/guidelines-carcinogen-supplement.htm](http://www.epa.gov/raf/publications/cancer_guidelines/guidelines-carcinogen-supplement.htm)
- Health, M.D.o. (2008). Statement of Need and Reasonableness: In the Matter of Proposed Rules Of the Minnesota Department of Health Relating to Health Risk Limits for Groundwater. Final, St. Paul, MN. July 11, 2008. <http://www.health.state.mn.us/divs/eh/risk/rules/hrlsonar08.pdf>
- Minnesota Department of Health, (2001). Methods for Estimating the Carcinogenic Health Risks from Polycyclic Aromatic Hydrocarbons. Herbrandson, C. and C. Stroebel, Environmental Assessment and Surveillance, Environmental Health, St. Paul, MN. Memo To: Goeden, H., Minnesota Pollution Control Agency. May 29, 2001. <http://www.health.state.mn.us/divs/eh/risk/guidance/pahmemo.html>
- California Environmental Protection Agency Office of Environmental Health Hazard Assessment (2009). Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. Final, Air Toxicology and Epidemiology Branch, Sacramento, CA. May 2009. [http://www.oehha.org/air/hot\\_spots/tsd052909.html](http://www.oehha.org/air/hot_spots/tsd052909.html)
- Wenck Associates Inc. (2009). Human Health Risk Assessment (HHRA) for the proposed PDSWM Unit 3 Expansion Project. Working Draft, Woodbury, MN. June 19, 2009.



## Certification

The Minnesota Department of Health prepared this Letter Health Consultation, Pope/Douglas Waste to Energy, under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). At the time this Health Consultation was written, it was in accordance with the approved methodologies and procedures. Editorial review was completed by the Cooperative Agreement partner.



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Technical Project Officer, Cooperative Agreement Team, CAPEB, DHAC, ATSDR

The Division of Health Assessment and Consultation, ATSDR, has reviewed this public health consultation and concurs with the findings.



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Team Leader, Cooperative Agreement Team, CAPEB, DHAC, ATSDR

# Attachment 1

**Carl Herbrandson (MDH)**

**From:** Magee-Hill, Heather [Heather.Magee-Hill@state.mn.us]

**Sent:** Thursday, July 16, 2009 12:21 PM

**To:** Magee-Hill, Heather; Bill Desmond; Carl Herbrandson (MDH); Luke N. Taylor; pdswpao@rea-alp.com; ehoefs@wenck.com; Libbie L. Henderson

**Cc:** Braaten, Bruce

**Subject:** RE: Summary of 7/15/09 conference call and requested attachments

**Attachments:** comparison IRAP recp air parameters.xls

I tried to send the project files but they say they exceed the mailbox size of the recipients so here is the excel sheet I said I would send and I will work on getting the project files on to our ftp site. Mean while my previous e-mail gave very specific instructions on how to replicate them so I hope that any delay in getting the actual project files to you won't slow down progress.

Heather Magee-Hill  
Risk Assessment  
Minnesota Pollution Control Agency  
520 Lafayette Road North  
St. Paul, MN 55155-4194  
**As of 11/20/08 use 651-757-2545**  
Fax: 651-297-7709  
[Heather.Magee-Hill@state.mn.us](mailto:Heather.Magee-Hill@state.mn.us)

-----Original Message-----

**From:** Magee-Hill, Heather

**Sent:** Thursday, July 16, 2009 12:20 PM

**To:** 'Bill Desmond'; Carl Herbrandson; 'Luke N. Taylor'; 'pdswpao@rea-alp.com'; 'ehoefs@wenck.com'; 'Libbie L. Henderson'

**Cc:** Braaten, Bruce; Schutt, Carolina; Kain, Kevin; Burman, Shelley

**Subject:** Summary of 7/15/09 conference call and requested attachments

Greetings,

Attached are the excel files, and tables that I used on Tuesday to talk with the MPCA team about next steps, which I said I would send you. Below is a summary of the risk assessment portion of yesterdays call and a summary of the results from the attached project files. I tried to e-mail the zipped project files but couldn't so I will try to send them separately.

On the 7/15/09 weekly call we went through progress on previous comments based on a previous e-mail (italicized):

1. *I asked for an updated Hg-01 Form as a reference for the MMREM and TMDL work, which the consulting team has submitted a draft to Anne Jackson, I and Pope Douglas consultants said we would ask Anne about setting up a separate call to review it.*
2. Ruth's question about the MMREM modeling, still needs a response from Pope Douglas.
3. *I asked for a list of potentially emitted chemicals lacking any inhalation health benchmarks. This is a standard tab on our RASS. I did not understand the reference to "see table 8.3 in report" in the original set of RASS's submitted. Which everyone mutually agreed will be the same as the one used for Olmstead.*
4. *I asked about the reference to Appendix I and explained I couldn't find it. The team explained that it was not included. Which was submitted and clarified.*
5. *I asked more about how the non NO2 acute risks were calculated and if there were any additional files or spreadsheets which would enable me to verify the acute emissions used and the acute AIEC values used. The Pope Douglas team consulting team is checking into it. Which was submitted with corrections on 7/14/09.*

# Attachment 1

6. I asked for confirmation that sulfuric acid risks were calculated using the tox value from the RASS, even though it was not included in the COPC database that the MPCA sent, which the consulting team sufficiently confirmed they added. No further action was needed.

7. I asked for more information regarding the use of the 20 year operating scenario, which the consulting team will look into. Pope Douglas will submit a more detailed written response than the verbal response given on 7/7/09, which indicated that the 20 year operating scenario was chosen to be consistent with the depreciation expected for the facility, since no more pertinent rationale was found supporting the selection of the 30 year default.

8. I asked for more information about when plume depletion was used, which Stephanie has already answered in an e-mail.

We then discussed new comments from the MPCA:

-Further investigation into the life span of similar facilities in the state indicates the use of the default of 30 years of operation is more defensible than 20 years (based on financial depreciation). Pope Douglas will submit a more detailed explanation about the choice of the 20 year operating scenario.

-The depth of water column in the IRAP projects submitted are not the ones previously recommended. For Lake Burgen it should be more like 9.45 meters (31 ft) than the 24.3 meters (79.7 ft) which was used. For Lake Victoria it should be more like 9.75 meters (32 ft) instead of 30.2 meters (99 ft) which was used.

-There is a question about whether the receptor used for maximally impacted individual captures the highest expected chronic risks.

Below is the comparison that Bill asked for (larger values highlighted). I also attached the excel sheet it is derived from. These are directly exported from the project files I have attached. The risks from the Resident Adult in the 50 ug/dscm scenario with all units from the receptor I added are slightly higher (Total HQ 1.408 cancer 9.88E-6) than at the D-OffSiteMEI (1.358 cancer 9.407E-6). I added the receptor in order to see how much a difference in risk would be, after looking at the OTHERS (Other, all, annual.plt) plot file and seeing that the D-OffSiteMEI receptor didn't seem to be in the area of the highest annual concentrations. Overall this does not make a real difference in the risk estimates but the discussion, exercise Bill suggested, and further inspection of the plot files has clarified for me how the MEI receptor was chosen. Thank you.

# Attachment 1

Acute & Residential Receptor - Off-Site MEI			highest receptor from annual all units			mpca highest chronic		
RECEPTOR :	D_OffSiteMEI	UTM X:	315,941.20	UTM Y:	5,082,262.70	RECEPTOR :	UTM X:	UTM Y:
SOURCE:	AIR PARAMETER DESCRIPTION	VALUE	SV015	SV001	sum	sum	SV001	SV015
Hourly air concentration - particle phase	chp	ug-s/g-m <sup>3</sup>	18.00263	76.0026	94.00523	72.30055	66.91928	5.38127
Hourly air concentration - particle bound	chp_pb	ug-s/g-m <sup>3</sup>	17.90836	75.9399	93.84826	72.28875	66.92163	5.36712
Hourly air concentration - vapor phase	chv	ug-s/g-m <sup>3</sup>	17.90388	75.93575	93.83963	72.28557	66.91928	5.36629
Hourly air concentration - vapor phase hg	chv_hg	ug-s/g-m <sup>3</sup>	0	0	0	0	0	0
Air concentration - particle phase	cyp	ug-s/g-m <sup>3</sup>	0.4698	3.5185	3.9883	3.91944	3.90074	0.0187
Air concentration - particle bound	cyp_pb	ug-s/g-m <sup>3</sup>	0.46634	3.51515	3.98149	3.91943	3.90084	0.01859
Air concentration - vapor phase	cyv	ug-s/g-m <sup>3</sup>	0.46618	3.51482	3.981	3.91928	3.9007	0.01858
Air concentration - vapor phase hg	cyv_hg	ug-s/g-m <sup>3</sup>	0	0	0	0	0	0
Dry deposition - particle phase	dydp	s/m <sup>2</sup> year	0.17151	0.69369	0.8652	0.79362	0.78347	0.01015
Dry deposition - particle bound	dydp_pb	s/m <sup>2</sup> year	0.01176	0.05514	0.0669	0.06201	0.06133	0.00068
Dry deposition - vapor phase	dydv	s/m <sup>2</sup> year	0.01813	0.08891	0.10704	0.13244	0.13195	0.00049
Dry deposition - vapor phase hg	dydv_hg	s/m <sup>2</sup> year	0	0	0	0	0	0
Wet deposition - particle phase	dywp	s/m <sup>2</sup> year	0.01476	0.01408	0.02884	0.02755	0.00456	0.02299
Wet deposition - particle bound	dywp_pb	s/m <sup>2</sup> year	0.00062	0.00059	0.00121	0.00115	0.00019	0.00096
Wet deposition - vapor phase	dywv	s/m <sup>2</sup> year	0.00153	0.00138	0.00291	0.00265	0.00023	0.00242
Wet deposition - vapor phase hg	dywv_hg	s/m <sup>2</sup> year	0	0	0	0	0	0

Attached are the results I generated in order to get a feel for what the risks might be if my previous comments were included in the project files e-mailed me. While these changes do not change the overall message of the draft risk report, they do result in higher chronic risks estimates. Based on both the draft submittal and these calculations the overall potential risks from the addition of unit 3, **are the same or lower**, with units 1 & 2 having a 50 ug/dscm limit for dioxins/furans and unit 1 & 2 and unit 3 at 60 ug/dscm total, than operating under currently permitted conditions (if the stack were raised), except for acute risks which are estimated to potentially increase by 10%. Depending on the location of the receptor and exposure scenario assumptions, estimated risks after the modification for the entire facility are both above and below risk thresholds. These estimates are similar but not exactly the same as the preliminary risk results I shared with the team (attached) in order to get a sense for possible next steps to move the project forward.

I suggested that Pope Douglas schedule a call with Bruce Braaton, Carolina Schutt and possibly others, to discuss the possibility for reducing potential risks at all locations by taking lower limits for pollutants, such as NOx (which is an acute risk driver), Hg (which is a risk driver for the fish pathway), lead (which is a risk driver for ingestion) and dioxins/furans (which is a cancer risk driver). Looking into the best available control technology for sulfuric and hydrochloric acid (both risk drivers for chronic inhalation risks) controls is another possibility to discuss. I also indicated



# Attachment 1

on the call that I did not expect to have any more comments on the draft submittal and that Pope Douglas could go ahead and submit a finalized version.

Thus the following results and project files were generated by:

1. Unzipping the project folders PopeDouglas09May50.irp and PopeDouglas09May125.irp.
2. Changing the Source Specific parameter time period over which deposition occurs from 20 years to the default of 30 years.
3. Changing the Water body Site Parameter of Depth of water column to 9.45 meters for Lake Burgen and 9.75 meters for Lake Victoria.
4. Adding a receptor at UTM X 315,926.41, UTM Y 5,082,233.00.
4. As we had discussed previously since the MPCA COPC database I sent you was missing sulfuric acid I added sulfuric acid to the user COPC database (using MW=98.08 and RfC=0.001) and emissions of 0.157 g/sec so that I didn't have to do the calculation separately.
5. I changed the Hg emissions to 1.86E-4 (which was done before I received your Hg updates so I haven't compared these emissions to the later ones you submitted). This Hg emission is straight from the limit with no adjustments for speciation. As indicated in my original (7/16/08) e-mail because of the complication with Hg wizard "If mercury is not a risk driver from inhalation it does not have to be included in the IRAP analysis." Since it is not a risk driver in inhalation I don't believe differences in speciation will make a real difference in the risk assessment.
6. Changing the activation and exposures of different receptors.

Please note that the fisher risks in the spreadsheet add the fish pathway risks from the representative "fisher receptors" to total residential risks at the residential receptors summarized above them in the table.

In order to answer the question "What are risks before modification?" I took the "PopeDouglas09May125" project made the above changes and deactivated SV015 and summarized them first for comparison. While this is not a true picture of the before modification risks, because this models the proposed higher stack, it does demonstrate the point that even with unit 3 because of the lower dioxin/furan emissions limits, the potential cancer risks decrease because of this expansion.

If you have any difficulties replicating these risks please notify me. Thanks.  
It might be useful in the final submittal to summarize the reports in a similar format.

- a. **Currently Permitted Operating Conditions (Unit 1 & Unit 2, but with a higher stack) Worst Case (Potential To Emit at 125 ng/dscm dioxin limit, at zoned maximally impacted locations, at worst case exposures like subsistence fishing and milk consumption from farming, for 30 years of operation). All risks are for adults except where otherwise indicated.**

# Attachment 1

Refined Risk Assessment							
Exposure	Total Inhalation Hazard Indices and Cancer Risks			Total Indirect Pathway Screening Hazard Indices and Cancer Risks		Total Multipathway Hazard Indices and Cancer Risks	
Receptor	Acute [1]	Chronic Noncancer [1]	Cancer [2]	Chronic Noncancer [1]	Cancer [2]	Chronic Noncancer [1]	Cancer [2]
Residential							
North side Property Boundary Highest 1 hour MEI for Residential Zoning	1.0	1.1	1.3E-5	0.7 child 0.3 adult	0.7E-5	1.8 child 1.4 adult	2.0E-5
Farmer							
Zoned Agricultural Maximally Exposed Individual		<0.1	0.1E-5	0.1 child <0.1 adult	5.7E-5 (milk) 1.6E-5 (beef)	0.2 child 0.1 adult	7.6E-5 (with milk)
Fisher							
-					-		3.2E-5

- b. Current Permitted Operating Conditions (Unit 1 & 2, but with a higher stack) at current maximally impacted residential receptors, assuming current farming conditions, and recreational fishing, 30 years of operation. All risks are for adults except where otherwise indicated.

Refined Risk Assessment							
Exposure	Total Inhalation Hazard Indices and Cancer Risks			Total Indirect Pathway Screening Hazard Indices and Cancer Risks		Total Multipathway Hazard Indices and Cancer Risks	
Receptor	Acute [1]	Chronic Noncancer [1]	Cancer [2]	Chronic Noncancer [1]	Cancer [2]	Chronic Noncancer [1]	Cancer [2]
Residential							
Bridgewater Estates Apartments maximally impacted exposed current residence	0.2	0.2	0.2E-5	0.1 child <0.1 adult	0.1E-5	0.3 child 0.2 adult	0.3 E-5
Cattle Farm 1 (No milk production)		<0.1	<0.1E-5	<0.1	0.9E-5 (beef)	0.1 child <0.1 adult	1.1E-5 (no milk)
Recreational Fisher (Lake Burgen)				<0.1	0.3E-5	0.3 child and adult	0.6E-5

- c. Worst case **Total facility After Modification** (potential to emit 50 ug/dscm for unit 1 & 2 and 13 ug/dscm for unit 3, assuming maximum exposures such as subsistence farming

# Attachment 1

and milk consumption, at maximally impacted zoned locations and 30 years of operation). All risks are for adults except where otherwise indicated.

Refined Risk Assessment							
Exposure	Total Inhalation Hazard Indices and Cancer Risks			Total Indirect Pathway Screening Hazard Indices and Cancer Risks		Total Multipathway Hazard Indices and Cancer Risks	
Receptor	Acute [1]	Chronic Noncancer [1]	Cancer [2]	Chronic Noncancer [1]	Cancer [2]	Chronic Noncancer [1]	Cancer [2]
Residential							
North side Property Boundary Highest annual MEI or Highest hourly MEI for Residential Zoning	1.1	1.1	0.6E-5	0.7 child 0.3 adult	0.4E-5	1.8 child 1.4 adult	1.0E-5
Farmer							
Zoned Agricultural Maximally Exposed Individual		<0.1	<0.1E-5	0.1 (milk) 0.1 (beef)	2.9E-5 (milk) 0.8E-5 (beef)		3.9E-5 (with milk)
Fisher							
Subsistence Fisher MEI Residential Zoning (Lake Burgen)					1.7E-5 (fish)	2.2 child 2.0 adult	2.7E-5

d. After Modification at most impacted current residence and farmer under current farming conditions (no milk production) and recreational fishing, 30 years of operation. All risks are for adults except where otherwise indicated.

Refined Risk Assessment							
Exposure	Total Inhalation Hazard Indices and Cancer Risks			Total Indirect Pathway Screening Hazard Indices and Cancer Risks		Total Multipathway Hazard Indices and Cancer Risks	
Receptor	Acute [1]	Chronic Noncancer [1]	Cancer [2]	Chronic Noncancer [1]	Cancer [2]	Chronic Noncancer [1]	Cancer [2]
Residential							
Bridgewater Estates Apartments maximally impacted exposed current resident	0.4 (0.3 NO2)	0.3 child and adult	0.1E-5	0.2 child <0.1 adult	0.1E-5		0.2E-5
Farmer							
Cattle Farm 1 (No milk production)		<0.1	<0.1E-5	<0.1	0.5E-5 (beef)	0.1 child and adult	0.7E-5 (without milk)
				<0.1 adult (fish) 0.1 adult (fish)			
						0.4 child 0.4 adult	0.7E-5
						0.9 child 0.9 adult	1.9E-5
				0.4 child (fish) 0.6 adult (fish)			

# Attachment 1

## SUMMARY OF MMREM RESULTS

Emissions Scenario	Water Body	Subsistence Fisher <sup>1</sup> (consumption of 0.142 kg/day) Hazard Quotient		Recreational Fisher <sup>2</sup> (consumption of 0.03 kg/day) Hazard Quotient	
		Ambient	Incremental	Ambient	Incremental
Potential to Emit, Units 1 and 2 (existing with a limit of 60 ug/dscm)	Lake Burgen	9.4	2.5	2.0	0.5
	Lake Victoria	9.4	2.0	2.0	0.4
Future Potential to Emit, All Units (same total facility limit of 60 ug/dscm )	Lake Burgen	9.4	2.3	2.0	0.5
	Lake Victoria	9.4	1.8	2.0	0.4
Potential to Emit, Unit 3 (expansion project 30 ug/dscm for the unit)	Lake Burgen	9.4	1.0	2.0	0.2
	Lake Victoria	9.4	0.8	2.0	0.2
Future-Projected Actual Emissions, All Units	Lake Burgen	9.4	0.1	2.0	<0.1
	Lake Victoria	9.4	<0.1	2.0	<0.1

Notes:

<sup>1</sup> Roughly equivalent to 2.2 pounds of fish consumed per week, 52 weeks per year, from the listed water body.

<sup>2</sup> Roughly equivalent to 0.5 pounds of fish consumed per week, 52 weeks per year, from the listed water body.

Discussed MDH comments (italicized) and MPCA responses:

*1. reporting childhood cancer risk as < adult cancer risk (likely a result of multiplying by 16/70ths vs 32/70ths - or whatever is plugged into the IRAP) - I'm proposing that P-D (IRAP) assume adulthood follows childhood...*

*2. In addition, P-D (IRAP) should be using the EPA early-life susceptibility methodology for childhood cancer (except dioxin)*

Childhood risks are not included in the AERA screening analysis (using the RASS) and have historically only been included in more detailed risk assessments when they have been calculated using IRAP and exceed the adult risks, like in the case of non-cancer risks. Since the use of IRAP was approved during the development of this protocol and in order to be consistent with how previous projects have reported risks, the MPCA recommends continuing discussion about the best way to calculate childhood cancer risks with MDH but from an overall guidance improvement instead of including it as part of this project.



# Attachment 1

Similarly adding childhood cancer risks to adult risks has neither been done as part of the AERA screening analysis or in past more detailed risk assessments and the MPCA recommends continuing discussing this with MDH as an overall guidance improvement instead of including it as part of this project.

Preliminary calculations, such as the ones you suggested, indicate that for this project, with the proposed modification, at the most impacted area zoned for agriculture, assuming with worst case assumptions about exposure (including milk consumption), the child farmer cancer risk estimate would be more like  $1.77E-5$  instead of  $1.00E-5$  and thus the entire lifetime adult farmer cancer risk estimate (including the child risk) would be more like  $6.25E-5$  instead of  $4.75E-5$ . Thus the MPCA recommends noting that these are two ways where this risk assessment may under predict risks.

*3. P-D needs to characterize Pb inhalation hazard - I recommend using NAAQS minus(-) monitored levels or better (?plus an additional margin for future, unrelated, development?)*

The MPCA agrees that risks from Pb inhalation should be characterized and has asked Pope Douglas to look more at how modeled monthly lead concentrations from their facility compare to the November 2008 lead NAAQS standard.

*4. cPAH should address extended list - either w/data or with some source-specific fingerprint data (may not be important at this site, but gathering data for a MPCA/MDH source/media dBase is important for future projects.*

The MPCA agrees that more data about the extended list of cPAH is important and is considering how best to gather the data (including investigating the best stack testing methodology) but the MPCA knows of no applicable cPAH extended list data that could currently be used in this risk assessment. Instead the MPCA recommends noting this as an area where this risk assessment may under predict risks.

*5. soil risk/hazard from IRAP is >0 for "current risk". If the project is new this should be 0, but the "future risk" should be >0. Is there a justification for reporting risk this way when the project has a "future risk" category? Having 0 for "current" and >0 for "future", at a known time point, allows one to estimate the risk at different times in the future. Soil (and sediment for some projects) is the one media where concentrations will be increasing over time.*

The MPCA agrees that the terminology "current operating conditions" needs more explanation in order to not be interpreted as meaning "current risks". Further clarification is need to explain that all of the risk estimates in this risk assessment refer to different scenarios of potential future risks.

Please notify me of any problems in opening any of the attached files and feel free to contact me with any questions or concerns

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# Attachment 2



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## TECHNICAL MEMORANDUM

To: Heather Magee-Hill  
Minnesota Pollution Control Agency

From: Stephanie Kuphal, Ed Hoefs, Libbie Henderson  
Wenck Associates, Inc.

Date: July 21, 2009

Subject: Lead Ambient Air Dispersion Modeling  
Unit 3 Project  
Pope/Douglas Solid Waste Management Facility

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Air dispersion modeling was completed for the Pope/Douglas facility for lead air emissions. This air dispersion modeling was requested by MPCA to support the Air Emissions Risk Analysis and Environmental Assessment Worksheet for the Unit 3 project. Predicted lead concentrations were compared to the lead National Ambient Air Quality Standard. Modeling input and output files are attached.

With the exception of the pollutant, emission rates, and the applicable averaging period, all modeling options and modeling input are consistent with the earlier criteria pollutant modeling completed for Pope/Douglas on the Unit 3 project. Please see the Criteria Pollutant Modeling Analysis, Pope/Douglas Solid Waste Management Facility dated April 2009 for additional description. Deposition and plume depletion was not included in the lead air dispersion modeling. Lead emission rates are shown below in Table 1.

**Table 1.**  
**Modeled Pope/Douglas Lead Potential Air Emissions**

<b>Stack Number</b>	<b>Stack Description</b>	<b>Potential Lead Emissions (ton/yr)</b>	<b>Modeled Lead Emissions (g/s)</b>	<b>Basis for Emissions</b>
SV001	Units 1 and 2 Stack	$3.45 \times 10^{-1}$	$9.929 \times 10^{-3}$	120 ton/day and 40 CFR 62 Subp. JJJ limit
SV004	Auxiliary Boiler Stack	$7.515 \times 10^{-6}$	$2.162 \times 10^{-7}$	3.5 MMBtu/hr and AP-42 emission factor
SV015	Unit 3 Stack	$4.31 \times 10^{-2}$	$1.241 \times 10^{-3}$	120 ton/day and 40 CFR 60 Supb. AAAA limit

# Attachment 2

Memorandum to: Heather Magee-Hill  
Minnesota Pollution Control Agency  
July 21, 2009  
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The monthly averaging period was selected. The lead National Ambient Air Quality Standard is on a rolling, 3-month average basis, with no exceedances allowed. The high monthly average is a conservative estimate of rolling 3-month concentrations. In addition, a smaller receptor grid was modeled and all concentrations were saved in a "MAX" output file. A very low threshold concentration was specified for the MAX file to save all monthly modeled concentrations. The max file was pulled into a spreadsheet and sorted by receptor and month. Rolling 3-month averages were calculated. The spreadsheet calculations avoid the need to include 58 separate modeling runs to identify the rolling average.

A lead background concentration of 0.01 ug/m<sup>3</sup> was selected. This value was identified from MPCA's Draft Annual Air Monitoring Network Plan for 2010. As reported by MPCA, ambient lead concentrations for 2006 through 2008 were below 0.01 ug/m<sup>3</sup> for the "majority of sites". The two ambient monitoring sites located next to large lead stationary sources were not considered representative of background for Pope/Douglas and were not considered. The remainder of the state is below this 0.01 ug/m<sup>3</sup> value.

Estimated lead ambient concentrations surrounding the Pope/Douglas facility are shown in Table 2 below in comparison to the lead National Ambient Air Quality Standard.

**Table 2.**  
**Pope/Douglas Estimated Lead Ambient Air Concentrations**

<b>Source</b>	<b>High Monthly Lead Concentration (ug/m<sup>3</sup>)</b>	<b>Rolling, 3-Month High Lead Concentration* (ug/m<sup>3</sup>)</b>
Pope/Douglas Facility	0.091	0.056
Background	---	0.01
<b>Total Ambient Impact</b>	---	0.066
<b>National Ambient Air Quality Standard</b>	---	0.15

\* The rolling, 3-month high lead concentration was calculated in a spreadsheet from monthly concentrations saved to a max file. The high monthly lead concentration is obtained directly from the modeling output file.

Pope/Douglas meets the National Ambient Air Quality Standard for lead at all areas surrounding the facility.