DEPARTMENT OF
ECOLOGY
State of Washington

Technical Support Document for Second Tier Review

241-SY, 241-AP, and 241-AY/AZ Tank Farm Ventilation System Upgrades at the Hanford Site, Benton County, Washington

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TABLE OF CONTENTS

1. EXECUTIVE SUMMARY 1
   1.1. Proposal Summary 1
   1.2. Health Impacts Evaluation 1
   1.3. Conclusions and Recommendation 2
2. PERMITTING PROCESS OVERVIEW 2
   2.1. The Regulatory Process 2
      2.1.1. The Three Tiers of Permitting Toxic Air Pollutants 2
      2.1.2. Second Tier Review Processing Requirements 3
3. FACILITY INFORMATION 5
   3.1. Facility Location 5
   3.2. Permitting History 5
   3.3. The Proposed Project 6
4. POLLUTANT SCREENING 10
   4.1. Emissions 10
   4.2. Best Available Control Technology for Toxics (tBACT) 12
   4.3. Air Dispersion Modeling 13
   4.4. Points of Compliance 13
   4.5. Maximum TAP Concentrations 17
   4.6. Pollutants Subject to Second Tier Review 17
5. HEALTH IMPACT ASSESSMENT 17
   5.1. Introduction 17
   5.2. Hazard Identification 18
      5.2.1. DMM 21
      5.2.2. Hexavalent Chromium 21
      5.2.3. N-Nitrosodimethylamine 21
   5.3. Environmental Fate 22
   5.4. Exposure Assessment 22
      5.4.1. Multi-Route Exposures 22
      5.4.2. Identification of Exposed Populations 23
      5.4.3. Estimates of Exposure Durations of Identified Populations 25
      5.4.4. TAP Concentration Estimates 25
         5.4.4.1. Existing Background Levels 26

5.5. Exposure-Response Assessment 27
5.5.1. Risk-Based Dose Limit for Exposed Populations 27
5.6. Risk Characterization 29
5.6.1. Hazard Quotient for Total DMM Dose 29
5.7. Uncertainty Characterization 29
5.7.1. Emissions Uncertainty and Variability 30
5.7.2. TAP Concentration Modeling Uncertainty 30
5.7.3. Background TAP Concentration Estimates Uncertainties 31
5.7.4. Exposure Uncertainty 31
5.7.5. Toxicity Uncertainty 31
6. CONCLUSION 32
7. LIST OF ACRONYMS AND ABBREVIATIONS 33

LIST OF FIGURES

Figure 1. Map showing the Hanford Site 200 W Area, 200 E Area, and surroundings 7
Figure 2. A schematic of a typical DST at the Hanford Site in WA..... 8
Figure 3. A conceptual drawing of a DST exhauster at the Hanford Site in WA..... 9
Figure 4. Twenty-four hour time-weighted average DMM concentrations attributable to the new DST ventilation systems 14
Figure 5. Modeled peak 24-hour average DMM concentrations in the vicinity of Hanford..... 24

LIST OF TABLES

Table 1. TAPs Detected in Tank Headspaces..... 10
Table 2. Project Farm Ventilation Rate 13
Table 3. TAP Emission Rates and Comparisons to the WAC 173-460-150 De Minimis, SQER, and ASIL Levels 15
Table 4. Potential Adverse Effects of TAPs to be Emitted in Amounts Above SQERs 18
Table 5. Specific Pathways to be Analyzed for Each Multi-Pathway Substance..... 23
Table 6. Maximum Off-Site DMM Atmospheric Concentrations and Deposition Rates Attributable to the Tank Farm Exhauster Emissions 26
Table 7. Summary of how the Uncertainty Affects the Quantitative Estimate of Risks or Hazards 30

1. EXECUTIVE SUMMARY

1.1. Proposal Summary

The U.S. Department of Energy (DOE), through their tank operations contractor, Washington River Protection Solutions, LLC (WRPS), has proposed to upgrade primary tank ventilation systems for the 241-AP, 241-SY, and 241-AY/AZ radioactive waste tank farms. The tank farms are located at the 200 West and 200 East areas of the Hanford Site in Benton County, Washington. The ventilation system upgrades will replace the existing exhaust systems and will support Waste Feed Delivery (WFD) from the 200 area tank farms to the Waste Treatment and Immobilization Plant (WTP). The WTP planned to start operations in 2019 and finish in 2049. The proposed tank farm ventilation system upgrades are being performed as part of the overall plan to treat stored radiological and chemical waste from historical plutonium production. The tank farm ventilation systems will be constructed, installed, tested, and fully operational in three phases, beginning with 241-AP, to be followed by 241-SY and 241-AY/AZ, respectively.

Operation of the new ventilation systems (exhausters) will support the storage, treatment, retrieval, sampling, transfers, and mixing of waste in the 241-SY, 241-AP, and 241-AY/AZ tank farms. The new exhausters will have increased flow rates to support WFD operations. Two mixer pumps will be installed to up to two tanks in each farm to make the waste homogeneous for sampling and processing at the waste treatment plant. The heat generated during mixing and by the pumps will be removed from the tanks using the new ventilation systems.

During the WFD process, double-shell tanks (DSTs) will be mixed to transfer waste between tank farms to be blended and to provide feed to the WTP. The mixing and transfer process is expected to take approximately 15 to 20 days to complete. For the rest of the time, the mixer pumps will not operate or will be removed, the waste will be quiescent, and the exhausters will run at their normal flow rates.

Potential emissions of dimethyl mercury (DMM) from all three proposed exhausters will exceed the applicable acceptable source impact level (ASIL) promulgated in Washington Administrative Code (WAC) 173-460-150. Therefore, in accordance with WAC 173-460-090, DOE petitioned for second tier review of DMM emissions expected from the project.

1.2. Health Impacts Evaluation

DOE retained WRPS to complete the second tier petition on their behalf. Review of data in the Health Impact Assessment (HIA) conducted by WRPS and of other data indicates there is a limited amount of data on background DMM concentrations. The available data show DMM emissions from the tank exhausters could result in no more than a 2.5 percent increase of DMM concentrations above the existing background airborne concentration at the point of maximum off-site impact located on Highway 240. Due to the conservative assumptions about emissions, actual increase in airborne concentrations is likely to be far lower than 2.5 percent.

As required by Chapter 173-460 WAC, DOE/WRPS evaluated exposure through inhalation and ingestion of DMM from the exhausters. They estimated the tank exhausters' total attributable DMM dose to a hypothetical maximally exposed person as 6.9E-07 micrograms per kilogram (μg/kg) of body weight per day. At this exposure, the hazard quotient (HQ) is 6.79E-6, which is much less than one.

Because accidental exposures to liquid DMM have previously caused human fatalities, the Washington State Department of Ecology (Ecology) chose an ASIL for DMM so low as to force extra caution in review of all DMM air emission permit applications. However, as further explained below, Ecology's technical review of potential DMM emissions from the proposed 241-SY, 241-AP, and 241-AY/AZ Tank Farm ventilation system upgrades shows that public exposures to DMM emissions from the ventilation system upgrades will be trivial. Specifically, the HQ of the most sensitive effect—damage to the fetal central nervous system—is well below one, which indicates the potential for effect is extremely low.

Although there is no numerically defined acceptable limit of noncancer adverse health risks in Chapter 173-460 WAC, the amount of increased neurological impairment risk is far less than the HQ of one. This demonstrates that adverse noncancer effects are not expected from the project. Therefore, the project is approvable under WAC 173-460-090(7).

1.3. Conclusions and Recommendation

Based upon the available literature, very low emissions, and resulting ambient concentrations, the emissions of DMM from the proposed new ventilation systems should not pose a risk to the public. Ecology's analysis also indicates no potential for significant emissions of any other neurotoxic toxic air pollutants (TAPs). Based on review of the technical analysis provided by DOE and WRPS, and provided the exhausters are operated as proposed, the additional health risks attributable to this project will be permissible under Chapter 173-460 WAC. The remainder of this document describes the technical review performed by Ecology.

2. PERMITTING PROCESS OVERVIEW

2.1. The Regulatory Process

The requirements for performing a toxics screening are established in Chapter 173-460 WAC. This regulation requires a review of any increase in regulated toxic emissions for all new or modified stationary sources in the state of Washington.

2.1.1. The Three Tiers of Permitting Toxic Air Pollutants

The objectives of permitting TAPs are to establish the systematic control of new sources emitting TAPs in order to prevent air pollution, reduce emissions to the extent reasonably possible, and maintain such levels of air quality as will protect human health and safety.

There are three levels of review when processing a new or modified emissions unit emitting TAPs: (1) first tier review (toxics screening), (2) second tier review (health impact assessment), and (3) third tier review (risk management decision).

All projects are required to undergo a toxics screening (first tier review) as required by WAC 173-460-040. There are two ways to perform a first tier review. If proposed emissions are below the Small Quantity Emission Rates (SQERs) found in WAC 173-460-150, no further analysis is required. If emissions are greater than the SQERs, those emissions must be modeled and the resultant ambient concentration compared against the appropriate ASIL. If the ambient concentration is below the ASIL, then no further analysis is required.

A second tier review, required by WAC 173-460-090, is a site-specific HIA. The objective of a second tier review is to quantify the increase in lifetime cancer risk for persons exposed to the increased concentration of any carcinogenic TAP and to quantify other increased health hazards from any TAP in ambient air that would result from a proposed project. Once quantified, the cancer risk is compared to the maximum risk allowed under a second tier review, which is one in one hundred thousand, and the concentration of any TAP emission from the proposed project is compared to a noncancer health risk-based concentration value (RBC).

If the emission of a TAP results in additional cancer risk greater than one in one hundred thousand, or Ecology finds that other health hazards are not acceptable, an applicant may request Ecology perform a third tier review. A third tier review is a risk management decision made by the director of Ecology about whether or not the health risks posed by a project are acceptable. The decision is based on a determination that emissions will be maximally reduced through available preventive measures, assessment of environmental benefits, disclosure of risks at a public hearing, and related factors associated with the facility and the surrounding community.

The proposed 241-SY, 241-AP, and 241-AY/AZ Tank Farm ventilation system upgrades triggered second tier review because the project's exhausters will emit DMM at levels that exceed the DMM ASIL. It must be noted that unlike other TAPs listed in WAC 173-460-150, the ASILs for DMM and diethyl mercury are not based on toxicity data. These ASILs have no foundation in scientific principle. Instead, they were set at an implausibly low level to assure a second tier review would be performed for any emission from a source subject to Chapter 173-460 WAC.

2.1.2. Second Tier Review Processing Requirements

Processing requirements for second tier petitions are found in WAC 173-460-090(2). Ecology shall evaluate a source's second tier petition only if:

(i) The permitting authority submits to Ecology a preliminary order of approval that addresses all applicable new source review issues with the exception of the outcome of second tier review, State Environmental Policy Act review, public notification, and Prevention of Significant Deterioration (PSD) review (if applicable);

(ii) Emission controls contained in the preliminary approval order represent at least Best Available Control Technology for Toxics (tBACT);

(iii) The applicant has developed a HIA protocol that has been approved by Ecology;

(iv) The ambient impact of the emissions increase of each TAP that exceeds its ASIL has been quantified using refined air dispersion modeling techniques as approved in the HIA protocol; and

(v) The second tier petition contains a HIA conducted in accordance with the approved HIA protocol.

To date, communications between DOE/WRPS and Ecology include various documents and related meetings:

1. Ecology's Richland Field Office (RFO) submits a revised preliminary Notice of Construction (NOC) Order of Approval for the project (June 30, 2011).

2. RFO submits a preliminary NOC Order of Approval for the project (May 19, 2011).

3. Ecology deems second tier petition incomplete, pending receipt of a preliminary order of approval and review fees (March 18, 2011).

4. Complete second tier review petition received by Ecology from applicant (February 18, 2011).

5. Receipt—circa February 14, 2011—of the final electronic versions of the applicant's NOC application and second tier petition including:

    - Criteria & Toxics Air Emissions Notice of Construction for the Operation of the 241-AP, 241-SY, and 241-AY/AZ Tank Farm Ventilation System Upgrades

    - Second Tier Review Petition for the Operation of the 241-SY, 241-AP, and 241-AY/AZ Tank Farm Ventilation System Upgrades

6. DST Ventilation Systems Draft NOC review sent to applicant (December 14, 2010).

7. Completed reviews of the draft HIA (Clint Bowman) sent to applicant (December 8, 2010).

8. Receipt of the applicant's draft NOC application and second draft HIA application for the Hanford DST Ventilation System Upgrade (November 23, 2010).

9. Hanford Exhauster Permit Meeting in Lacey (June 18, 2010).

10. Receipt of the applicant's revised tBACT and source-term documents for the Hanford DST permit application (circa June 4, 2010).

11. Ecology sent review of applicant's draft HIA Protocol (circa December 14, 2009).

12. Draft HIA Protocol received from applicant (circa December 04, 2009).

13. First meeting with applicant/consultants for the Hanford Tier 2 (November 17, 2009).

Ecology considers the preliminary order of approval, as revised on June 30, 2011, to satisfy items (i) and (ii) above. Additionally, the communications between DOE/WRPS and Ecology include items (iii), (iv) and (v) above for the proposed project. The documents and electronic files submitted by DOE/WRPS as of February 14, 2011, contained sufficient information to perform a health impacts review in accordance with standard risk assessment procedures.

In summary, DOE/WRPS and RFO satisfied the second tier review requirements listed above. Thus, Ecology considers the second tier petition complete.

3. FACILITY INFORMATION

3.1. Facility Location

The 241-SY, 241-AP, and 241-AY/AZ DSTs farms are located northwest of Richland at:

U.S. Department of Energy, Office of River Protection
Hanford Site
200 East and West Area Tank Farms
Richland, WA 99352

The DSTs are located in the 200 East and West areas of the Hanford Site (see Figure 1).

The exact latitude and longitude coordinates of each set of tanks is provided in the petition documents.[1]

3.2. Permitting History

The permit issuance history for the 241-SY, 241-AP, and 241-AY/AZ ventilation systems is documented in the petition documents.[2]

[1] RPP-ENV-48231, Rev. 0, Table 1 on p. 3 (10/214)
[2] RPP-ENV-48231, Rev. 0, Section 1.3 on p. 2 (8/214)

3.3. The Proposed Project

As stated above, DOE/WRPS submitted a draft NOC application to RFO for the Hanford DST Ventilation System Upgrades on November 23, 2010, and the final "Criteria & Toxics Air Emissions Notice of Construction for the Operation of the 241-AP, 241-SY, and 241-AY/AZ Tank Farm Ventilation System Upgrades" circa February 14, 2011. The NOC application requests approval to install and operate new 241-SY, 241-AP, and 241-AY/AZ primary ventilation system exhausters. The new ventilation systems will operate for waste storage, treatment, retrieval, sampling, and transfers of the waste to the WTP. The existing exhausters will be upgraded with increased air flow rates because thermal hydraulic analysis of the WFD process determined that increased air flow rates are necessary to maintain the tanks within operating temperature limits.[3]

[3] The minimum treatment systems will include a demister, pre-heater, and two stages of HEPA filtration (each 99.97% control at 3 micrometer). Mists are low probability – but can be present in the ~1500 acfm ventilation. If they control the organic mercury compounds they will likely be doing that with doped granular activated carbon. Doug Hendrickson, e-mail message, October 21, 2009.

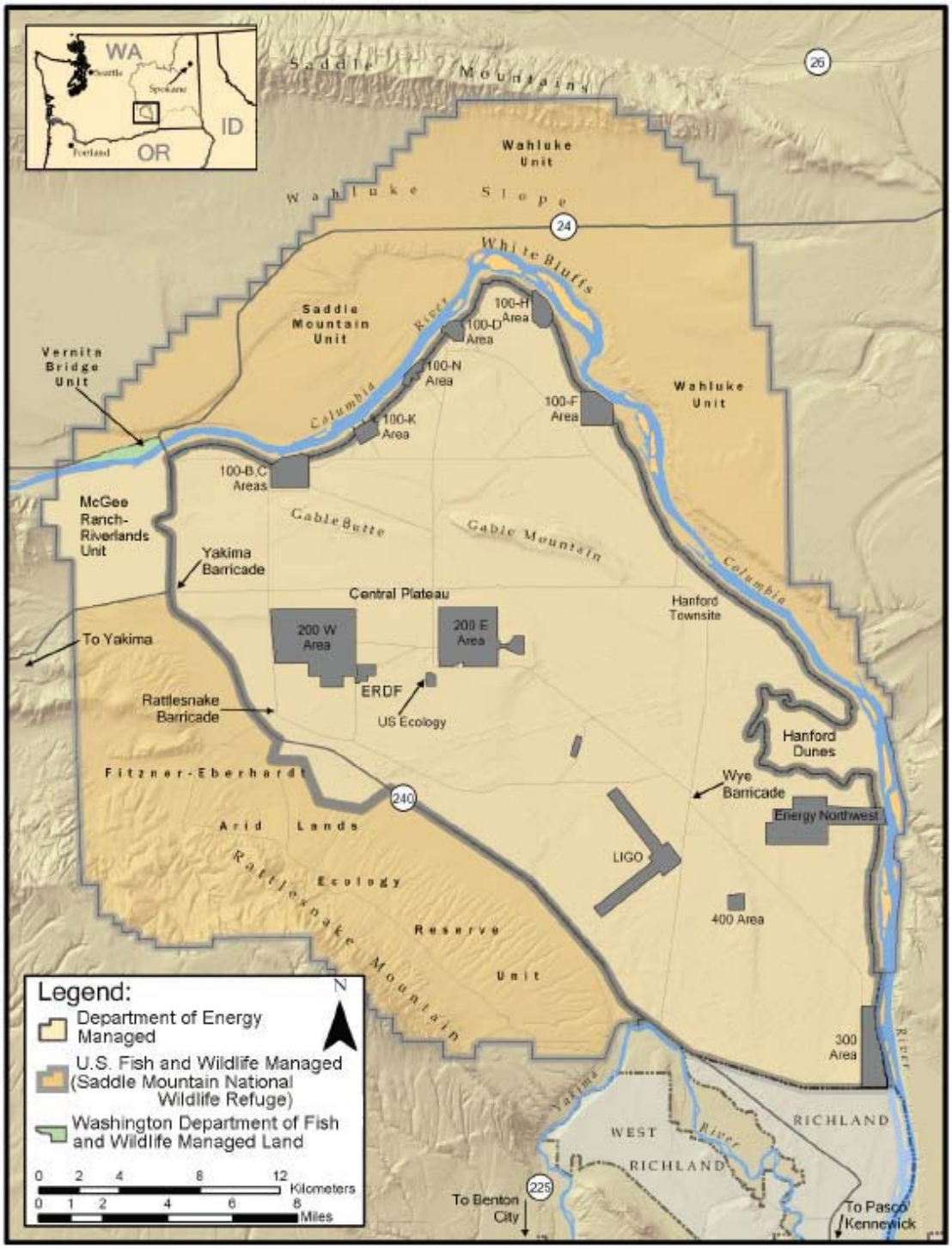


Figure 1. Map showing the Hanford Site 200 W Area, 200 E Area, and surroundings (Source: DOE/WRPS)

Future operations of the WTP (vitrification plant) requires storage and transfer of Hanford tank wastes from both double- and single-shell tanks. Current single-shell tank (SST) wastes are sludges and salt-cakes, having had the pumpable liquor removed. SST wastes will be sluiced or otherwise removed and transferred to DSTs. DOE proposes to upgrade the ventilation systems of two farms of DSTs to be able to assure confinement of gases and mists from the filling, pumping, and mixing of wastes within these DSTs. Each tank in these farms has a capacity of approximately 1.16 million gallons. Operation of the new ventilation systems will support the storage, treatment, retrieval, sampling, transfers, and mixing of the waste in the new 241-SY, 241-AP, and 241-AY/AZ exhausters. Figures 2 and 3 show a diagram of a typical DST and a DST exhauster, respectively.

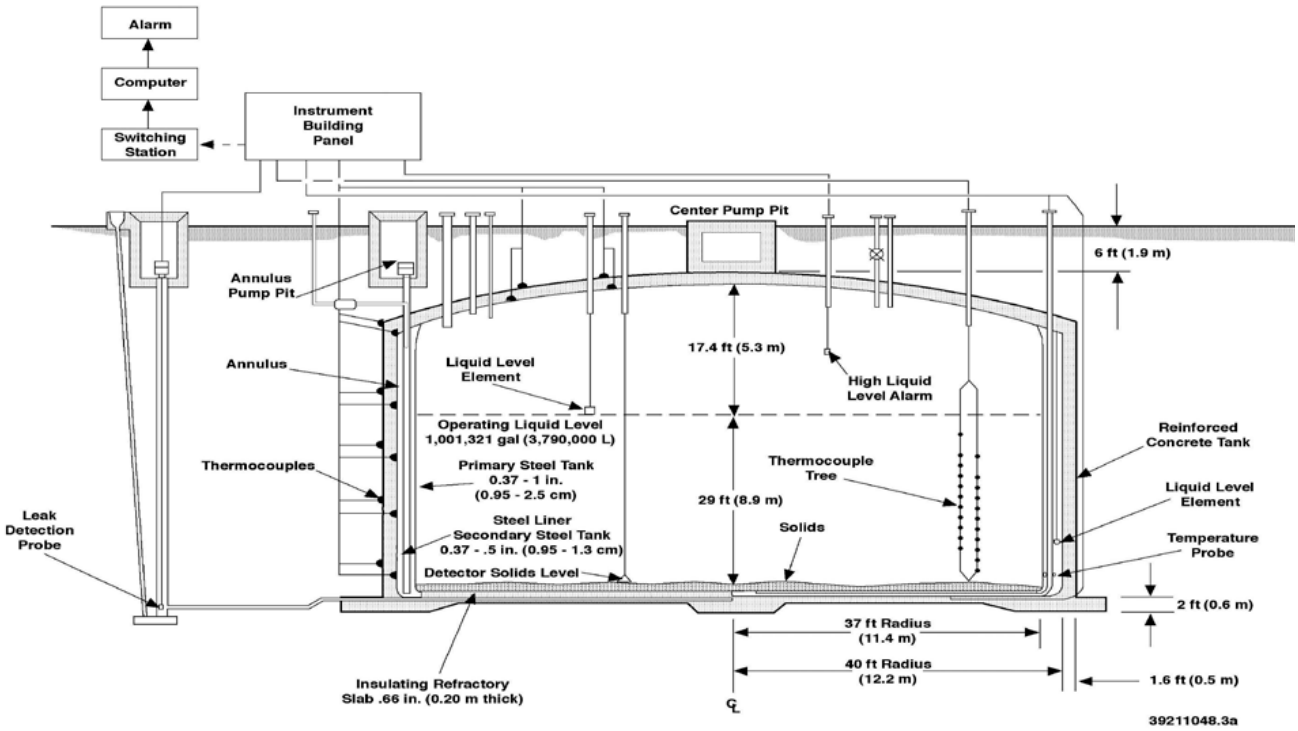


Figure 2. A schematic of a typical DST at the Hanford Site in WA

(Source: DOE/WRPS)

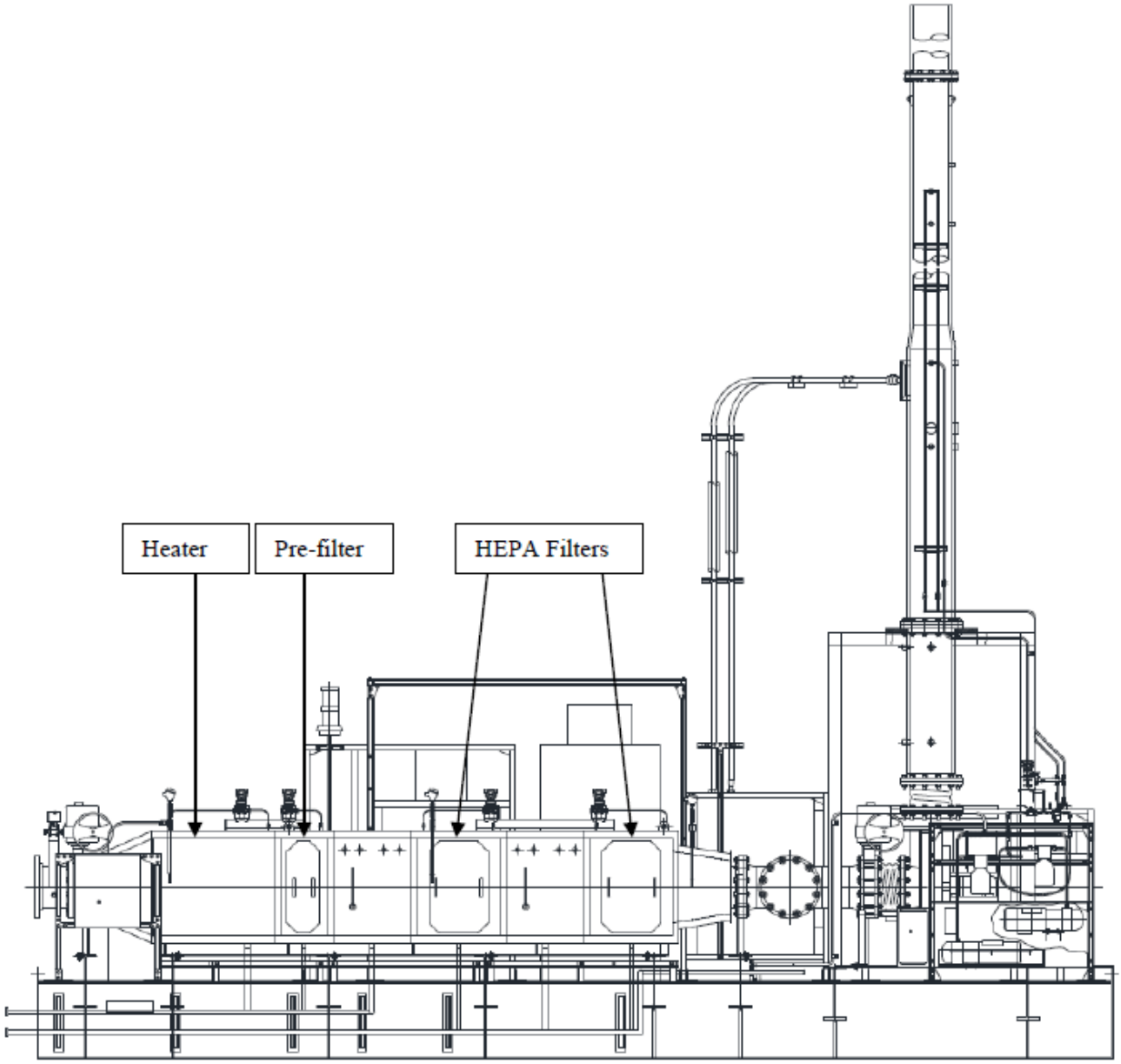


Figure 3. A conceptual drawing of a DST exhauster at the Hanford Site in WA

(Source: DOE/WRPS)

The exhausters are being upgraded with increased flow rates to handle the increased heat from mixer pumps that will be added to mix the tanks during waste transfers between tank farms and waste delivery to the WTP. The WTP is planned to start operations in 2019 and finish in 2049. Design of the 241-SY and 241-AP ventilation systems, as well as partial construction and procurement, is the result of funding made available by the American Recovery and Reinvestment Act.

The proposed ventilation system upgrades will support the tanks WFD mission as part of the overall plan to treat stored radiological and chemical waste from plutonium production. During

the WFD process, DSTs will be mixed to transfer waste between tank farms to be blended and to provide feed to the WTP. Mixing will require up to two mixing pumps in up to two tanks per tank farm to ensure that the waste is homogeneous for sampling and waste acceptance before transfers begin. Since the heat generated from the operation of the mixer pumps will increase the temperature of the waste, the new exhausters are designed with higher flow rates to remove additional heat so that tanks remain within their operational temperature parameters. The mixing and transfer process should take approximately 15 to 20 days to complete. The rest of the time, the mixer pumps will be not be operating or will be removed, the waste will be quiescent, and the exhausters will run at their normal flow rates.

Delivery of the new exhausters is expected to take place between July 2011 and July 2012. After installation and testing, operations are scheduled to begin between October 2012 and October 2013. The exhausters will be designed for a 40-year design-life, consistent with the scheduled Hanford Site cleanup completion.[4]

WAC 173-400-113(5) requires a proposed new source or modification to comply with the TAP regulations in Chapter 173-460 WAC. Many volatile chemicals could be emitted from the tanks during the WFD process. Of these chemicals, estimated emissions and atmospheric modeling showed that only DMM could be emitted at a rate that would result in air concentrations greater than its ASIL concentration. Therefore, DOE and WRPS are required to submit a second tier petition per WAC 173-460-090.

4. POLLUTANT SCREENING

4.1. Emissions

DOE/WRPS listed information on emissions of 91 TAPs and other substances found in tank headspaces (Table 1).[5]

Table 1. TAPs Detected in Tank Headspaces

WAC 173-460-150 Name	CASRN
Trans-1,2-dichloroetherie	1 56-60-5
Ethylbenzene	100-41-4
Styrene	100-42-5
Benzyl Chloride	100-44-7
Nitrogen oxide	10102-43-9
Nitrogen dioxide	10102-44-0
n-Nitroso-n-methylethylamine	10595-95-6
p-Xylene	106-42-3
1,4-Dichlorobenzene	106-46-7
1,2-Epoxybutane	106-88-7
1,2-Dibromoethane	106-93-4
1,3-Butadiene	106-99-0

[4] Specific details are given in RPP-ENV-48229, Rev. 0, Section 4.0 Proposed Action, pp. 14–15.
[5] Table C-1, Toxic air pollutant emission rates and comparison to the WAC 173-460-150 *de minimis*, SQER, and ASIL levels in RPP-ENV-48231 Appendix C, p. 3 (p. 87–91 of 214).

WAC 173-460-150 Name	CASRN
Acrolein	107-02-8
Allyl Chloride	107-05-1
1,2-Dichloroethane	107-06-2
Acrylonitrile (2-Propenenitrile)	107-13-1
Vinyl acetate	108-05-4
Methyl Isobutyl Ketone	108-10-1
m-Xylene	108-38-3
3-Methylphenol	108-39-4
Toluene	108-88-3
Chlorobenzene	108-90-7
Phenol	108-95-2
n-Hexane	110-54-3
Cyclohexane	110-82-7
Ethylene glycol monobutyl ether	111-76-2
Propylene	115-07-1
Di(2-ethyIhexy)phthaIate	117-81-7
1,4-Dioxane	123-91-1
Perchloroethylene	127-18-4
Vanadium pentoxide	1314-62-1
PCBs	1336-36-3
Butylated hydroxyanisole	25013-16-5
Formaldehyde	50-00-0
n-Nitrosodiethylamine	55-18-5
Carbon Tetrachloride	56-23-5
1,1-Dimethylhydrazine	57-14-7
Propylene Glycol	57-55-6
Dimethyl mercury	593-74-8
n-Nitrosomorpholine	59-89-2
Hexachloroethane	6 7-72-1
Acetamide	60-35-5
n-Nitrosodi-n-propylamine	62 1-64-7
Methyl Isocyanate	624-83-9
n-Nitrosodimethylamine	62-75-9
Carbon monoxide	630-08-0
Methyl Alcohol	67-56-1
Isopropyl Alcohol	67-63-0
Chloroform	67-66-3
Benzene	71-43-2
1,1 1-Trichloroethane	71-55-6
Lead	7439-92-1
Mercury, Elemental	7439-97-6
Cadmium & Compounds	7440-43-9
Cobalt	7440-46-4
Methyl Bromide	74-83-9
Methyl Chloride	74-87-3
Hydrogen Cyanide	74-90-8
Ethyl Chloride	75-00-3
Vinyl Chloride	75-01-4
Acetonitrile	75-05-8
Acetaldehyde	75-07-0
Dichloromethane	75-09-2

WAC 173-460-150 Name	CASRN
Carbon disulfide	75-15-0
Ethylene oxide	75-21-8
Bromoform	75-25-2
1,1-Dichloroethane	7534-3
1,1-Dichloroethylene	75-35-4
Chlorodifluoromethane	75-45-6
1-Chloro-1,1-difluoroethane	75-68-3
Ammonia	7664-41-7
1 2-Dichloropropane	78-87-5
Methyl Ethyl Ketone	78-93-3
1,1,2- Trichloroethane	79-00-5
Trichloroethylene	79-01-6
Acrylic Acid	79-10-7
1,1,2,2-Tetrachloroethane	79-34-5
2-Nitro-Propane and others	79-46-9
Hexachlorobutadiene	87-68-3
Naphthalene and others	91-20-3
n-Nitroso-di-n-butylamine	924-16-3
n-Nitrosopyrrolidine	930-55-2
o-Xylene	95-47-6
2-methylphenol and others	95-48-7
Cumene	98-82-8
SO_X Total	
Sulfur dioxide (SO_X)	
NO_X	
NO_X total	
Total organic Carbon (non-methane)	

4.2. Best Available Control Technology for Toxics (tBACT)

The proposed DST ventilation systems are unique compared to ordinary pollution control devices. They are designed to control and greatly reduce emissions a wide range of radioactive and toxic substances in the DST headspaces. Specific details are given in RPP-ENV-48229, Rev. 0, Section 4.2, Best Available Control Technology, pp. 18-19.

RFO was responsible for establishing BACT and tBACT for the new DST ventilation systems. RFO has determined that BACT and tBACT for this project includes operation of each primary tank ventilation exhauster system not exceeding ventilation rates for WFD in Table 2, with a moisture de-entrainer, heater, prefilters, and a two-stage High Efficiency Particulate Air (HEPA) filtration system in service in each treatment train.

Table 2. Project Farm Ventilation Rate

Tank Farm	Storage, Retrieval, and Sampling Operations	Waste Feed Delivery Operations
241-SY	1,360 scfm	2,500 scfm
241-AP	1,500 scfm	3,000 scfm
241-AY/AZ	1,500 scfm	3,000 scfm
scfm = standard cubic foot per minute, 1 atmosphere pressure at 20°C		

Additionally, DMM emissions will be limited to approximately 7.85E-06 pounds per 24-hour period.

4.3. Air Dispersion Modeling

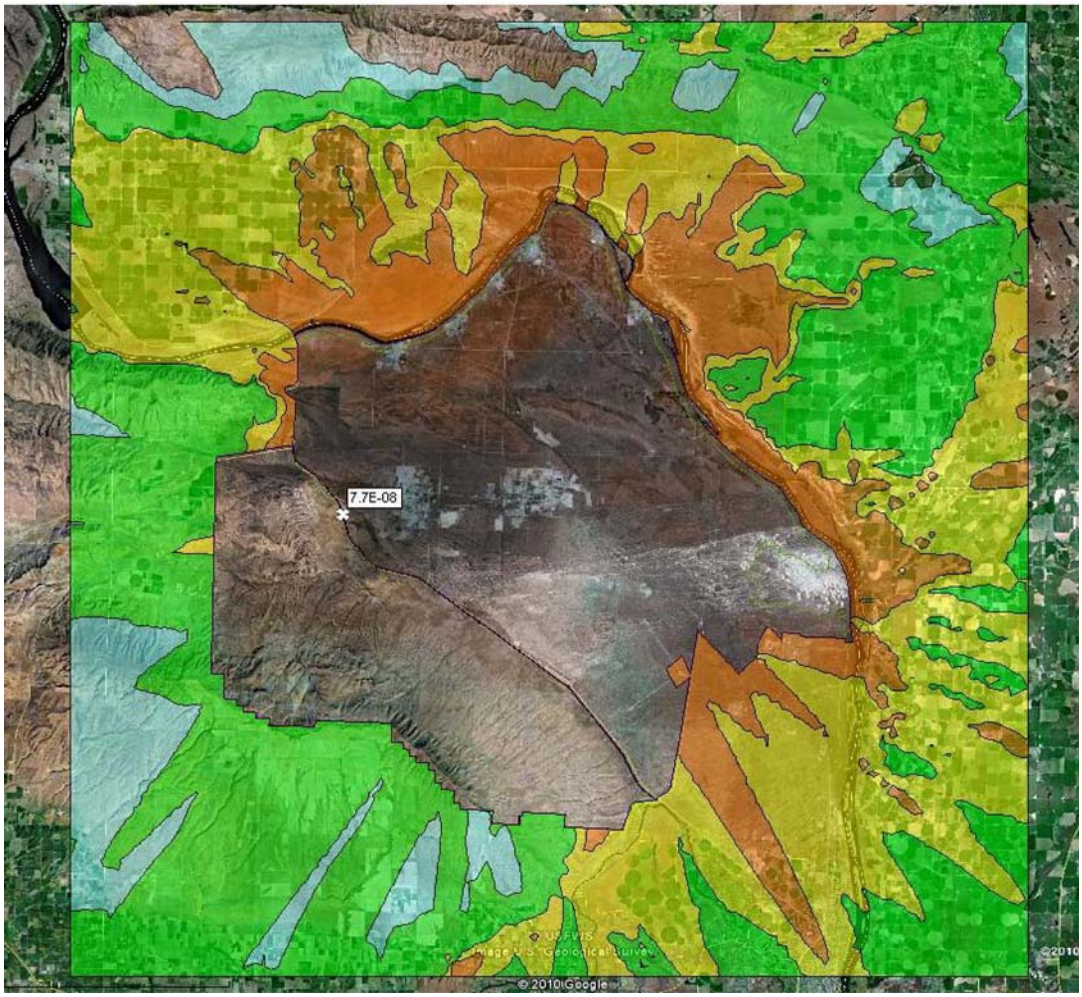
WRPS conducted air dispersion modeling of project emissions at the Hanford Site boundary and beyond.[6] WRPS used the United States EPA AERMOD dispersion model, Version 09292. EPA-454/B-03-001, *User's Guide for the AMS/EPS Regulatory Model – AERMOD* and Ecology's *Guidance Document: First, Second, and Third Tier Review of Toxic Air Pollution Sources* (08-02-025) for guidance. Specific modeling inputs are described in the petition.

4.4. Points of Compliance

The Hanford Site is restricted to public access by a fence and guards. The zone of compliance includes all areas outside the fence and the public right-of-way encompassed by Highway 240, which is to the south and west of the tank farms. The access boundary and Highway 240 are shown in Figure 1.

In meetings with DOE, WRPS, RFO, and Ecology, it was agreed that the U.S. Ecology Disposal Facility, which is inside the fence but not otherwise affiliated with DOE, would not be considered an off-site receptor since access to the U.S. Ecology Disposal Facility is controlled the same way as access to other portions of the Hanford Site.

[6] Specific details are given in RPP-ENV-48229, Rev. 0, Sections 4.3 and 4.4, pp. 19–25.



Concentration (µg/m3)	Contour Color
1E-08 to < 5E-08	
5E-09 to < 1E-08	
1E-09 to < 5E-09	
5E-10 to < 1E-09	

Figure 4. Twenty-four hour time-weighted average DMM concentrations attributable to the new DST ventilation systems

(Source: DOE/WRPS)

Table 3. TAP Emission Rates and Comparisons to the WAC 173-460-150 De Minimis, SQER, and ASIL Levels

Chemical Name	CAS#	Avg. Period	Emissions				De Minimis (lb/avg. period)	Above De Minimis?	SQER (lb/avg. period)	Above SQER?	Dispersed Conc. (µg/m^3)	ASIL (µg/m^3)	Above ASIL?
			(g/s)	(lb/hr)	(lb/24-hr)	(lb/yr)							
1,1,2,2-Tetrachloroethane	79-34-5	Year	7.45E-04	-	-	51.8	0.165	Yes	3.3	Yes	4.17E-05	0.0172	No
1,1,2-Trichloroethane	79-00-5	Year	5.92E-04	-	-	41.1	0.6	Yes	12	Yes	3.31E-05	0.0625	No
1,2-Dibromoethane	106-93-4	Year	7.97E-05	-	-	5.54	0.135	Yes	2.71	Yes	4.46E-06	0.0141	No
1,2-Dichloroethane	107-06-2	Year	1.63E-03	-	-	113.2	0.369	Yes	7.39	Yes	9.12E-05	0.0385	No
1,3-Butadiene	106-99-0	Year	1.99E-04	-	-	13.9	0.0564	Yes	1.13	Yes	1.12E-05	0.00588	No
1,4-Dioxane	123-91-1	Year	7.02E-04	-	-	48.8	1.25	Yes	24.9	Yes	3.93E-05	0.13	No
Acetaldehyde	75-07-0	Year	4.10E-03	-	-	285	3.55	Yes	71	Yes	2.30E-04	0.37	No
Acrylonitrile	107-13-1M	Year	1.23E-05	-	-	0.856	0.0331	Yes	0.662	Yes	6.90E-07	0.00345	No
Ammonia	7664-41-7	24-hr	1.45E-02	-	191	-	0.465	Yes	9.31	Yes	2.76E-02	70.8	No
Arsenic & Inorganic Arsenic Compounds	7440-38-2	Year	6.55E-05	-	-	4.56	0.00291	Yes	0.0581	Yes	3.67E-06	0.000303	No
Benzene	71-43-2	Year	1.63E-03	-	-	113.2	0.331	Yes	6.62	Yes	9.12E-05	0.0345	No
Beryllium & Compounds (NOS)	7440-41-7	Year	3.28E-06	-	-	0.228	0.004	Yes	0.08	Yes	1.84E-07	0.000417	No
Cadmium & Compounds	7440-43-9	Year	3.28E-05	-	-	2.28	0.00228	Yes	0.0457	Yes	1.84E-06	0.000238	No
Carbon Tetrachloride	56-23-5	Year	1.64E-03	-	-	114	0.228	Yes	4.57	Yes	9.18E-05	0.0238	No
Chloroform	67-66-3	Year	1.64E-03	-	-	114	0.417	Yes	8.35	Yes	9.18E-05	0.0435	No
Chromium Hexavalent: Soluble, except Chromic Trioxide	7440-47-3	Year	1.00E-04	-	-	6.98	0.000064	Yes	0.00128	Yes	5.63E-06	6.67E-06	No
Dichloromethane	75-09-2	Year	1.11E-02	-	-	773	9.59	Yes	192	Yes	6.23E-04	1	No

Chemical Name	CAS#	Avg. Period	Emissions				De Minimis (lb/avg. period)	Above De Minimis?	SQER (lb/avg. period)	Above SQER?	Dispersed Conc. (µg/m^3)	ASIL (µg/m^3)	Above ASIL?
			(g/s)	(lb/hr)	(lb/24-hr)	(lb/yr)							
Dimethyl Mercury	593-74-8	24-hr	4.12E-08	-	7.85E-06	-	1.00E-99	Yes	1.00E-99	Yes	7.83E-08	1.00E-99	Yes
Ethylbenzene	100-41-4	Year	1.41E-03	-	-	98.1	3.84	Yes	76.8	Yes	7.90E-05	0.4	No
Hexachlorobutadiene	87-68-3	Year	1.16E-03	-	-	80.8	0.437	Yes	8.73	Yes	6.50E-05	0.0455	No
Hexachloroethane	67-72-1	Year	1.68E-03	-	-	117	0.872	Yes	17.4	Yes	9.42E-05	0.0909	No
Manganese & Compounds	7439-96-5	24-hr	6.55E-05	-	0.0125	-	0.000263	Yes	0.00526	Yes	1.25E-04	0.04	No
n-Nitrosodiethylamine	55-18-5	Year	1.94E-06	-	-	0.135	0.000959	Yes	0.0192	Yes	1.09E-07	0.0001	No
n-Nitrosodimethylamine	62-75-9	Year	2.65E-03	-	-	184	0.00208	Yes	0.0416	Yes	1.48E-04	0.000217	No
n-Nitroso-di-n-butylamine	924-16-3	Year	1.94E-06	-	-	0.135	0.0031	Yes	0.062	Yes	1.09E-07	0.000323	No
n-Nitrosodi-n-propylamine	621-64-7	Year	1.94E-06	-	-	0.135	0.0048	Yes	0.0959	Yes	1.09E-07	0.0005	No
n-Nitrosomorpholine	59-89-2	Year	8.96E-06	-	-	0.623	0.00505	Yes	0.101	Yes	5.01E-07	0.000526	No
n-Nitroso-n-methylethylamine	10595-95-6	Year	1.94E-06	-	-	0.135	0.00153	Yes	0.0305	Yes	1.09E-07	0.000159	No
Perchloroethylene	127-18-4	Year	1.62E-03	-	-	112.4	1.62	Yes	32.4	Yes	9.06E-05	0.169	No
Polychlorinated Biphenyls (PCBs)	1336-36-3	Year	2.04E-05	-	-	1.415	0.0168	Yes	0.336	Yes	1.14E-06	0.00175	No
Trichloroethylene	79-01-6	Year	1.63E-03	-	-	113.2	4.8	Yes	95.9	Yes	9.12E-05	0.5	No
Vinyl Chloride	75-01-4	Year	1.64E-03	-	-	114	0.123	Yes	2.46	Yes	9.18E-05	0.0128	No
Source: DOE/WRPS RPP-ENV-48229 –Table A-2													

4.5. Maximum TAP Concentrations

DOE/WRPS calculated maximum air-dispersion-modeled concentrations of all 91 TAPs that have been detected in tank headspace.[7] Only DMM emissions would produce ground-level concentrations exceeding its ASIL as listed in WAC 173-460-150. The air-dispersion-modeled maximum concentrations of all other TAPs are below their ASIL values.

TAPs that exceeded their SQERs are shown in Table 3. The highest modeled off-site concentration of each TAP is compared to its respective ASIL.

Figure 4 shows the average DMM concentration gradient attributable to the new DST ventilation systems that could occur in the single worst year among five recent years. Because the DMM concentration will exceed its ASIL, a second tier petition, as defined in WAC 173-460-090, is required to evaluate the potential health impacts of the project.

The remainder of this document describes the results of Ecology's review of the HIA supplied by DOE/WRPS.

4.6. Pollutants Subject to Second Tier Review

As shown in Table 3, DMM is subject to second tier review. The air dispersion modeling analysis presented in the new exhausters permit application predicts that in a 24-hour averaging period, the off-site or extra-boundary concentration of DMM would exceed its ASIL. Another 23 TAPs exceed their SQER values.

5. HEALTH IMPACT ASSESSMENT

5.1. Introduction

Information pertaining to potential health impacts of DMM to be emitted from the new exhausters was prepared by DOE and WRPS. Ecology reviewed the information and prepared this assessment of health risks to the public associated with exposure to the planned emissions. Ecology's Air Quality Program (AQP) review team consists of a professional engineer, a meteorologist, and a toxicologist. Ecology's assessment follows the requirements promulgated in Chapter 173-460 WAC. The analysis is not a complete risk assessment, but it follows the four steps of the standard health risk assessment approach proposed by the National Academy of Sciences (NAS, 1983, 1994)[8,9]: (1) hazard identification, (2) exposure assessment, (3) dose-response assessment, and (4) risk characterization. The assessment constitutes the basis for the AQP risk manager's decision.

[7] Table C-1, Toxic air pollutant emission rates and comparison to the WAC 173-460-150 *de minimis*, SQER, and ASIL levels in RPP-ENV-48231 on Appendix C, p. 3 (p. 87–91 of 214).
[8] NAS, 1983, National Academy of Sciences, Risk Assessment in the Federal Government: Managing the Process, National Research Council, National Academy Press, Washington, D.C.
[9] NAS, 1994, National Academy of Sciences, Science and Judgment in Risk Assessment, National Research Council, National Academy Press, Washington, D.C.

5.2. Hazard Identification

DOE/WRPS provided emission rates of each TAP in the list of substances found in tank headspaces. The emission rates of 41 of these TAPs exceed their de minimis values. Among these, 32 exceed their SQER values (Table 3).

The hazard identification step of the health risk analysis involves assessing information on potential adverse health effects associated with TAPs that exceed their SQERs. Table 4 summarizes the potential health effects of the TAPs that exceed their SQERs.

Table 4. Potential Adverse Effects of TAPs to be Emitted in Amounts Above SQERs

TAP Emissions That Exceed SQERs	Potential Effects and Hazard Index Targets
1,1,2,2-Tetrachloroethane	Possible human carcinogen; increased incidence of hepatocellular carcinomas in mice; hepatic and respiratory effects
1,1,2-Trichloroethane	Possible human carcinogen; hepatocellular carcinomas and pheochromocytomas in one strain of mice; hepatic and neurological effects. A LOAEL concentration of 418 ppm in air for neurological effects in experimental animals was presented by ATSDR, but they found no studies regarding neurodevelopment effects in humans or animals.[10] 418 ppm (2.32E6-µg/m^3) is far more than the 0.000000184-µg/m^3 dispersed concentration estimate from proposed Hanford emissions.
1,2-Dibromoethane	Carcinogenicity; dermal, hepatic, renal, reproductive effects and nasal inflammation; no reported neurodevelopmental effects
1,2-Dichloroethane	Probable human carcinogen. Based on the induction of several tumor types in rats and mice treated by gavage and lung papillomas in mice after topical application.
1,3-Butadiene	Carcinogenicity; ovarian atrophy; neurological effects at very high exposure concentrations; no reported neurodevelopmental effects
1,4-Dioxane	Probable human carcinogen; eye and respiratory irritation; neurological and developmental effects only at very high exposures
Acetaldehyde	Probable human carcinogen; degeneration of olfactory epithelium; may cause neural tube defects at high doses as seen in Fetal Alcohol Syndrome
Acrylonitrile	Carcinogenicity; degeneration and inflammation of nasal respiratory epithelium; hyperplasia of mucous secreting cells; inhalation exposure results in teratogenic effects in rats. There is no information on potential neurodevelopmental effects.
Ammonia	Eye and respiratory irritation; increased severity of rhinitis and pneumonia with respiratory lesions
Arsenic & Inorganic Arsenic Compounds	Known human carcinogen; cardiovascular effects. There is evidence from epidemiological studies that inhaled inorganic arsenic can produce neurological effects, peripheral neuropathy sensory and motor

[10] http://www.atsdr.cdc.gov/ToxProfiles/tp148-c2.pdf

TAP Emissions That Exceed SQERs	Potential Effects and Hazard Index Targets
	polyneuropathy, pseudoneurasthenic syndrome, toxic encephalopathy, auditory nerve damage, reduced verbal IQ impairment, developmental effects (skeletal malformations and the retarded growth) by inhalation exposure in laboratory animals, although it is unclear whether or not the effects occur only at maternally toxic doses.[11]
Benzene	Known human carcinogen. At high exposure levels, adverse effects involve multiple organs and biological processes. The acute hazard targets are reproductive and developmental organs, immune system, hematologic system; chronic hazard targets are hematopoietic system, development. Chronic inhalation exposure has been associated with distal neuropathy, difficulty in sleeping, and memory loss. Studies in animals suggest that inhalation exposure to benzene results in depressed electrical activity in the brain, loss of involuntary reflexes and narcosis, decrease in hind-limb grip strength and tremors, and narcosis, among other symptoms. However, benzene levels in ambient air near Hanford are far lower than levels associated with chronic neurological effects and therefore not likely to be of concern.[12]
Beryllium & Compounds (NOS)	Known human carcinogen; acute or chronic beryllium disease (inflammatory reaction in the respiratory tract)
Cadmium & Compounds	Known human carcinogen. Cardiovascular, developmental effects during periods when organs are developing, gastrointestinal, neurological, renal, reproductive respiratory. Neurodevelopmental effects have been observed at lower doses. Impaired performance on neurobehavioral tests were observed in the offspring of rats exposed to 0.02 mg/m^3 or ≥0.04 mg/kg-day. Neurodevelopmental effects including alterations in motor activity and delays in the development of sensory motor coordination reflexes. An MRL of 0.01-µg-Cd/m^3 has been derived for chronic-duration inhalation exposure.[13] This is far more than the estimated dispersed concentration of 0.000000184-µg/m^3 that could result from proposed Hanford emissions.
Carbon Tetrachloride	Probable human carcinogen; reproductive impairment; developmental effects including fetal growth retardation
Chloroform	Probable human carcinogen; reproductive and developmental toxicant; developmental toxicity involves the GI tract and kidney.
Chromium Hexavalent: Soluble, except Chromic Trioxide	Known human carcinogen. Chronic exposure has been reported to be associated with adverse effects in the respiratory tract, gastrointestinal system, eyes and conjunctiva, kidney, hematopoietic and immunological systems. High doses to pregnant experimental animals have been associated with a range of abnormalities in offspring but not with neurological deficits.
Dichloromethane	Probable human carcinogen; cardiovascular, hepatic, and neurological effects have been observed. Absence of CNS effects except at high

[11] http://www.atsdr.cdc.gov/ToxProfiles/tp2.pdf
[12] http://www.atsdr.cdc.gov/ToxProfiles/tp3-c2.pdf
[13] http://www.atsdr.cdc.gov/ToxProfiles/tp5.pdf

TAP Emissions That Exceed SQERs	Potential Effects and Hazard Index Targets
	exposure levels in experimental animals suggests it is not likely to cause developmental effects or behavioral changes at levels normally encountered in the environment which, according to research cited by ATSDR, is as low as 0.17-µg/m^3.[14] This is far more than the 0.000623-µg/m^3 dispersed concentration estimate from proposed Hanford emissions.
Dimethyl Mercury	DMM can cause delayed, permanent brain damage, weakness, impaired hearing, difficulty walking, personality changes, tremors, and death. DMM is readily absorbed through skin. It is an environmental contaminant found with monomethyl mercury in fish and birds, and throughout the atmosphere. There is no published RBC.
Ethylbenzene	Development; alimentary system (liver); kidney; endocrine system; irritation; CNS, Developmental effects have been reported in the offspring of pregnant animals exposed to ethylbenzene during gestation. Neurodevelopment in offspring of rats exposed to up to 500 ppm (2.2E6 –µg/m^3) ethylbenzene in a two-generation reproductive toxicity study was not significantly affected statistically or biologically. 2.2E6 –µg/m^3 is far more than the 0.000079-µg/m^3 dispersed concentration estimate from proposed Hanford emissions.
Hexachlorobutadiene	Possible human carcinogen
Hexachloroethane	Possible human carcinogen
Manganese & Compounds	Manganism (neurotoxicity); tolerable upper intake in pregnant women is the same as men and non-pregnant women but lower for infants and children.[15] Exposures in childhood are associated with impaired neurodevelopment including decrements in intellectual function.
n-Nitrosodiethylamine	Probable human carcinogen
n-Nitrosodimethylamine	Probable human carcinogen; immune system; no studies regarding neurological or developmental effects could be located
n-Nitroso-di-n-butylamine	Probable human carcinogen
n-Nitrosodi-n-propylamine	Probable human carcinogen
n-Nitrosomorpholine	Carcinogenicity
n-Nitroso-n-methylethylamine	Probable human carcinogen
Perchloroethylene	Carcinogenicity; mild CNS effects; eye and respiratory tract irritation
Polychlorinated Biphenyls (PCBs)	Probable human carcinogen
Trichloroethylene	Carcinogenicity; neurotoxic effects (drowsiness, fatigue, headache) and eye irritation in workers; developmental toxicity studies are not available
Vinyl Chloride	Known human carcinogen; liver cell polymorphism; mild CNS effects; eye and respiratory irritation; no evidence of developmental toxicity

[14] http://www.atsdr.cdc.gov/ToxProfiles/tp14.pdf
[15] http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD1_final.pdf#page=170

DMM is the only TAP whose concentration will exceed its ASIL. The neurotoxic potential of DMM is the health hazard that triggers second tier review.

Two other TAPs, hexavalent chromium and n-nitrosodimethylamine, may be emitted at rates that could result in their concentrations approaching their ASILs, but these toxicants are not known to be neurotoxic. Because of this, Ecology did not consider them in a neurotoxicity hazard index with DMM. The modeled concentrations of DMM and all the other potentially neurotoxic TAPs attributable to Hanford exhaust emissions are far less than their effects concentrations. In the following sections, Ecology explains its conclusions that the emissions of these TAPs present no appreciable potential to cause neurotoxicity.

5.2.1. DMM

As shown in Table 3, the estimated maximum 24-hour time-weighted average (TWA) DMM concentration attributable to Hanford exhaust emissions anywhere beyond the Hanford facility public assess boundary is likely to be 7.7E-05-$\mu g/m^3$. DMM can cause delayed, permanent brain damage, weakness, impaired hearing, difficulty walking, personality changes, tremors, and death. The earliest effects of high exposures are symptoms such as paresthesia, blurred vision, and malaise. Effects at higher doses include deafness, speech difficulties, and constriction of the visual field. It is an environmental contaminant found with monomethyl mercury (MMM) in fish and birds. There is no published RBC for DMM; however, there is a reference dose (RfD) for MMM based on developmental neurologic abnormalities in human infants.

5.2.2. Hexavalent Chromium

As shown in Table 3, the estimated maximum annual average chromium VI concentration attributable to Hanford exhaust emissions beyond the Hanford boundary is likely to be 5.63E-06-$\mu g/m^3$ or less. Chromium VI is a known human carcinogen. Chronic exposure is associated with adverse effects in the respiratory tract, gastrointestinal tract, eyes and conjunctiva, and kidneys. Chromium VI exposure is also associated with hematopoietic and immunological dysfunctions. High doses to pregnant experimental animals have been associated with a range of abnormalities in offspring but not with neurological deficits. Because exposure to chromium VI is not associated with abnormal neurodevelopment, and because proposed emissions will not result in concentrations greater than the effects levels of other forms of toxicity, Ecology did not evaluate chromium VI further in the subsequent analyses.

5.2.3. N-Nitrosodimethylamine

As shown in Table 3, the estimated maximum annual average n-nitrosodimethylamine concentration attributable to Hanford exhaust emissions beyond the Hanford boundary is likely to be 0.000217-$\mu g/m^3$ or less. N-Nitrosodimethylamine is a probable human carcinogen. Immunological impairment is associated with exposure to amounts greater than those associated with significant cancer risk. No studies regarding its potential for neurological or developmental effects could be located in the scientific literature. However, because n-nitrosodimethylamine is highly chemically reactive, it is unable to pass through the placenta into fetal circulation. Even if

it is harmful to the fetal brain, it cannot reach it from the maternal circulation. Accordingly, Ecology did not evaluate n-nitrosodimethylamine further.

5.3. Environmental Fate

DOE/WRPS reviewed scientific literature on the atmospheric fate of DMM.[16] Ecology concurs with their finding that the lifetime of DMM in the atmosphere ranges from roughly 1 to 100 hours.

A literature search did not yield information on the fate of DMM deposited in terrestrial and aquatic environmental compartments. Nonetheless, some DMM in the atmosphere must deposit onto soil, plants, and other objects. DOE/WRPS calculated the amount of Hanford-attributable atmospheric DMM that would enter plants and soil in the vicinity of the exhausters.[17] DMM is not water soluble.

5.4. Exposure Assessment

In order for pollutants to cause harm, people must be exposed. The exposure assessment step of the HIA involves measuring or estimating concentrations, durations, and frequencies of exposures to agents present in the environment, and the estimation of hypothetical exposures that might arise from the release of TAPs into the air outside of space controlled by the permit applicant. Ambient air is publicly accessible air in the vicinity of a proposed project. To the practical extent possible, the current exposure assessment characterizes past, current, and expected TAP exposures.

Inhalation will be the dominant exposure route of humans to Hanford exhaust DMM emissions. Lesser exposures via ingestion and skin contact will also occur.

5.4.1. Multi-Route Exposures

The following paragraph and Table 5 are from the California OEHHA's *Air Toxics Hotspots Risk Assessment Guidance*.[18]

> Table [4] shows the multipathway substances that, based on available scientific data, can be considered for each non-inhalation exposure pathway. The exposure pathways that are evaluated for a substance depend on two factors: 1) whether the substance is considered a multipathway substance for the Hot Spots Program (Table 5.1), and 2) what the site-specific conditions are. A multipathway substance may be excluded from a particular exposure pathway because its physical-chemical properties can preclude significant exposure via the pathway.

[16] RPP-ENV-48231, Rev. 0, Part 5.4 ATMOSPHERIC FATE, p. 28.
[17] RPP-ENV-48231, Rev. 0, Part 7.2 ESTIMATION OF EXPOSURE THROUGH INGESTION, pp. 37–38.
[18] The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, August 2003.

For example, some water-soluble chemicals do not appreciably bioaccumulate in fish; therefore, the fish pathway is not appropriate. In addition, if a particular exposure pathway is not impacted by the facility or is not present at the receptor site, then the pathway is not evaluated. For example, if surface waters are not impacted by the facility, or the water source is impacted but never used for drinking water, then the drinking water pathway is not evaluated.

Table 5. Specific Pathways to be Analyzed for Each Multi-Pathway Substance

Substance	Ingestion Pathway									
	Soil	Dermal	Meat, Milk & Eggs	Fish	Exposed Vegetable	Leafy Vegetable	Protected Vegetable	Root Vegetable	Water	Breast Milk
4,4'-Methylene dianiline	X	X		X	X	X	X	X	X	
Creosotes	X	X	X	X	X	X			X	
Diethylhexylphthalate	X	X		X	X	X	X	X	X	
Hexachlorocyclohexanes	X	X		X	X	X			X	
PAHs	X	X	X	X	X	X			X	
PCBs	X	X	X	X	X	X	X	X	X	X
Cadmium & compounds	X	X	X	X	X	X	X	X	X	
Chromium VI & compounds	X	X	X	X	X	X	X	X	X	
Inorganic arsenic & compounds	X	X	X	X	X	X	X	X	X	
Beryllium & compounds	X	X	X	X	X	X	X	X	X	
Lead & compounds	X	X	X	X	X	X	X	X	X	
Mercury & compounds	X	X		X	X	X	X	X	X	
Nickel	X	X	X		X	X	X	X	X	
Fluorides (including hydrogen fluoride)	To be determined									
Dioxins & furans	X	X	X	X	X	X	X		X	X

DMM emitted from the exhausters will enter food crops and soil near Hanford in minute amounts. Ecology requested that DOE/WRPS calculate the amount of Hanford-attributable atmospheric DMM people could be exposed to via contact and ingestion of plants and soil in the vicinity of the exhausters. Ecology deemed that quantifying exposures via the dermal route, and via ingestion of meat, milk, eggs, and water, was very unlikely to yield significant concerns. Under the circumstances of the Hanford exhausters, inhalation of air and ingestion of vegetables and soil are the only routes of exposure with significant potential to increase DMM body burden.

5.4.2. Identification of Exposed Populations

To assess exposure to TAPs and ultimately estimate potential health risks to people exposed to exhauster emissions, DOE/WRPS identified key locations where people might be exposed, and evaluated population demographic characteristics and land-use zoning in the area around the

Hanford site.[19] The high level of detail in the information they presented is exemplary. However, the location of the maximally impacted commercial receptor (MICR) that could experience highest average DMM concentrations according to AERMOD results was not clearly disclosed.[20]

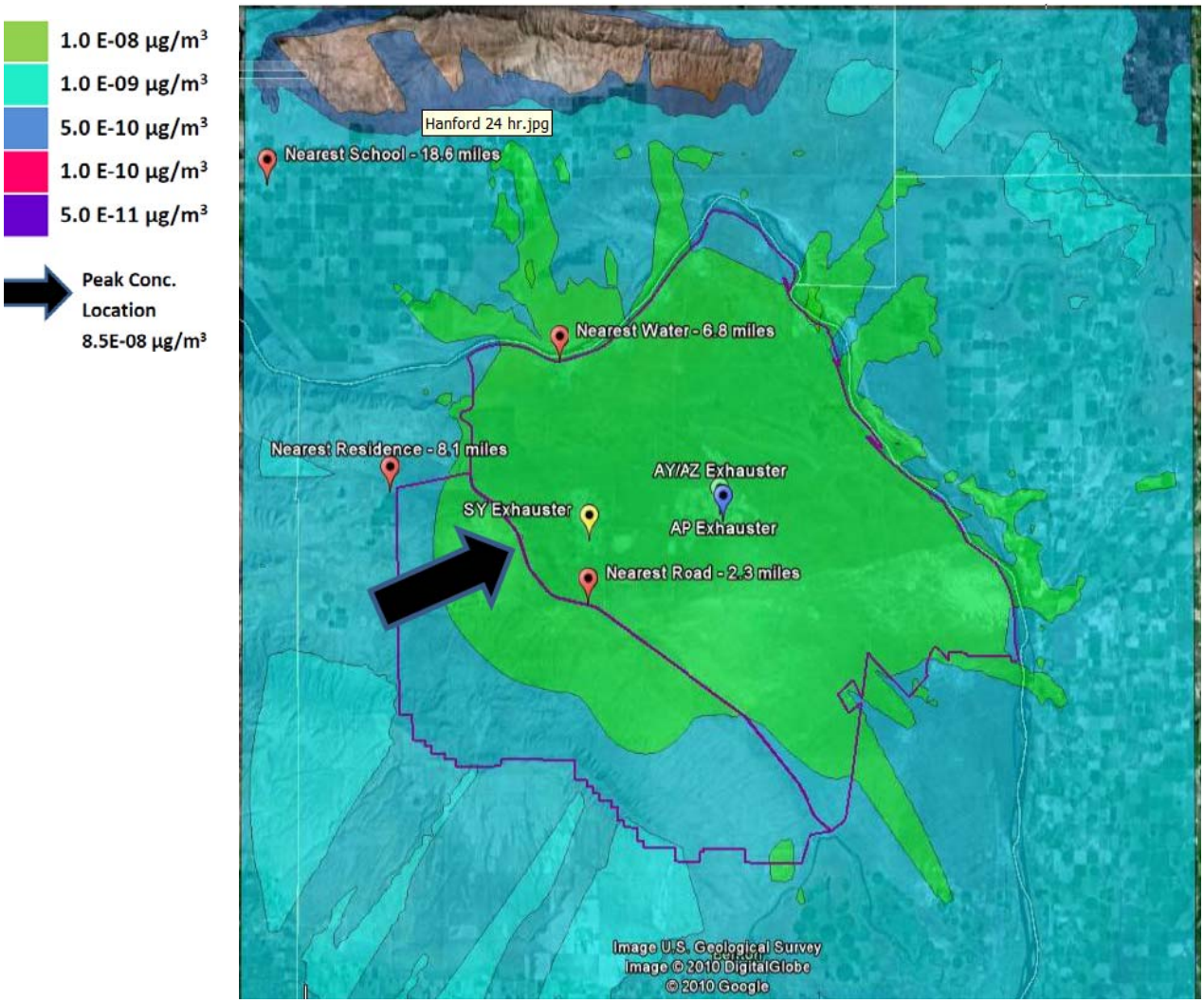


Figure 5. Modeled peak 24-hour average DMM concentrations in the vicinity of Hanford (Source: RPP-ENV-48231, Rev. 0)

DOE/WRPS states, "[the] nearest resident is impacted more than the highest commercial receptor so the resident scenario is assumed to be more conservative."[21] Also, "[onsite]

[19] RPP-ENV-48231, Rev. 0, Part 6.0 SENSITIVE POPULATION ANALYSIS, pp. 29–34.
[20] RPP-ENV-48231, Rev. 0, Part 2.6 RECEPTORS, pp. 15–16.
[21] RPP-ENV-48231, Rev. 0, Part 4.3 AIR DISPERSION MODELING, p. 21.

modeling has been performed to assess worker exposure (RPP-RPT-47978, *Atmospheric Modeling of the Stack Heights for the AP and SY Exhausters*, 2010).[22]

DOE/WRPS identified the dwelling that could experience highest average DMM concentrations based on AERMOD results. This dwelling is to the west of the exhausters. It is the maximally impacted residential receptor (MIRR).

DOE/WRPS also identified a location on Highway 240 as the outdoor location, beyond the controlled access area of the Hanford Site, where simulated DMM concentration maxima could occur; i.e., the maximally impacted extra-boundary receptor (MIBR).

The MIRR and MIBR attributable to the exhauster DMM emissions are indicated in Figure 5.

5.4.3. Estimates of Exposure Durations of Identified Populations

Neurotoxicity risk from exposure to DMM is estimated by determining the DMM concentration at each receptor point. These concentrations are divided by the DMM RBC limit. The DMM RBC is based on preventing significant prenatal exposure, the most vulnerable period. In addition, a full lifetime of continuous exposure to concentrations at or below the RBC is not likely to result in neurotoxicity or other deleterious effects.

For this analysis, DOE/WRPS estimated the exposure a person would have if they lived at and consumed produce grown at the location of maximum DMM concentration along Highway 240 to the south and west of the 241-SY Tank Farm.[23] This hypothetical exposure is the highest–albeit improbable and inconceivable. No one lives at or near that location now nor is anyone likely to at any time in future years while tank waste treatment and exhauster emissions continue.

5.4.4. TAP Concentration Estimates

To assess exposure to TAPs attributable to the tank farm ventilation systems, DOE/WRPS used AERMOD to calculate the 1-hour and 24-hour TWA maximum concentrations and the average annual concentrations for each TAP according to its required TWA duration. The TAP concentrations were centered in breathing zone air at each of the grid points in the modeling domain.[24] The model used emissions rate estimates combined with recent meteorological data. DOE/WRPS reported estimates of concentrations at the grid points with the highest concentrations (Table 3).

[22] RPP-ENV-48231, Rev. 0, Part 4.3 AIR DISPERSION MODELING, p. 20.
[23] RPP-ENV-48231, Rev. 0, Part 7.0 EXPOSURE ESTIMATION, pp. 35–39.
[24] RPP-ENV-48231, Rev. 0, Part 4.3 AIR DISPERSION MODELING, pp. 19–21.

Table 6. Maximum Off-Site DMM Atmospheric Concentrations and Deposition Rates Attributable to the Tank Farm Exhauster Emissions

Maximally Impacted	24-Hour Average ($\mu g/m^3$)	Peak Annual ($\mu g/m^3$)	Peak 24-Hour Deposition ($\mu g/m^2 \cdot day$)
Extra-boundary location (a point on Hwy 240)	7.7E-8	2.3E-09	1.9E-05
Residence (a dwelling 8.1 miles west of the 241-SY Tank Farm)	2.8E-09		1.0E-06
Source: DOE/WRPS			

Table 6 presents maximum off-site DMM concentrations and deposition rates attributable to emissions from the exhausters. Ecology verified that the DMM concentrations at the MIBR and MIRR locations reported by DOE/WRPS were correct and that the given locations of these receptors agree with AERMOD results.

5.4.4.1. Existing Background Levels

In accordance with WAC 173-460-090(5), Ecology considered background concentrations of DMM as part of this second tier review. Under anaerobic conditions, inorganic mercury is transformed into MMM and DMM by certain bacteria species. DMM is probably emitted by such bacteria from any anaerobic environment. The global geochemical mercury cycle includes both methylated and inorganic forms of mercury. In bottom sediments of natural waters, bacteria methylate inorganic mercury ions into MMM and DMM. These methylated forms subsequently bioaccumulate in aquatic food chains. DMM also evaporates into the atmosphere.[25]

DOE/WRPS reviewed the state of current knowledge on existing levels of DMM as needed for predicting how much exposure there will be from both existing and proposed emissions.[26] As noted in their review, very little research about background atmospheric DMM concentrations has been published; however, a mean of 0.04 ng/m^3 has been observed in Antarctica, and a mean of 0.003 ng/m^3 has been observed in Seattle, Washington.[27] Municipal landfills have been identified as an anthropogenic source of atmospheric DMM.

The peak modeled 24-hour DMM concentration attributable to the tank ventilation system is 7.7E-05 ng/m^3. Peak DMM concentrations attributable to the tank ventilation exhauster emissions are likely to be 39-fold lower than the measured mean background concentration in Seattle and 519-fold lower than the mean measured background concentration in Antarctica.

Ecology is not aware of any data on background DMM deposition rates or measured concentrations in plants and soil in the Hanford area.

[25] Selin, 2009.
[26] RPP-ENV-48231, Rev. 0, Part 5.3 BACKGROUND CONCENTRATIONS, pp. 27–28.
[27] RPP-ENV-48231, Part 5.3 BACKGROUND CONCENTRATIONS, pp. 27–28.

5.5. Exposure-Response Assessment

Exposure-response assessment is the process of characterizing the potential incidence of adverse health effects in humans resulting from exposure and uptake of toxicants. The process often involves establishing risk-based toxicity values or criteria to use in assessing potential health risk from each toxicant. Exposure-response assessment attempts to consider time-changing exposure magnitudes in whole populations and in theoretically maximally exposed individuals.

5.5.1. Risk-Based Dose Limit for Exposed Populations

The DMM ASIL is not proportional to DMM's toxicity. Emissions of DMM are subject to second tier review based on a trigger level of one emitted molecule.

There is no risk-based dose published specifically for DMM. There is a paucity of scientific information concerning the toxicity of this chemical. However, enough information exists to make an informed estimate of how much DMM people may be exposed to without the likelihood they will experience neurotoxic effects.

MMM is similar to DMM in its toxic effects. There is no reference concentration (RfC) for MMM, but it does have a published RfD. The RfD is the oral dose that is believed to be without harm. The MMM RfD is 1E-4-mg/kg-day, which is 0.1-µg/kg-day.[28]

DMM is not toxic itself. DMM is internally inert until metabolized to a tissue reactive product, which is most likely MMM. When DMM is absorbed internally, some metabolizes to MMM, which is toxic. MMM reacts primarily with thiol-containing amino acids and secondarily with carboxyl and amine containing biomolecules. Brain uptake of MMM is via the blood brain barrier methionine transporter.[29] The observed uptake of mercury into rat central nervous system (CNS) from doses of DMM matches the uptake rate observed of mercury in dosed MMM.[30] This experimental evidence strongly suggests DMM is metabolized to MMM, which then can enter the brain. Similarly, the observed course of human toxicosis is consistent with this kinetic pattern.[31]

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[28] The RfD for MMM is expressed as MMM/body mass-day (IRIS 1995). EPA established an RfD of 0.0001 mg/kg-day (0.0001 milligram of methylmercury per day for each kilogram of a person's body mass) (USEPA, 2002). EPA believes that exposures at or below the RfD are unlikely to be associated with an appreciable risk of deleterious effects. It is important to note, however, that the RfD does not define an exposure level corresponding to zero risk; mercury exposure near or below the RfD could pose a very low level of risk that EPA deems nonappreciable. It is also important to note that the RfD does not define a bright line above which individuals are at risk of adverse effects (USEPA, 2005).

[29] From EPA's 1996 Mercury Report to Congress: "…mediated by the formation of a methylmercurycysteine complex (Aschner and Aschner 1990; Tanaka et al. 1991, 1992; Kerper et al. 1992). The complex is structurally similar to methionine and is transported into cells via a widely distributed neutral amino acid carrier protein. Methylmercury associates with water-soluble molecules (e.g., proteins) or thiol-containing amino acids because of the high affinity of the methylmercuric cation (CH3Hg) + for the sulfhydryl groups (SH)-. Complexes of methylmercury with cysteine have been identified in blood, liver and bile of rats (Aschner and Aschner 1990)."

[30] Ostlund, 1969.

[31] Nierembuerg, et al., 1998.

The primary exposure route to MMM is through the gastrointestinal (GI) tract; mostly by consumption of contaminated fish and shellfish.[32] About 95 percent of MMM in ingested food is absorbed from the GI by adult humans.[33] Therefore, 95 percent of 0.1-µg/kg-day is the tolerable internal dose of MMM.

Tolerable internal dose of MMM = 0.95 x 0.1-µg/kg-day = 0.095-µg/kg-day

There is no information available about how much absorbed DMM is metabolized to MMM by humans after a single dose, much less in a continuous long-term exposure. If all the DMM absorbed was metabolized to MMM, the RfD for DMM would be 100 percent of the tolerable internal dose (TID) of MMM adjusted for the difference in molecular weights. The MMM molecular weight is 215.63; the DMM molecular weight is 230.66. Thus, the molecular weight ratio is:

DMM MW ÷ MMM MW = 1.0697

The TID of DMM equivalent to the TID of MMM is:

0.1016-µg/kg-day	=	0.095-µg/kg-day	x	1.0697

The internal dose of DMM for a person weighing 67-kg[34] is:

6.809-µg DMM/day	=	0.1016-µg/kg-day	x	67-kg

Developing fetuses are more sensitive to the neurotoxic effects of MMM than at any other life stage. The EPA/NRC's RfC derivation literature and the ATSDR[35] literature review concluded the developing nervous system is the most vulnerable tissue[36] and the fetal life stage is the most sensitive period for nervous system effects. The MMM RfD is intended to protect the fetal subpopulation from neurotoxicity, but neurotoxic effects can occur due to exposure at any time in life. The National Research Council (NRC) recommended a RfD of 0.1-µg/kg-day to protect pregnant women and developing fetuses. In developing the RfD, the NRC applied uncertainty factors of three each to pharmacokinetic variability and uncertainty, and to pharmacodynamic variability and uncertainty. They used a factor of 10, in total, to derive the RfD.[37]

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[32] The primary route by which the U.S. population is exposed to MMM is through the consumption of fish. The exposure levels at which neurological effects have been observed in children can occur through maternal consumption of fish. U.S. Environmental Protection Agency, *Integrated Risk Information System (IRIS), Methylmercury*, 2002. U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment, http://www.epa.gov/iris/ subst/0073.htm.
[33] John B. Sullivan, Jr. and Gary R. Krieger (eds.), *Clinical Environmental Health and Toxic Exposures*, 2d ed., Lippincott, Williams & Wilkins, Philadelphia, 2001.
[34] The EPA/NRC used 67 kg as the weight of a pregnant woman for setting the MMM RfD.
[35] http://www.atsdr.cdc.gov/toxprofiles/tp46.pdf 6/15/2011
[36] Cardiovascular, kidney, immune and reproductive systems impairments have also been observed at higher MMM doses.
[37] EPA, IRIS, http://www.epa.gov/iris/subst/0073.htm, 2001.

The total DMM dose limit used by DOE/WRPS was 0.1-µg/kg body weight per day. This is less than (and more protective of health than) the tolerable internal DMM dose derived by Ecology of 0.1016-µg/kg-day.

5.6. Risk Characterization

In the risk characterization, conclusions about hazards and exposure responses are integrated with the exposure assessment conclusions. Health hazards are quantified and attempts are made to estimate increased likelihoods of these effects in populations exposed to anticipated TAP emissions. In addition, confidence about these conclusions, including information about the uncertainties associated with each aspect of the assessment, is highlighted.

5.6.1. Hazard Quotient for Total DMM Dose

To determine if the tank farm ventilation system emissions will pose a significant toxicity risk, DOE/WRPS calculated the HQ for the TAP that exceeded its ASIL (DMM). DOE/WRPS used the RfD recommended for MMM, 0.1 µg/kg body weight per day[38] with an estimate of the 30-year exposure total dose, 6.9E-07 µg/kg body weight per day. The resulting HQ was 6.9E-06. DOE/WRPS also estimated the 70-year exposure total dose of 6.9E-08 µg/kg body weight per day. The resulting HQ was 6.9E-07.[39]

Neurotoxicity HQs in both the 30-year and the 70-year DMM exposure scenarios are far less than one. This indicates there is no neurotoxicity hazard posed by DMM emissions from the tank farm ventilation system upgrades to people in the vicinity of the Hanford facility in public access areas.

5.7. Uncertainty Characterization

Uncertainty may be defined as imperfect knowledge concerning the present and future conditions of a system under consideration. In risk assessments undertaken in support of regulatory decisions, many uncertainties are encountered. Knowledge of these uncertainties allows us to assess the strength of decisions.

Evaluating potential impacts of the project involves several key elements including emission rate assumptions, air dispersion and chemical fate modeling, estimates of resulting environmental concentrations, exposure modeling to estimate received doses, and exposure-response relationships to estimate the possibilities of different types of health impacts. Each of these elements is encumbered by uncertain science and measurement variability that prevent absolute confidence in predictions about adverse health impacts of this project.

[38] NRC, 2000.
[39] RPP-ENV-48231, Part 7.3 ESTIMATION OF TOTAL EXPOSURE, p. 39.

DOE/WRPS evaluated uncertainties in the assessment.[40] To the extent that people may be exposed to DMM emissions from the tank ventilation systems, and despite the uncertainties in concentration estimates, exposure estimates, and neurotoxicity hazard, the potential health risk appears to be acceptable. Quantitative assessments of the effects of tanks ventilation systems emission impacts on human health cannot be made with greater confidence. As in any risk assessment, the current risk assessment involves circumstances of incomplete scientific information. Overall risk uncertainties are summarized in Table 7. The largest sources of uncertainty and variability are:

Table 7. Summary of how the Uncertainty Affects the Quantitative Estimate of Risks or Hazards

Source of Uncertainty	How Does it Affect Estimated Risk From This Project?
Emissions estimates	Likely to overestimate risk
Concentration modeling	Likely to overestimate risk
Exposure assumptions	Likely to overestimate risk
Toxicity of DMM at low dose	Possible overestimate of neurotoxic potency

5.7.1. Emissions Uncertainty and Variability

Emissions uncertainty includes measurement uncertainty and process variability. The emissions factors used to estimate emission rates from the proposed tank ventilation system upgrades are estimates based on concentrations measured in tank headspaces. It was assumed that the three tank farms had DMM at the highest concentration found in all of the tanks, but only 10 tanks have been found to have DMM. Also, the assumption that the DSTs will be mixed for the entire year is probably an exaggeration. They are not likely to be mixed nearly that much, so the continuous-mixing assumption will tend to overestimate the emissions and consequent exposures.

5.7.2. TAP Concentration Modeling Uncertainty

TAP concentration modeling uncertainty results from uncertainties about future meteorology, and the measurement variability and applicability of past meteorological conditions of the air data used for the current analyses. Additionally, TAP concentrations uncertainty arises from uncertainty in the precision and accuracy of the air pollutant dispersion model used—EPA's AERMOD and its associated pre- and post-processors. Therefore, the results of TAP concentration modeling are just as likely to be underestimates as to overestimates, given an accurate mission rate.

[40] RPP-ENV-48231, Part 9.0 UNCERTAINTY, pp. 41–42 explains some of the very conservative assumptions used in the DOE/WRPS's assessment.

5.7.3. Background TAP Concentration Estimates Uncertainties

Background TAP concentration uncertainty results from the apparent discrepancy between reported concentrations of DMM in Seattle relative to Antarctic air. Counter-intuitively, the monitored levels in Seattle were much lower than in Antarctica. This casts doubt on the validity of the reports of background DMM concentrations. The reports may be underestimates or overestimates of actual concentrations.

5.7.4. Exposure Uncertainty

Exposure uncertainty results from potential inaccuracies of assumptions about the time people will spend in various locations.

DOE/WRPS assumed a continuous exposure for a hypothetical resident at the location along Highway 240 where the maximum DMM concentration is most likely to occur. They assumed this resident would routinely consume homegrown produce, and would occupy the location for 30 or 70 years. The need to ensure that uncertainty and variability are addressed is met by ensuring that the maximal exposures are not underestimated. However, this level of exposure overestimates what will occur in all probability. It is difficult to assess the length of time that people will be exposed to DMM emissions.

In addition, a soil half-life of DMM estimate is not available. Therefore, DOE/WRPS used the value for inorganic mercury. This most likely overestimates the soil elimination constant, and yields unrealistically longer DMM persistence. Conversely, the plant uptake factor DOE/WRPS used was based on a soil concentration derived from octanol-water partition and soil organic carbon coefficients for inorganic mercury not for DMM. This likely underestimated the root uptake factor for leafy vegetables. The coefficients from DMM were not available in scientific literature.

5.7.5. Toxicity Uncertainty

Toxicity uncertainty results from potential inaccuracies in the RBC used in the risk characterization. In general, RBCs are based on inherently variable experimental toxicology and epidemiological studies. In the process of developing RBCs, there are uncertainties in the assumptions used to extrapolate these data, especially for chemicals with little or no human exposure-response data. Many RBCs are based on animal studies at high levels of exposure.

DOE/WRPS's characterization of potential neurotoxicity risk involved comparisons of possible exposures to the RBC for MMM under the assumption that its potency is equal to that of DMM. Available evidence suggests DMM is slightly less toxic than MMM, but the data needed to confirm or refute this assumption are nearly nonexistent. The risk-based dose for prevention of neurotoxicity by DMM might be significantly different from the value used in this assessment. Ecology acknowledges this uncertainty but, based on what we do know, exhauster-attributable DMM exposures for humans, including fetuses, are unlikely to result in appreciable risk.

6. CONCLUSION

Treatment of the tank wastes at Hanford is a net benefit to the health of the people in the surrounding communities now and in future generations. The proposed tank transfer ventilation exhausters will be designed with emission controls that are capable of removing nearly all entrained air pollutants. The emissions of DMM necessitated by waste treatment are inconsequential. People at existing nearby residences are very unlikely to suffer adverse health effects from the emissions.

On the basis of Ecology's review of scientific literature, a long-term continuous exposure to DMM that would result in a total daily internal dose of 0.1016-µg/kg-day would pose as little hazard to humans as would the same duration exposure to MMM at its RfD. The RfD was derived from data on mother MMM exposures and consequent neurological deficits in their children by the NRC. The NRC recommended a RfD for MMM of 0.1 µg/kg body weight per day to protect the most sensitive populations, which are developing fetuses.

The relevance of the MMM RfD to DMM is plausible. Once DMM has been absorbed into the body and metabolically demethylated to MMM, its apparent toxic effects are the same as those of MMM. MMM and DMM damage is almost exclusively limited to the nervous system. In human adults, the damage is selective to certain areas of the brain associated with sensory and coordination functions, particularly neurons in the visual cortex and granule cells of the cerebellum. DMM itself is apparently biologically inert but readily absorbed by the body. Following absorption of DMM into the body, most is exhaled; however, some DMM is metabolized to MMM before it can be exhaled.

MMM is the neurotoxic metabolite. Research using mice suggests DMM is readily metabolized to MMM by the placenta and/or by fetuses. Fetuses are more sensitive to MMM than animals at other life stages. Thus, limitation of exposure to pregnant women is of the highest importance.

Currently, there is no numerically defined acceptable limit of noncancer adverse health risks in Chapter 173-460 WAC. However, the amount of increased neurological impairment risk due to the new exhausters is far less than the HQ of one, which indicates that adverse health effects are unlikely to occur as a result of the project.

Based on the above analysis, the increased risks from the proposed project, as a result of exhauster DMM emissions, are permissible because they fall within the limits defined in WAC 173-460-090(7). Provided the exhausters are operated as described in the second tier petition and the NOC application, and health risks are evaluated if any significant increase in DMM or other TAP emissions come to light, the additional health hazards attributable to tank ventilation system upgrades are permissible under Chapter 173-460 WAC. The project review team recommends approval of the proposed project in accordance with WAC 173-460-090(7).

7. LIST OF ACRONYMS AND ABBREVIATIONS

AERMOD	Air dispersion model
ASIL	Acceptable Source Impact Level
ATSDR	Agency for Toxic Substances and Diseases Registry
BACT	Best Available Control Technology
C	Celsius
CASRN	Chemical Abstract Service Registry Number
Conc.	Concentration
CAir	Concentration in air
DMM	Dimethyl mercury
DOE	United States Department of Energy
DST	Double-Shell Tank
Ecology	Washington State Department of Ecology
EPA	United States Environmental Protection Agency
HIA	Health Impact Assessment
HQ	Hazard Quotient
hr	Hour(s)
Max.	Maximum
MIBR	Maximally Impacted Boundary Receptor
MICR	Maximally Impacted Commercial Receptor
$\mu g/m^3$	Micrograms per Cubic Meter
MIRR	Maximally Impacted Residential Receptor
MMM	Monomethyl Mercury
NOC	Notice of Construction Order of Approval
NRC	National Research Council
NWS	National Weather Service
PSD	Prevention of Significant Deterioration
RBC	Risk-Based Concentration
RfC	Reference Concentration
RfD	Reference Dose
RFO	Washington State Department of Ecology–Richland Field Office

SQER	Small Quantity Emission Rate
TAP	Toxic Air Pollutant
tBACT	Best Available Control Technology for Toxics
TWA	Time-weighted Average
UF	Uncertainty Factor
WAC	Washington Administrative Code
WFD	Waste Feed Delivery
WTP	Waste Treatment and Immobilization Plant
WRPS	Washington River Protection Solutions, LLC
y or yr	Year(s)