

APPENDIX M
HUMAN HEALTH RISK ASSESSMENT

**The Oeser Company Superfund Site
Final Human Health Risk Assessment
Bellingham, Washington
TDD: 01-03-0016**

Contract: 68-S0-01-01
April 2002

Region 10

START-2

Superfund Technical Assessment and Response Team

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ADDENDUM

Following receipt of public comments, changes were made to *The Oeser Company Superfund Site Interim Final Human Health Risk Assessment* (HHRA). Comments were incorporated into the text and tables of *The Oeser Company Superfund Site Final Human Health Risk Assessment*; however, no changes were made to the Technical Approach for Risk Assessment (TARA) tables (E & E 2001). Therefore, any new calculations or changes of previous calculations will not be reflected in the TARA tables. To assure transparency in the risk assessment process, this addendum provides an explanation of changes made to the interim final HHRA, including changes to exposure scenarios and associated calculations.

1. Human health risks due to exposure to total petroleum hydrocarbon (TPH) mixtures were revised according to recent Model Toxics Control Act (MTCA) amendments effective August 15, 2001. Risks from dermal and ingestion exposure to TPH in soil were calculated using the Washington State Department of Ecology's *Workbook for Calculating Cleanup Levels for Petroleum Contaminated Sites* version MTCATPH10. Default factors for soil characteristics and fractionated data for TPH in soil were used. The resulting risks and hazard indices are reported in Appendix D. Note that the TARA tables were not updated to reflect the recalculation of risks from exposure to TPH in soil although risks from exposure to TPH are included in Tables 5-1 through 5-8.
2. Exposure to particulates in air was a concern for local residents. Therefore, for each residence, risks calculated for the nearest air station location were added to the risks associated with other residential exposure pathways (dermal, ingestion, vegetable consumption). It should be kept in mind that the risks associated with a particular air station do not reflect accurate risks for surrounding residences. The air monitoring data reflect only a brief period of time and exposures are influenced by wind speed and wind direction, among other factors. Addition of inhalation risks with other residential risks is provided in Figures 5-1 and 5-2. Table AD-1 of this addendum provides a list of which air station result was combined with each residence.
3. It was previously assumed that 50% of the soil contacted by workers was contaminated, on-facility soil. To reflect this assumption, a fractional value of 0.5 was incorporated into the exposure equation for dermal contact with on-facility soil. However, this fractional value has been removed as a result of public comments and to maintain consistency with worker exposure scenarios at other sites in Region 10. Therefore, all worker risks from dermal exposure to on-facility soil are doubled. These changes are shown in Tables 5-1 through 5-8. Note that the TARA tables have not been updated to reflect this change. (No change has been made to the fraction of groundwater ingested by workers.)
4. The conceptual site model has been revised to improve readability and update exposure pathways for workers at the Tilbury Cement Company. Receptors of exposure are now designated for either current or future scenarios and a complete exposure pathway was added for Tilbury Cement Company workers' exposure to groundwater while showering.
5. Table 1 in Appendix A of the final HHRA was revised to maintain consistency with the revised conceptual site model.
6. Dermal exposure to groundwater (while showering) for Tilbury Cement Company workers was added as a complete exposure pathway. Exposure factors and exposure equations for this

ADDENDUM (CONTINUED)

pathway are provided in Table 4.17 in Appendix A of the final HHRA. A summary of the risk calculations is provided in Table 5-10 of the HHRA and further detail is provided in Table AD-2 of this addendum.

7. A table containing screening levels for contaminants in berries is included as Table AD-3 of this addendum.
8. Tables containing dermal absorption factors for soil and groundwater are included as Tables AD-4 and AD-5 of this addendum.
9. A table containing volatilization factors is included as Table AD-6 of this addendum.
10. Air station locations used to calculate risks for workers are shown on Figure C-1 of Appendix C of the HHRA. This figure will not be updated. Locations for off-facility air stations are provided in Figures 2-11 and 2-12 of *The Oeser Company Superfund Site Remedial Investigation Report* (E & E 2002).

Table AD-1

**GROUPING OF AIR STATIONS AND
RESIDENCES FOR RISK ESTIMATION
THE OESER COMPANY
BELLINGHAM, WASHINGTON**

Residence	Air Station
RES-BKG	NA
RES-01	27
RES-03	27
RES-04	27
RES-06	32
RES-09	32
RES-10	32
RES-13	32
RES-14	33
RES-15	33
RES-17	33
RES-19	33
RES-20	33
RES-21A	33
RES-22	33
RES-23	33
RES-24	33
RES-25	33
RES-26	33
RES-29	33
RES-30	24
RES-31	24
RES-33	24
RES-34	24
RES-35	24
RES-37	24
RES-44A	28
RES-58	24

Key:

NA = No air station was applicable to the background location.

RES = Residence.

Table AD-2

**CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
GROUNDWATER
THE OESER COMPANY
BELLINGHAM, WASHINGTON**

Scenario Timeframe: Current/Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Deep Aquifer--Tap Water
Receptor Population: Worker
Receptor Age: Worker

Exposure Route	Location	Analyte	Medium EPC	Medium EPC Units	Intake (cancer)	Intake (cancer) Units	Slope Factor	Slope Factor Units	Risk
Ingestion	TC-5	2,3,7,8-TCDD TEQ	8.91E-06	ug/L	3.12E-11	mg/kg-day	1.50E+05	1/(mg/kg-day)	4.67E-06
Ingestion	TC-5	B(a)P Equivalent	1.33E-01	ug/L	4.66E-07	mg/kg-day	7.30E+00	1/(mg/kg-day)	3.40E-06
		Subtotal							8.1E-06
Dermal	TC-5	2,3,7,8-TCDD TEQ	8.91E-06	ug/L	8.97E-10	mg/kg-day	3.00E+05	1/(mg/kg-day)	2.69E-04
Dermal	TC-5	B(a)P Equivalent	1.33E-01	ug/L	1.45E-05	mg/kg-day	8.20E+00	1/(mg/kg-day)	1.19E-04
		Subtotal							3.9E-04
		Total							4.0E-04
Ingestion	TC-6	2,3,7,8-TCDD TEQ	2.95E-06	ug/L	1.03E-11	mg/kg-day	1.50E+05	1/(mg/kg-day)	1.54E-06
Ingestion	TC-6	B(a)P Equivalent	1.36E-01	ug/L	4.76E-07	mg/kg-day	7.30E+00	1/(mg/kg-day)	3.48E-06
		Subtotal							5.0E-06
Dermal	TC-6	2,3,7,8-TCDD TEQ	2.95E-06	ug/L	2.97E-10	mg/kg-day	3.00E+05	1/(mg/kg-day)	8.90E-05
Dermal	TC-6	B(a)P Equivalent	1.36E-01	ug/L	1.48E-05	mg/kg-day	8.20E+00	1/(mg/kg-day)	1.22E-04
		Subtotal							2.1E-04
		Total							2.2E-04

Uses same format as TARA Table 8.1

Table AD-3

**RISK-BASED SCREENING LEVELS FOR BERRIES
THE OESER COMPANY
BELLINGHAM, WASHINTONG**

Analyte	Oral Slope Factor	Berry risk RBC	Oral Reference Dose	Berry HQ RBC
1,2,3,4,6,7,8-HpCDD	1.50E+03	2.93E-05	NA	NA
1,2,3,4,6,7,8-HpCDF	1.50E+03	2.93E-05	NA	NA
1,2,3,4,7,8,9-HpCDF	1.50E+03	2.93E-05	NA	NA
1,2,3,4,7,8-HxCDD	1.50E+04	2.93E-06	NA	NA
1,2,3,4,7,8-HxCDF	1.50E+04	2.93E-06	NA	NA
1,2,3,6,7,8-HxCDD	1.50E+04	2.93E-06	NA	NA
1,2,3,6,7,8-HxCDF	1.50E+04	2.93E-06	NA	NA
1,2,3,7,8,9-HxCDD	1.50E+04	2.93E-06	NA	NA
1,2,3,7,8,9-HxCDF	1.50E+04	2.93E-06	NA	NA
1,2,3,7,8-PeCDD	1.50E+05	2.93E-07	NA	NA
1,2,3,7,8-PeCDF	7.50E+03	5.85E-06	NA	NA
1,2,4-Trimethylbenzene	NA	NA	5.00E-02	9.41E+01
1,3,5-Trimethylbenzene	NA	NA	5.00E-02	9.41E+01
2,3,4,6,7,8-HxCDF	1.50E+04	2.93E-06	NA	NA
2,3,4,7,8-PeCDF	7.50E+04	5.85E-07	NA	NA
2,3,7,8-TCDD	1.50E+05	2.93E-07	NA	NA
2,3,7,8-TCDD (dioxin)	1.50E+05	2.93E-07	NA	NA
2,3,7,8-TCDD TEQ	1.50E+05	2.93E-07	NA	NA
2,3,7,8-TCDF	1.50E+04	2.93E-06	NA	NA
2-Methylnaphthalene	NA	NA	2.00E-02	3.76E+01
Acenaphthene	NA	NA	6.00E-02	1.13E+02
B(a)P Equivalent	7.30E+00	6.01E-03	NA	NA
Benzene	5.50E-02	7.98E-01	1.00E-03	1.88E+00
Benzidine	2.30E+02	1.91E-04	3.00E-03	5.65E+00
Benzo(a)anthracene	7.30E-01	6.01E-02	NA	NA
Benzo(a)pyrene	7.30E+00	6.01E-03	NA	NA
Benzo(b)fluoranthene	7.30E-01	6.01E-02	NA	NA
Benzo(g,h,i)perylene	NA	NA	3.00E-02	5.65E+01
Benzo(j)fluoranthene	7.30E-01	6.01E-02	NA	NA
Benzo(k)fluoranthene	7.30E-02	6.01E-01	NA	NA
Carbazole	2.00E-02	2.20E+00	NA	NA
Chrysene	7.30E-03	6.01E+00	NA	NA
Dibenz(a,h)anthracene	7.30E+00	6.01E-03	NA	NA
Dibenzo(a,e)pyrene	7.30E+00	6.01E-03	NA	NA
Dibenzo(a,h)acridine	7.30E-01	6.01E-02	NA	NA
Dibenzo(a,h)anthracene	7.30E+00	6.01E-03	NA	NA
Dibenzo(a,h)pyrene	7.30E+01	6.01E-04	NA	NA
Dibenzo(a,i)pyrene	7.30E+01	6.01E-04	NA	NA
Dibenzo(a,j)acridine	7.30E-01	6.01E-02	NA	NA
Dibenzo(a,l)pyrene	7.30E+01	6.01E-04	NA	NA
Dibenzofuran	NA	NA	4.00E-03	7.53E+00
Fluoranthene	NA	NA	4.00E-02	7.53E+01
Fluorene	NA	NA	4.00E-02	7.53E+01
Indeno(1,2,3-cd)pyrene	7.30E-01	6.01E-02	NA	NA
Naphthalene	NA	NA	2.00E-02	3.76E+01
OCDD	1.50E+01	2.93E-03	NA	NA
OCDF	1.50E+01	2.93E-03	NA	NA
Pentachlorophenol	1.20E-01	3.66E-01	3.00E-02	5.65E+01
Phenanthrene	NA	NA	3.00E-01	5.65E+02
Polychlorinated biphenyls (PCBs)	2.00E+00	2.20E-02	NA	NA
Pyrene	NA	NA	3.00E-02	5.65E+01
sec-Butylbenzene	NA	NA	1.00E-02	1.88E+01

Key:

NA = Not applicable.

Table AD-4

**SOIL DERMAL ABSORPTION FACTORS
THE OESER COMPANY
BELLINGHAM, WASHINGTON**

Analyte	Absorption Factor
Acenaphthene	0.13
B(a)P Equivalent	0.13
Benzene	0.0005
Benzidine	0.1
Benzo(a)anthracene	0.13
Benzo(a)pyrene	0.13
Benzo(b)fluoranthene	0.13
Benzo(j)fluoranthene	0.13
Benzo(k)fluoranthene	0.13
sec-Butylbenzene	0.0005
Carbazole	0.1
Chrysene	0.13
Dibenzo(a,e)pyrene	0.13
Dibenzo(a,h)acridine	0.13
Dibenzo(a,h)anthracene	0.13
Dibenzo(a,h)pyrene	0.13
Dibenzo(a,i)pyrene	0.13
Dibenzo(a,j)acridine	0.13
Dibenzo(a,l)pyrene	0.13
7H-Dibenzo(c,g)carbazole	0.13
Dibenzofuran	0.03
7,12-Dimethylbenz(a)anthracene	0.13
Fluoranthene	0.13
Fluorene	0.13
Indeno(1,2,3-cd)pyrene	0.13
2-Methylnaphthalene	0.13
Naphthalene	0.13
Pentachlorophenol	0.25
Phenanthrene	0.13
n-Propylbenzene	0.0005
Pyrene	0.13
1,2,4-Trimethylbenzene	0.0005
1,3,5-Trimethylbenzene	0.0005
Dioxin TEQ	0.03
2,3,7,8-TCDD	0.03
2,3,7,8-TCDF	0.03
1,2,3,7,8-PeCDD	0.03
1,2,3,7,8-PeCDF	0.03
2,3,4,7,8-PeCDF	0.03
1,2,3,4,7,8-HxCDD	0.03
1,2,3,4,7,8-HxCDF	0.03
1,2,3,6,7,8-HxCDD	0.03
1,2,3,6,7,8-HxCDF	0.03
1,2,3,7,8,9-HxCDD	0.03
1,2,3,7,8,9-HxCDF	0.03
2,3,4,6,7,8-HxCDF	0.03
1,2,3,4,6,7,8-HpCDD	0.03
1,2,3,4,6,7,8-HpCDF	0.03
1,2,3,4,7,8,9-HpCDF	0.03
OCDD	0.03
OCDF	0.03

Table AD-5

**GROUNDWATER DERMAL ABSORPTION FACTORS
THE OESER COMPANY
BELLINGHAM, WASHINGTON**

Analyte	CAS Number	Kp Predicted (cm/hr)	B	t (hr/event)	t* (hr)	FA
1,1,1-Trichloroethane	71556	1.30E-02	0.1	5.90E-01	1.41E+00	1
1,1,2,2-Tetrachloroethane	79345	7.30E-03	0	9.20E-01	2.20E+00	1
1,1,2-Trichloroethane	79005	6.70E-03	0	5.90E-01	1.41E+00	1
1,2,3,4,6,7,8-HpCDD		9.00E-01	6.2	6.68E+00	2.97E+01	0.5
1,2,3,4,6,7,8-HpCDF		9.00E-01	6.2	6.68E+00	2.97E+01	0.5
1,2,3,6,7,8-HxCDD		9.00E-01	6.2	6.68E+00	2.97E+01	0.5
1,2,3,7,8,9-HxCDD		9.00E-01	6.2	6.68E+00	2.97E+01	0.5
1,2,4-Trichlorobenzene	120821	7.10E-02	0.4	1.09E+00	2.62E+00	1
1,2-Dichloropropane	78875	8.10E-03	0	4.50E-01	1.08E+00	1
1,3-Dichloropropene	542756	4.50E-03	0	4.40E-01	1.06E+00	1
1-Methylnaphthalene		4.90E-02	0.2	5.50E-01	1.32E+00	1
2,3,7,8-TCDD TEQ		9.00E-01	6.2	6.68E+00	2.97E+01	0.5
2,4,6-Trichlorophenol	88062	3.70E-02	0.2	1.34E+00	3.22E+00	1
2,4-Dichlorophenol	120832	1.80E-02	0.1	8.60E-01	2.06E+00	1
2-amino-4-Nitrophenol	99570	1.80E-03	0	7.70E-01	1.84E+00	1
2-Methylnaphthalene		4.90E-02	0.2	5.50E-01	1.32E+00	1
2-Nitrophenol	88755	4.20E-03	0	6.30E-01	1.52E+00	1
3-Nitrophenol	554847	5.80E-03	0	6.30E-01	1.52E+00	1
4,4'-Thiodianiline	139651	2.20E-03	0	1.70E+00	4.09E+00	1
4-Nitrophenol	100027	5.00E-03	0	6.30E-01	1.52E+00	1
Acetaldehyde	75070	6.40E-04	0	1.90E-01	4.50E-01	1
Acetamide	60355	1.10E-04	0	2.30E-01	5.40E-01	1
Acetylaminofluorene, 2-	53963	1.30E-02	0.1	1.86E+00	4.48E+00	1
Acrolein	107028	6.60E-04	0	2.20E-01	5.20E-01	1
Acrylamide	79061	2.30E-04	0	2.60E-01	6.30E-01	1
Acrylonitrile	107131	1.20E-03	0	2.10E-01	5.00E-01	1
Aldrin	309002	1.50E-03	0	1.16E+01	2.79E+01	1
Allyl chloride	107051	5.50E-03	0	2.80E-01	6.80E-01	1
Amino-2-methylanthraquinone, 1-	82280	5.60E-03	0	2.24E+00	5.38E+00	1
Aminoanthraquinone, 2-	117793	2.50E-03	0	1.86E+00	4.48E+00	1
Aminoazobenzene, p-	60093	7.10E-03	0	1.33E+00	3.20E+00	1
Aminoazotoluene, o-	97563	3.70E-02	0.2	1.92E+00	4.61E+00	1
Aminobiphenyl, 4-	92671	1.30E-02	0.1	9.30E-01	2.24E+00	1
Aniline	62533	1.90E-03	0	3.50E-01	8.40E-01	1
Anisidine, o-	90040	1.50E-03	0	6.80E-01	1.64E+00	1
Auramine	492808	1.20E-02	0.1	3.30E+00	7.93E+00	1
B(a)P Equivalent		7.70E-01	4.7	2.64E+00	1.15E+01	1
Benzene	71432	1.50E-02	0.1	2.90E-01	6.90E-01	1
Benzidine	92875	1.20E-03	0	1.13E+00	2.71E+00	1
Benzo(a)anthracene	56553	5.20E-01	3	2.00E+00	8.44E+00	1
Benzo(a)pyrene	50328	7.70E-01	4.7	2.64E+00	1.15E+01	1
Benzo(b)fluoranthene	205992	7.80E-01	4.7	2.72E+00	1.19E+01	1
Benzoic acid	65850	5.90E-03	0	5.10E-01	1.22E+00	1
Benzotrichloride	98077	1.20E-02	0.1	1.30E+00	3.12E+00	1
Benzyl chloride	100447	1.10E-02	0	5.40E-01	1.30E+00	1
Bis(2-chloroethyl)ether	111444	1.80E-03	0	6.60E-01	1.60E+00	1
Bromodichloromethane	75274	4.80E-03	0	8.70E-01	2.09E+00	1

Table AD-5

**GROUNDWATER DERMAL ABSORPTION FACTORS
THE OESER COMPANY
BELLINGHAM, WASHINGTON**

Analyte	CAS Number	Kp Predicted (cm/hr)	B	t (hr/event)	t* (hr)	FA
Bromoform	75252	2.40E-03	0	2.74E+00	6.57E+00	1
Bromomethane	74839	2.90E-03	0	3.60E-01	8.60E-01	1
Bromophenol, p-	106412	1.00E-02	0.1	9.80E-01	2.35E+00	1
Butadiene, 1,3-	106990	1.70E-02	0	2.10E-01	5.10E-01	1
Butanediol, 2,3-	513859	1.20E-04	0	3.40E-01	8.10E-01	1
Butanol, n-	71363	1.70E-03	0	2.70E-01	6.60E-01	1
Butoxyethanol, 2-	111762	1.20E-03	0	4.80E-01	1.16E+00	1
Captan	133062	1.20E-03	0	5.03E+00	1.21E+01	1
Carbon disulfide	75150	1.80E-02	0.1	3.00E-01	7.10E-01	1
Carbon tetrachloride	56235	1.70E-02	0.1	7.60E-01	1.83E+00	1
Chlordane	57749	4.10E-02	0.3	2.07E+01	4.98E+01	0.7
Chlordane (cis)	5103719	3.70E-02	0.3	2.08E+01	4.99E+01	0.7
Chlordane (trans)	5103742	3.70E-02	0.3	2.08E+01	4.99E+01	0.7
Chlorobenzene	108907	3.00E-02	0.1	4.50E-01	1.08E+00	1
Chlorocresol	59507	3.00E-02	0.1	6.60E-01	1.59E+00	1
Chlorodibromomethane	124481	3.40E-03	0	1.54E+00	3.70E+00	1
Chloroethane	75003	6.30E-03	0	2.40E-01	5.80E-01	1
Chloroform	67663	7.10E-03	0	4.90E-01	1.18E+00	1
Chloromethane	74873	3.40E-03	0	2.00E-01	4.80E-01	1
Chlorophenol, o-	95578	8.50E-03	0	5.50E-01	1.33E+00	1
Chlorophenol, p-	106489	1.20E-02	0.1	5.50E-01	1.33E+00	1
Chlorothalonil	1897456	2.00E-02	0.1	3.24E+00	7.78E+00	0.9
Chrysene	218019	5.20E-01	3	2.00E+00	8.44E+00	1
Cresidine, p-	120718	3.60E-03	0	6.20E-01	1.48E+00	1
Cresol, m-	108394	8.10E-03	0	4.20E-01	1.02E+00	1
Cresol, o-	95487	8.00E-03	0	4.20E-01	1.02E+00	1
Cresol, p-	106445	7.80E-03	0	4.20E-01	1.02E+00	1
DDD	72548	2.00E-01	1.4	6.51E+00	2.57E+01	0.8
DDE	72559	1.70E-01	1.2	6.35E+00	2.47E+01	0.8
DDT	50293	3.00E-01	2.2	1.02E+01	4.20E+01	0.7
Decanol	112301	1.20E-01	0.6	8.10E-01	1.94E+00	1
Di-2-ethylhexyl phthalate	117817	2.70E-02	0.2	1.63E+01	3.91E+01	0.8
Diaminoanisole, 2,4-	615054	2.20E-04	0	6.20E-01	1.50E+00	1
Diaminotoluene	95807	5.60E-04	0	5.10E-01	1.22E+00	1
Diaminotoluene, 2,4-	101804	2.90E-03	0	1.39E+00	3.33E+00	1
Dibenzo(a,h)anthracene	53703	1.70E+00	10.7	3.81E+00	1.73E+01	0.6
Dibutyl phthalate	84742	2.60E-02	0.2	3.79E+00	9.10E+00	0.9
Dichlorobenzene, 1,2-	95501	4.40E-02	0.2	7.00E-01	1.68E+00	1
Dichlorobenzene, 1,3-	541731	6.10E-02	0.3	7.00E-01	1.68E+00	1
Dichlorobenzene, 1,4-	106467	4.40E-02	0.2	7.00E-01	1.68E+00	1
Dichlorobenzidine, 3,3'	91941	1.40E-02	0.1	2.75E+00	6.60E+00	1
Dichlorodifluoromethane	75718	9.30E-03	0	5.00E-01	1.20E+00	1
Dichloroethane, 1,1-	75343	7.00E-03	0	3.80E-01	9.00E-01	1
Dichloroethane, 1,2-	107062	4.30E-03	0	3.80E-01	9.00E-01	1
Dichloroethylene, 1,1-	75354	1.20E-02	0	3.70E-01	8.80E-01	1
Dichloroethylene, 1,2- (trans)	540590	8.00E-03	0	3.70E-01	8.80E-01	1
Dichlorvos	62737	8.90E-04	0	1.82E+00	4.36E+00	1

Table AD-5

**GROUNDWATER DERMAL ABSORPTION FACTORS
THE OESER COMPANY
BELLINGHAM, WASHINGTON**

Analyte	CAS Number	Kp Predicted (cm/hr)	B	t (hr/event)	t* (hr)	FA
Dieldrin	60571	1.30E-02	0.1	1.43E+01	3.43E+01	0.8
Diepoxybutane	1464535	3.10E-05	0	3.20E-01	7.70E-01	1
Diethyl phthalate	84662	4.10E-03	0	1.84E+00	4.42E+00	1
Diethyl sulfate	64675	1.30E-03	0	7.70E-01	1.84E+00	1
Dimethoxybenzidine, 3,3'-	119904	9.70E-04	0	2.80E+00	6.71E+00	1
Dimethyl phthalate	131113	1.40E-03	0	1.28E+00	3.08E+00	1
Dimethyl sulfate	77781	1.90E-03	0	5.30E-01	1.28E+00	1
Dimethylamine, n-nitroso-	62759	2.50E-04	0	2.70E-01	6.60E-01	1
Dimethylaminoazobenzene, 4-	60117	1.00E-01	0.6	1.91E+00	4.59E+00	1
Dimethylbenzidine, 3,3'-	119937	3.80E-03	0	1.62E+00	3.90E+00	1
Dimethylcarbamyl chloride	79447	4.00E-04	0	4.20E-01	1.01E+00	1
Dimethylhydrazine, 1,1-	57147	7.20E-05	0	2.30E-01	5.50E-01	1
Dimethylphenol, 2,4-	105679	1.10E-02	0	5.10E-01	1.22E+00	1
Dimethylphenol, 3,4-	95658	1.00E-02	0	5.10E-01	1.22E+00	1
Dinitrophenol, 2,4-	51285	1.60E-03	0	1.13E+00	2.71E+00	1
Dinitrotoluene, 2,4-	121142	3.20E-03	0	1.10E+00	2.64E+00	1
Dinitrotoluene, 2,6-	606202	2.20E-03	0	1.10E+00	2.64E+00	1
Dioxane, 1,4-	123911	3.40E-04	0	3.30E-01	7.90E-01	1
Diphenylamine, n-nitroso-	86306	1.50E-02	0.1	1.35E+00	3.25E+00	1
Diphenylhydrazine, 1,2-	122667	1.40E-02	0.1	1.13E+00	2.71E+00	1
Dipropylamine, n-nitroso-	621647	2.40E-03	0	5.60E-01	1.35E+00	1
Endrin	72208	1.30E-02	0.1	1.43E+01	3.43E+01	0.8
Epichlorohydrin	106898	3.50E-04	0	3.40E-01	8.30E-01	1
Ethanol	64175	5.40E-04	0	1.90E-01	4.60E-01	1
Ethanol, 2-(2-butoxyethoxy)-	112345	4.70E-05	0	8.50E-01	2.04E+00	1
Ethanol, 2-(2-ethoxyethoxy)-	111900	2.50E-04	0	5.90E-01	1.42E+00	1
Ethanol, 2-(2-methoxyethoxy)-	111773	1.80E-04	0	4.90E-01	1.19E+00	1
Ethoxyethanol, 2-	110805	4.30E-04	0	3.40E-01	8.10E-01	1
Ethoxyethyl acetate, 2-	111159	7.90E-04	0	5.80E-01	1.38E+00	1
Ethyl acrylate	140885	3.30E-03	0	3.80E-01	9.20E-01	1
Ethyl carbamate	51796	4.00E-04	0	3.30E-01	8.00E-01	1
Ethyl ether	60297	2.40E-03	0	2.70E-01	6.60E-01	1
Ethylbenzene	100414	5.20E-02	0.2	4.10E-01	9.90E-01	1
Ethylene oxide	75218	5.60E-04	0	1.90E-01	4.50E-01	1
Ethylenedibromide	106934	2.90E-03	0	1.19E+00	2.85E+00	1
Ethyleneimine	151564	1.60E-04	0	1.80E-01	4.40E-01	1
Ethylenethiourea	96457	1.70E-04	0	3.60E-01	8.70E-01	1
Ethylphenol, p-	123079	1.10E-02	0	4.90E-01	1.19E+00	1
Fluoranthene	206440	2.40E-01	1.3	1.43E+00	5.61E+00	1
Formaldehyde	50000	1.80E-03	0	1.50E-01	3.70E-01	1
Glycerol	56815	3.20E-05	0	3.40E-01	8.30E-01	1
Heptachlor	76448	9.30E-03	0.1	1.30E+01	3.12E+01	0.8
Heptanol	111706	1.50E-02	0.1	4.70E-01	1.13E+00	1
Hexachlorobenzene	118741	1.50E-01	0.9	4.14E+00	1.59E+01	0.9
Hexachlorobutadiene	87683	8.80E-02	0.5	3.04E+00	7.29E+00	0.9
Hexachloroethane	67721	3.20E-02	0.2	2.23E+00	5.34E+00	1
Hexamethylphosphoramide	680319	1.70E-04	0	1.06E+00	2.54E+00	1

Table AD-5

**GROUNDWATER DERMAL ABSORPTION FACTORS
THE OESER COMPANY
BELLINGHAM, WASHINGTON**

Analyte	CAS Number	Kp Predicted (cm/hr)	B	t (hr/event)	t* (hr)	FA
Hexanol	111273	9.70E-03	0	3.90E-01	9.40E-01	1
Hydrazine/Hydrazine sulfate	302012	4.30E-05	0	1.60E-01	3.80E-01	1
Indeno(1,2,3-cd)pyrene	193395	1.20E+00	7.4	3.71E+00	1.66E+01	0.6
Isophorone	78591	3.50E-03	0	6.20E-01	1.50E+00	1
Lindane	58899	1.20E-02	0.1	4.48E+00	1.08E+01	0.9
m-Xylene	108383	5.60E-02	0.2	4.10E-01	9.90E-01	1
Mechlorethamine	51752	1.10E-03	0	7.90E-01	1.89E+00	1
Methanol	67561	3.20E-04	0	1.60E-01	3.80E-01	1
Methoxyethanol, 2-	109864	1.80E-04	0	2.80E-01	6.70E-01	1
Methoxypropan-2-ol, 1-	107982	3.80E-04	0	3.40E-01	8.10E-01	1
Methyl ethyl ketone	78933	9.80E-04	0	2.70E-01	6.40E-01	1
Methyl hydroxybenzoate	99763	4.30E-03	0	7.50E-01	1.79E+00	1
Methyl iodide	74884	2.60E-03	0	6.60E-01	1.57E+00	1
Methylaziridine, 2-	75558	3.00E-04	0	2.20E-01	5.30E-01	1
Methylene chloride	75092	3.60E-03	0	3.10E-01	7.50E-01	1
Methylenedianiline, 4,4'-	101779	1.40E-03	0	1.35E+00	3.24E+00	1
Michler's ketone	90948	2.60E-02	0.2	3.35E+00	8.03E+00	0.9
Mustard Gas	505602	4.70E-03	0	8.20E-01	1.96E+00	1
Naphthalene	91203	4.90E-02	0.2	5.50E-01	1.32E+00	1
Naphthol, b-	135193	2.00E-02	0.1	6.80E-01	1.62E+00	1
Naphthylamine, 1-	134327	8.00E-03	0	6.70E-01	1.60E+00	1
Naphthylamine, 2-	91598	8.40E-03	0	6.70E-01	1.60E+00	1
Nitrotriactic acid	139139	1.00E-04	0	1.23E+00	2.96E+00	1
Nitro-o-anisidine, 5-	99592	2.10E-03	0	7.50E-01	1.81E+00	1
Nitrobiphenyl, 4-	92933	4.10E-02	0.2	1.37E+00	3.29E+00	1
Nitrofen	1836755	2.10E-01	1.3	4.10E+00	1.61E+01	0.9
Nitrophenol, 4-amino-2-	119346	9.60E-04	0	7.70E-01	1.84E+00	1
Nitropropane, 2-	79469	9.00E-04	0	4.30E-01	1.04E+00	1
Nitroso-di-n-butylamine, n-	924163	4.00E-03	0	8.10E-01	1.94E+00	1
Nitroso-N-ethylurea, n-	759739	5.00E-04	0	4.80E-01	1.14E+00	1
Nitroso-N-methylurea, n-	684935	4.00E-04	0	4.00E-01	9.50E-01	1
Nitrosodiethanolamine, n-	1116547	2.50E-05	0	5.90E-01	1.42E+00	1
Nitrosodiethylamine, n-	55185	1.10E-03	0	3.30E-01	7.80E-01	1
Nitrosodiphenylamine, p-	156105	2.70E-02	0.1	1.35E+00	3.25E+00	1
Nitrosomethylvinylamine, n-	4549400	5.20E-04	0	3.20E-01	7.70E-01	1
Nitrosomorpholine, n-	59892	1.80E-04	0	4.70E-01	1.13E+00	1
Nitrosornicotine, n-	16543558	1.70E-04	0	1.03E+00	2.48E+00	1
Nitrosopiperidine, n-	100754	3.00E-05	0	9.62E+00	2.31E+01	1
Nonanol	143088	5.20E-02	0.2	6.70E-01	1.62E+00	1
o-Toluidine	95534	3.10E-03	0	4.20E-01	1.00E+00	1
o-Toluidine hydrochloride	636215	1.80E-03	0	6.70E-01	1.60E+00	1
OCDD		9.00E-01	6.2	6.68E+00	2.97E+01	0.5
OCDF		9.00E-01	6.2	6.68E+00	2.97E+01	0.5
Octanol	111875	2.90E-02	0.1	5.60E-01	1.35E+00	1
Parathion	56382	1.40E-02	0.1	4.48E+00	1.08E+01	0.9
PCB-chlorobiphenyl, 4-	2051629	8.30E-01	5.5	4.54E+00	2.00E+01	0.6
PCB-hexachlorobiphenyl	26601649	4.80E-01	3.5	1.11E+01	4.73E+01	0.5

Table AD-5

**GROUNDWATER DERMAL ABSORPTION FACTORS
THE OESER COMPANY
BELLINGHAM, WASHINGTON**

Analyte	CAS Number	Kp Predicted (cm/hr)	B	t (hr/event)	t* (hr)	FA
Pentachloronitrobenzene	82688	4.50E-02	0.3	4.74E+00	1.14E+01	0.9
Pentachlorophenol	87865	4.30E-01	2.7	3.26E+00	1.37E+01	0.9
Pentanol	71410	5.70E-03	0	3.30E-01	7.80E-01	1
Pentanone, 4-methyl-2-	108101	2.70E-03	0	3.80E-01	9.20E-01	1
Phenanthrene	85018	1.50E-01	0.8	1.05E+00	4.03E+00	1
Phenol	108952	4.50E-03	0	3.50E-01	8.50E-01	1
Phenol, 4,6-dinitro-2-methyl-	534521	3.20E-03	0	1.35E+00	3.25E+00	1
Propanol	71238	1.20E-03	0	2.30E-01	5.50E-01	1
Propiolactone, beta-	57578	3.10E-04	0	2.70E-01	6.40E-01	1
Propylene oxide	75569	7.80E-04	0	2.20E-01	5.30E-01	1
Resorcinol	108463	1.30E-03	0	4.30E-01	1.04E+00	1
Safrole	94597	1.20E-02	0.1	8.50E-01	2.04E+00	1
Styrene	100425	3.90E-02	0.2	4.00E-01	9.70E-01	1
Styrene oxide	96093	4.00E-03	0	4.90E-01	1.19E+00	1
TCDD	1746016	9.00E-01	6.2	6.68E+00	2.97E+01	0.5
Tetrachlorethylene	127184	3.50E-02	0.2	8.90E-01	2.14E+00	1
Thioacetamide	62555	1.80E-03	0	2.80E-01	6.60E-01	1
Thiourea	62566	1.40E-04	0	2.80E-01	6.70E-01	1
Thymol	89838	3.70E-02	0.2	7.30E-01	1.75E+00	1
Toluene	108883	3.30E-02	0.1	3.40E-01	8.30E-01	1
Toxaphene	8001352	1.30E-02	0.1	2.19E+01	5.25E+01	0.8
Trichloroethylene	79016	1.20E-02	0.1	5.70E-01	1.37E+00	1
Trichlorofluoromethane	75694	1.30E-02	0.1	6.20E-01	1.48E+00	1
Tris(2,3-dibromopropyl)phosphate	126727	4.30E-04	0	8.48E+02	2.04E+03	1
Tris(aziridiny)-para-benzoquinone	68768	1.00E-05	0	2.07E+00	4.98E+00	1
Urea	57136	2.80E-05	0	2.30E-01	5.50E-01	1
Vinyl bromide	593602	4.50E-03	0	4.20E-01	1.00E+00	1
Vinyl chloride	75014	5.80E-03	0	2.40E-01	5.70E-01	1
Water	7732185	1.50E-04	0	1.30E-01	3.20E-01	1

Key:

B - Ratio of the permeability coefficient of a compound through the stratum corneum relative to its permeability coefficient across the viable epidermis (dimensionless)

FA - Fraction Absorbed (dimensionless)

Kp Predicted - Dermal permeability coefficient of compound in water

t - Event duration (hr/event)

t* - Time to reach steady-state (hr)

Table AD-6

**CHEMICAL-SPECIFIC VOLATILIZATION FACTORS
THE OESER COMPANY
BELLINGHAM, WASHINGTON**

Analyte	VF
Acenaphthene	1.80E+05
B(a)P Equivalent	NA
Benzene	2.80E+03
Benzidine	NA
Benzo(a)anthracene	NA
Benzo(a)pyrene	NA
Benzo(b)fluoranthene	NA
Benzo(j)fluoranthene	NA
Benzo(k)fluoranthene	NA
sec-Butylbenzene	8.30E+03
Carbazole	NA
Chrysene	2.70E+06
Dibenzo(a,e)pyrene	NA
Dibenzo(a,h)acridine	NA
Dibenzo(a,h)anthracene	NA
Dibenzo(a,h)pyrene	NA
Dibenzo(a,i)pyrene	NA
Dibenzo(a,j)acridine	NA
Dibenzo(a,l)pyrene	NA
7H-Dibenzo(c,g)carbazole	NA
Dibenzofuran	6.50E+05
7,12-Dimethylbenz(a)anthracene	NA
Fluoranthene	NA
Fluorene	2.70E+05
Indeno(1,2,3-cd)pyrene	NA
2-Methylnaphthalene	4.30E+04
Naphthalene	4.30E+04
Pentachlorophenol	NA
Phenanthrene	NA
n-Propylbenzene	1.10E+04
Pyrene	3.10E+06
1,2,4-Trimethylbenzene	2.00E+04
1,3,5-Trimethylbenzene	8.00E+03
Dioxin TEQ	NA
2,3,7,8-TCDD	NA
2,3,7,8-TCDF	NA
1,2,3,7,8-PeCDD	NA
1,2,3,7,8-PeCDF	NA
2,3,4,7,8-PeCDF	NA
1,2,3,4,7,8-HxCDD	NA
1,2,3,4,7,8-HxCDF	NA
1,2,3,6,7,8-HxCDD	NA
1,2,3,6,7,8-HxCDF	NA
1,2,3,7,8,9-HxCDD	NA
1,2,3,7,8,9-HxCDF	NA
2,3,4,6,7,8-HxCDF	NA
1,2,3,4,6,7,8-HpCDD	NA
1,2,3,4,6,7,8-HpCDF	NA
1,2,3,4,7,8,9-HpCDF	NA
OCDD	NA
OCDF	NA

Key:

NA = Not applicable.

**THE OESER COMPANY SUPERFUND SITE
FINAL HUMAN HEALTH RISK ASSESSMENT
BELLINGHAM, WASHINGTON**

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LIST OF ACRONYMS

<u>Acronym</u>	<u>Definition</u>
B(a)P	benzo(a)pyrene
bgs	below ground surface
BTEX	benzene, toluene, ethylbenzene, and xylenes
CalEPA	California Environmental Protection Agency
CDIs	chronic daily intakes
COCs	contaminants of concern
COPCs	contaminants of potential concern
CPAHs	carcinogenic polycyclic aromatic hydrocarbons
CSM	conceptual site model
CT	central tendency
DL	detection limit
E & E	Ecology and Environment, Inc.
Ecology	Washington State Department of Ecology
ED	exposure duration
EFH	<i>Exposure Factors Handbook</i>
EPA	United States Environmental Protection Agency
EPCs	exposure point concentrations
EPHs	extractable petroleum hydrocarbons
FS	feasibility study
g/day	grams per day
g/kg BW-day	gram per kilogram body weight per day
HEAST	Health Effects Assessment Summary Tables
HHRA	human health risk assessment
HI	hazard indices
HQ	hazard quotient
IRIS	Integrated Risk Information System
kg	kilogram
LADIs	lifetime average daily intakes
MF	modifying factor

LIST OF ACRONYMS (CONTINUED)

<u>Acronym</u>	<u>Definition</u>
$\mu\text{g}/\text{m}^3$	micrograms per cubic meter
m^3/day	cubic meters per day
mg/m^3	milligrams per cubic meter
mg/kg	milligrams per kilogram
MTCA	Model Toxics Control Act
NCEA	National Center for Environmental Assessment
OSWER	Office of Solid Waste and Emergency Response
PAHs	polycyclic aromatic hydrocarbons
PCDD	polychlorinated dibenzo-p-dioxin
PCDF	polychlorinated dibenzofuran
PCP	pentachlorophenol
PEF	particulate emission factor
PRGs	Preliminary Remediation Goals
Q/C	inverse of the mean concentration at the center of a square source
RAGS	Risk Assessment Guidance for Superfund
RfCs	reference concentrations
RfD	reference dose
RfDi	inhalation reference dose
RME	reasonable maximum exposure
RPF	relative potency factor
RI	remedial investigation
SFs	slope factors
TCDD	tetrachlorodibenzo-p-dioxin
TARA	Technical Approach for Risk Assessment
TEF	toxicity equivalency factor
TEQ	toxicity equivalent
TICs	tentatively identified compounds
TPH	total petroleum hydrocarbon
TPH-D	total petroleum hydrocarbon as diesel

LIST OF ACRONYMS (CONTINUED)

<u>Acronym</u>	<u>Definition</u>
TPH-G	total petroleum hydrocarbon as gasoline
UCL	upper confidence limit
UFs	uncertainty factors
VF	volatilization factor
VOCs	volatile organic compounds
VPHs	volatile petroleum hydrocarbons
WAC	Washington Administrative Code
WHO	World Health Organization

**THE OESER COMPANY SUPERFUND SITE
FINAL HUMAN HEALTH RISK ASSESSMENT
BELLINGHAM, WASHINGTON**

1. INTRODUCTION

This baseline human health risk assessment (HHRA) is a component of the remedial investigation (RI) for The Oeser Company facility in Bellingham, Washington. The primary objective of this baseline HHRA was to evaluate potential adverse health effects attributable to site-related contaminants at The Oeser Company in the absence of remedial action. This baseline risk assessment provides conservative estimates of risks to potentially exposed populations assuming that no remediation or institutional controls are applied to the site. The resulting risk estimates are intended to not underestimate risks, and will likely overestimate risks for most scenarios in order to provide a conservative basis for remediation decisions. The results of the baseline HHRA will be used to support decisions regarding the necessity and extent of remediation and will aid in the selection of appropriate remedial technologies.

The baseline HHRA was conducted in accordance with national and regional guidance. The principal guidance documents include the following:

- *Risk Assessment Guidance for Superfund (RAGS), Human Health Evaluation Manual, Part A (EPA 1989); Part B, Development of Risk Based Preliminary Remediation Goals (EPA 1991a); Part C, Risk Evaluation of Remedial Alternatives (EPA 1991b); Part D, Standardized Planning, Reporting, and Review of Superfund Risk Assessments (EPA 1998b); and Part E, Supplemental Guidance for Dermal Risk Assessment, Interim Guidance (EPA 2000b);*
- *Exposure Factors Handbook (EFH; EPA 1997a);*
- *RAGS Human Health Evaluation Manual, Supplemental Guidance, Standard Default Exposure Factors, (EPA 1991d);*
- *Guidance for Data Useability in Risk Assessment, (EPA 1991e);*
- *EPA, Region 10, Supplemental Risk Assessment Guidance for Superfund (EPA 1991c, 1996b); and*
- *Interim Final Guidance: Developing Risk-Based Cleanup Levels at Resource Conservation and Recovery Act Sites in Region 10 (EPA 1998a).*

As required by RAGS, Part D (EPA 1998b), Technical Approach for Risk Assessment (TARA) tables were constructed to evaluate the potential excess human health risks attributable to The Oeser Company (E & E 2001). In some cases, the EPA format of these tables was modified slightly to reduce the number of tables or to clarify the information contained in them. For example, TARA Tables 9 and 10 were not produced because these tables present information similar to that presented in TARA Tables 7 and 8. More than 350 tables were generated following the process outlined in RAGS, Part D. In this baseline HHRA, an attempt has been made to summarize the information contained in TARA Table 2 and TARA Tables 7 and 8. These tables are presented in this baseline HHRA as Tables 2-1a through 2-1f and 5-1 to 5-15, respectively. Please refer to Ecology and Environment, Inc. (E & E) 2001 for detailed information regarding these TARA tables. TARA Tables 1, 4, 5, and 6 are included in Appendix A of this baseline HHRA. TARA Table 3 is provided in E & E 2001.

1.1 RISK ASSESSMENT OVERVIEW

This section describes the general approach used in this baseline HHRA to assess potential health risks posed by contaminants at The Oeser Company. Detailed descriptions of The Oeser Company and the sampling conducted on and off the facility property are contained in *The Oeser Company Remedial Investigation Report* (E & E 2002). This information, hereinafter, is incorporated by reference:

- Site background, including site locations and descriptions, operations history, previous investigations, and environmental setting;
- Initial evaluations;
- Nature and extent of contamination;
- Contaminant fate and transport; and
- Summary and recommendations for further work.

For this baseline HHRA, the facility subareas and residential areas were evaluated using exposure scenarios developed in collaboration with the EPA, Region 10. The selected scenarios reflect some site-specific information regarding current and future land uses; however, in general, these scenarios incorporate default exposure assumptions intended to create uniformity in the U.S. EPA Superfund risk assessment process and generate reasonable estimates of maximum exposure. Potential exposures to current and future workers, residents, and recreational users were evaluated. The use of a future residential scenario on the facility does not imply that any future residential use of the facility is

planned or anticipated. Rather, conservative exposure assumptions associated with residential land use were used in order to be protective of all future possible uses of the land.

1.2 ORGANIZATION OF THE BASELINE HUMAN HEALTH RISK ASSESSMENT

Following this introduction, this baseline HHRA is organized according to the following tasks:

- **Contaminant Screening and Evaluation (Section 2).** The chemicals that were evaluated in this baseline HHRA are identified in this section. Contaminants of potential concern (COPCs) are listed in Tables 2-1a through 2-1f, and the contaminant screening tables (TARA Table 2) are presented in E & E 2001;
- **Exposure Assessment (Section 3).** This section describes the characterization of the exposure setting, the identification of potentially exposed populations (i.e., receptors), the identification of exposure scenarios and pathways, and the quantification of exposure. In addition, statistical analyses used to derive exposure point concentrations (EPCs) are summarized. The selection of exposure pathways is summarized in TARA Table 1 (Appendix A). A summary of exposure parameters and equations is presented in TARA Table 4. Statistical summaries for each medium, area, and COPC are presented in TARA Table 3 (E & E 2001);
- **Toxicity Assessment (Section 4).** Toxicity values for each COPC are identified in this task. The toxicity values for COPCs are presented in TARA Tables 5 and 6. Health effects summaries of contaminants of concern (COCs) are presented in Appendix B; and
- **Risk Characterization (Section 5).** Potential health risks based on the estimated exposure doses (identified in the exposure assessment) and the toxicity values (identified in the toxicity assessment) are evaluated in this section. These risks are evaluated for COPCs for each exposure pathway, and for all pathways combined. The risks are summarized in Tables 5-1 to 5-15. TARA Tables 7 and 8 present the results for each COPC, medium, and exposure pathway (E & E 2001).

Conclusions, recommendations, and references are presented in Sections 6, 7, and 8, respectively.

2. CONTAMINANT SCREENING AND EVALUATION

2.1 CONTAMINANT OF POTENTIAL CONCERN SELECTION

This section describes the procedures used to select the COPCs for The Oeser Company. The methodology described herein reflects federal (EPA 1989, 1998b) and EPA, Region 10 (EPA 1991c, 1998a), risk assessment guidance.

Selection of COPCs involves the following steps:

- C Initial data review and analyses;
- C Evaluation of chemical concentrations;
- C Comparison of chemical concentrations to risk-based screening benchmarks for human health; and
- C Comparison of on-facility concentrations of inorganic chemicals to natural background concentrations.

The following paragraphs describe in detail the procedures used to determine COPCs for The Oeser Company.

2.1.1 Initial Data Review and Analysis

Only analytical data from fixed laboratory analyses were screened for COPCs. No results from field and laboratory screening analyses were included. Data validation results for fixed laboratory data were reviewed, and all results flagged with *R* qualifiers (indicating rejected results) were excluded from the screening procedure and all following steps. Results flagged with *B* qualifiers were evaluated on a case-by-case basis. No COPCs had results flagged with *B* qualifiers.

In general, tentatively identified compounds (TICs) were excluded from consideration; however, some TICs may have been evaluated as part of classes of compounds, such as petroleum hydrocarbons.

Data are summarized in a manner consistent with TARA Table 2s (EPA 1998b) and are presented in E & E 2001. The information presented in these tables includes the minimum and maximum detected concentrations (with any associated qualifiers) and the detection frequency of each contaminant in each medium. In addition, the range of analytical detection limits (DLs) is presented for each

contaminant detected at least once in a medium; the frequency of cases in which these DLs exceeded the screening levels also is presented.

2.1.2 Evaluation of Chemical Concentrations

All analytes detected in any medium were included in a risk-based screening method for identifying COPCs at The Oeser Company. The screening procedure is outlined in the TARA (EPA 1998b) and follows the conservative approach recommended by the EPA (1998a). Tables 2-1a through 2-1f summarize the COPCs for each medium. Tables 2-2a through 2-2d list the samples used in the analysis of each medium. The detailed screening tables are presented in TARA Table 2s (E & E 2001). As recommended by the EPA, Region 10, screening concentrations used in this evaluation were derived from the EPA, Region 9, Preliminary Remediation Goals (PRGs; EPA 2000d), which provides chemical-specific screening concentrations (for residential and industrial soil, tap water, and ambient air) that correspond to a 1×10^{-6} excess lifetime cancer risk for carcinogens or a hazard quotient (HQ) of 1 for noncarcinogens. Specific equations and intake parameters used to derive the PRGs are provided in the EPA, Region 9, PRG table (EPA 2000d). Soil PRGs were derived considering the following exposure pathways: ingestion, inhalation of particulates and volatiles, and dermal absorption of contaminants in soil. Tap water PRGs address potential ingestion of contaminants in water and inhalation of volatiles during household water use. Petroleum was screened using Model Toxics Control Act (MTCA) Method A cleanup levels (Ecology 2000).

The risk-based screening concentrations for berries were derived using berry consumption rates provided in the EPA's EFH (EPA 1997a). Table 9-13 of the EFH lists a mean per capita intake rate of 0.00642 gram per kilogram body weight per day (g/kg BW-day) for blackberries, which are the most common type of berry that grows near Little Squalicum Creek. However, residents near the creek could have a higher consumption of blackberries relative to other berries because of the relative abundance of blackberries. Consequently, the intake rates listed for blackberries, blueberries, strawberries, and raspberries were summed to produce a total berry intake rate of 0.0531 g/kg BW-day. Assuming a 70-kilogram (kg) adult body weight, the consumption rate equals about 3.72 grams per day (g/day). This is equivalent to about 3 pounds of berries per person per year. Risk-based screening levels were calculated using EPA default residential assumptions:

For carcinogens:

$$RBC = \frac{TR \times BW \times AT}{EF \times ED \times (IR/CF) \times SF_{oral}}$$

Where: RBC = Risk-based concentration, milligrams per kilogram (mg/kg)

TR = Target risk level, 1E-06

BW = Body weight, 70 kg

AT = Averaging time, 25,500 days

CF = Conversion factor, 1,000 grams per kilogram

EF = Exposure frequency, 365 days/year

ED = Exposure duration, 30 years

IR = Ingestion rate, 3.72 g/day

SF_{oral} = Oral slope factor, chemical-specific, (mg/kg-day)⁻¹

For noncarcinogens:

$$RBC = \frac{RfD_{oral} \times HQ \times BW \times AT}{ED \times EF \times (IR/CF)}$$

Where: RBC = Risk-based concentration, mg/kg

RfD_{oral} = Oral reference dose, chemical-specific, mg/kg-day

HQ = Target hazard quotient, 0.1

AT = Averaging time, 10,950 days

The maximum detected chemical concentrations in each medium were compared to screening benchmarks as follows:

- C Contaminants in all media were screened using a screening benchmark corresponding to an excess lifetime cancer risk of 1×10^{-6} for carcinogens or an HQ of 0.1 for noncarcinogens;
- C Surface soil, subsurface soil, and sediment contaminant concentrations were compared to the screening levels for residential soil;
- C Groundwater contaminant concentrations were compared to the screening levels for tap water;
- C Air contaminant concentrations were compared to the screening levels for ambient air;

- Dioxins/furans were evaluated individually and as 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity equivalent (TEQ). The 2,3,7,8-TCDD TEQ was calculated by multiplying each dioxin congener by its respective toxicity equivalency factor (TEF; Vanden Berg et al. 1998) and then by summing the results. For nondetected congeners, one-half the DL times the TEF was used. In some cases, the 2,3,7,8-TCDD TEQ was calculated when individual dioxins/furans were analyzed, but were not detected. The 2,3,7,8-TCDD TEQ was compared to the PRGs for 2,3,7,8-TCDD;
- Carcinogenic polycyclic aromatic hydrocarbons (cPAHs) were evaluated individually and as benzo(a)pyrene (B[a]P) equivalents, respectively. B(a)P equivalents were calculated by multiplying each cPAH by its relative potency factor (RPF; EPA 1993a, CalEPA 1996) and then by summing the result. For nondetected compounds, one-half the DL times the RPF was used. In some cases, B(a)P equivalents were calculated when individual polycyclic aromatic hydrocarbons (PAHs) were analyzed, but were not detected. B(a)P equivalents were compared to the PRGs for B(a)P; and
- A fractionated approach was used to evaluate petroleum hydrocarbons. Petroleum hydrocarbons were evaluated as extractable petroleum hydrocarbons (EPHs), volatile petroleum hydrocarbons (VPHs), total petroleum hydrocarbon as gasoline (TPH-G), and total petroleum hydrocarbon as diesel (TPH-D). The concentrations of the individual detected petroleum hydrocarbon ranges were summed. For nondetected hydrocarbon ranges, one-half the DL was used. In some cases, total EPHs and VPHs were calculated when individual hydrocarbon ranges were analyzed, but were not detected. These sums then were compared to the new MTCA Method A values for petroleum hydrocarbons (Ecology 2001).

If the appropriate screening concentration was exceeded, then the chemical was considered a preliminary COPC.

Use of residential exposure assumptions for selection of preliminary COPCs does not imply that residential land use will occur at The Oeser Company or that residential soil cleanup levels ultimately will be selected as remediation goals. Instead, use of these assumptions represents a conservative, standardized screening methodology recommended by EPA, Region 10 (EPA 1998a).

For several compounds without risk-based screening values, EPA, Region 10, recommends the use of surrogate values based on similar molecular structure and/or toxicological properties:

- The PRG for naphthalene was used for 1-methylnaphthalene and 2-methylnaphthalene;
- The PRG for acenaphthene was used for acenaphthylene;
- The PRG for pyrene was used for benzo(g,h,i)perylene; and
- The PRG for anthracene was used for phenanthrene.

For chemicals that lack an appropriate surrogate value, the risk-based screening procedure cannot be used; consequently, these chemicals are evaluated qualitatively, not quantitatively, in Section 2.2.2. The elimination of potential contaminants due to a lack of toxicity values does not necessarily mean that the contaminants are unimportant or nontoxic; rather, it only means that toxicity values either have not been promulgated yet or are under review. Detected compounds that lack toxicity values are presented in Table 2-3 for each medium.

2.1.3 Evaluation of Background

Background samples were collected for soil, air, groundwater, surface water, and berries. For organic chemicals, the analytical results of the background samples were not used in the screening process. Instead, organic chemicals detected in background samples were compared to PRGs and carried forward through the risk assessment. The risks based on organic chemical concentrations in the background samples versus those samples collected from areas impacted by The Oeser Company operations then were compared.

Inorganic chemical concentrations detected in the background samples were determined to not be significantly different from the concentrations detected in the samples impacted by The Oeser Company operations (E & E 2000). In addition, inorganic chemicals were not associated with facility operations. Therefore, inorganic chemicals are not evaluated in this baseline HHRA.

2.2 CONTAMINANT OF POTENTIAL CONCERN SELECTION UNCERTAINTIES

Uncertainties associated with the COPC selection process originate from the analytical data and the COPC screening procedure. These uncertainties are discussed in the following sections, along with an overall perspective on the influence of these uncertainties on this baseline HHRA.

2.2.1 Analytical Data

All analytical results (not only those flagged as *estimated* [or *J* qualified] during the data validation process) possess an inherent variability. This variability or uncertainty in the true result depends on several factors, including the sample matrix, analytical method, and analytical laboratory performing the analysis. A variability of -50% to +100% is typical for samples with concentrations detected at less than the quantitation limit. For samples containing higher contaminant concentrations, relative percent differences of 35% for soil are considered acceptable (EPA 1988).

2.2.2 Contaminant of Potential Concern Screening Procedure

Not all chemicals detected in The Oeser Company environmental media samples were selected as COPCs. Current EPA-verified toxicity values were unavailable for several chemicals detected at The Oeser Company (Table 2-3); consequently, these chemicals were not included in the risk-based screening procedure. This could underestimate risks; however, most of these compounds were detected at relatively low concentrations compared to other compounds of similar chemical structure that have toxicity values. For example, tetrachlorophenols and retene were detected consistently in soil at one to two orders of magnitude lower than pentachlorophenol (PCP) and other noncarcinogenic (i.e., noncancer) PAHs, respectively.

The use of surrogate compounds for many of the noncarcinogenic PAHs that lack toxicity values may overestimate or underestimate risks depending on the actual toxicities of these compounds.

Analytes with DLs greater than their respective PRGs in at least one sample are listed in Table 2-4 for each medium. The failure to detect compounds that are present above screening values could underestimate risks; some of these compounds were never detected in the respective media (e.g., bis(2-chloroethyl)ether in soil). However, most of these compounds were detected and were evaluated in each medium. Additionally, not evaluating individual dioxins/furans, cPAHs, and fuel ranges because of elevated DLs was accounted for in the calculation of 2,3,7,8-TCDD TEQs, B(a)P equivalents, total EPHs, and total VPHs, where one-half the DL of each nondetected analyte was used.

For dioxins/furans and cPAHs, calculating risks based solely on the use of one-half the DL of individual congeners or compounds, when these compounds were never detected, likely will overestimate risks.

Assuming that 3 pounds of berries per person per year are collected from Little Squalicum Creek likely will overestimate exposure. This value is based on the total intake rates for blackberries, blueberries, strawberries, and raspberries.

The COPC screening may result in selection of chemicals that may not pose significant potential risks at the site because the PRGs used in the screening procedure were based on conservative assumptions and may not reflect site-specific conditions (e.g., a residential scenario was assumed and the PRGs were compared to maximum detected concentrations). Because of the conservatism built into the screening procedure, it is unlikely that any chemical excluded from the screening process actually poses a significant human health risk.

2.2.3 Perspectives on Contaminant of Potential Concern Selection Uncertainties

Given the uncertainties associated with the analytical data and COPC screening process, the overall COPC selection process was designed to be sufficiently conservative in order to include the principal site-related contaminants in this baseline HHRA.

Table 2-1a

**Contaminants of Potential Concern for Air, Berries, Groundwater, Sediment, and Surface Water
The Oeser Company
Bellingham, Washington**

Air	Berries	Groundwater	Sediment	Surface Water
B(a)P Equivalent	None	B(a)P Equivalent	B(a)P Equivalent	B(a)P Equivalent
1,2,3,4,6,7,8-HpCDD		2,3,7,8-TCDD TEQ	Benzo(a)anthracene	Benzo(a)pyrene
1,2,3,6,7,8-HxCDD		EPH	Benzo(a)pyrene	1,2,3,7,8,9-HxCDD
1,2,3,4,7,8-HxCDD		Napthalene	Benzo(b)fluoranthene	1,2,3,4,6,7,8-HpCDD
1,2,3,7,8,9-HxCDD		Pentachlorophenol	Benzo(j)fluoranthene	1,2,3,4,6,7,8-HpCDF
1,2,3,7,8-PeCDD			Dibenzo(a,e)pyrene	1,2,3,6,7,8-HxCDD
2,3,7,8-TCDD TEQ			Dibenzo(a,h)anthracene	OCDD
1,2,4-Trimethylbenzene			Dibenzo(a,h)pyrene	2,3,7,8-TCDD TEQ
1,3,5-Trimethylbenzene			Dibenzo(a,i)pyrene	Pentachlorophenol
2-Methylnaphthalene			Dibenzo(a,l)pyrene	
Naphthalene			1,2,3,4,6,7,8-HpCDD	
Benzene			1,2,3,4,6,7,8-HpCDF	
sec-Butylbenzene			1,2,3,6,7,8-HxCDD	
Dibenzofuran			1,2,3,7,8,9-HxCDD	
Pentachlorophenol			1,2,3,7,8-PeCDD	
n-Propylbenzene			2,3,4,7,8-PeCDF	
			Dioxin TEQ	
			OCDD	
			7,12-Dimethylbenz(a)anthracene	

Table 2-1b

Contaminants of Potential Concern for On-Facility Surface Soil
The Oeser Company
Bellingham, Washington

East and West Treatment Area	North and South Pole Yard (Current)	North Pole Yard (Future)	North Treatment Area (Current)	North Treatment Area (Future)	South Pole Yard (Future)	Treated Pole Area (Future)	Wood Storage Area (Current)	Wood Storage Area (Future)
None	B(a)P Equivalent	B(a)P Equivalent	B(a)P Equivalent	B(a)P Equivalent	7,12-Dimethylbenz(a)anthracene	B(a)P Equivalent	B(a)P Equivalent	B(a)P Equivalent
	Benzo(a)pyrene	Benzo(a)pyrene	Benzo(a)pyrene	Benzo(a)anthracene	B(a)P Equivalent	Benzo(a)anthracene	Benzo(a)anthracene	Benzo(a)anthracene
	1,2,3,4,6,7,8-HpCDD	1,2,3,4,6,7,8-HpCDD	1,2,3,4,6,7,8-HpCDD	Benzo(a)pyrene	Benzo(a)anthracene	Benzo(a)pyrene	Benzo(a)pyrene	Benzo(a)pyrene
	1,2,3,4,6,7,8-HpCDF	1,2,3,4,6,7,8-HpCDF	1,2,3,4,6,7,8-HpCDF	Benzo(b)fluoranthene	Benzo(a)pyrene	Benzo(b)fluoranthene	Benzo(b)fluoranthene	Benzo(b)fluoranthene
	1,2,3,4,7,8,9-HpCDF	1,2,3,4,7,8,9-HpCDF	1,2,3,4,7,8,9-HpCDF	Dibenzo(a,h)anthracene	Benzo(b)fluoranthene	Benzo(k)fluoranthene	Dibenzo(a,e)pyrene	Dibenzo(a,e)pyrene
	1,2,3,4,7,8-HxCDD	1,2,3,4,7,8-HxCDD	1,2,3,4,7,8-HxCDD	Indeno(1,2,3-cd)pyrene	Benzo(j)fluoranthene	Dibenzo(a,h)anthracene	Dibenzo(a,h)anthracene	Dibenzo(a,h)anthracene
	1,2,3,4,7,8-HxCDF	1,2,3,4,7,8-HxCDF	1,2,3,4,7,8-HxCDF	1,2,3,4,6,7,8-HpCDD	Dibenzo(a,e)pyrene	Indeno(1,2,3-cd)pyrene	Dibenzo(a,h)pyrene	Dibenzo(a,h)pyrene
	1,2,3,6,7,8-HxCDD	1,2,3,6,7,8-HxCDD	1,2,3,6,7,8-HxCDD	1,2,3,4,6,7,8-HpCDF	Dibenzo(a,h)anthracene	1,2,3,4,6,7,8-HpCDD	Indeno(1,2,3-cd)pyrene	Indeno(1,2,3-cd)pyrene
	1,2,3,6,7,8-HxCDF	1,2,3,6,7,8-HxCDF	1,2,3,6,7,8-HxCDF	1,2,3,4,7,8,9-HpCDF	Dibenzo(a,h)pyrene	1,2,3,4,6,7,8-HpCDF	1,2,3,4,6,7,8-HpCDD	1,2,3,4,6,7,8-HpCDD
	1,2,3,7,8,9-HxCDD	1,2,3,7,8,9-HxCDD	1,2,3,7,8,9-HxCDD	1,2,3,4,7,8-HxCDD	Dibenzo(a,i)pyrene	1,2,3,4,7,8,9-HpCDF	1,2,3,4,6,7,8-HpCDF	1,2,3,4,6,7,8-HpCDF
	1,2,3,7,8,9-HxCDF	1,2,3,7,8,9-HxCDF	1,2,3,7,8,9-HxCDF	1,2,3,4,7,8-HxCDF	Indeno(1,2,3-cd)pyrene	1,2,3,4,7,8-HxCDD	1,2,3,4,7,8,9-HpCDF	1,2,3,4,7,8,9-HpCDF
	1,2,3,7,8-PeCDD	1,2,3,7,8-PeCDD	1,2,3,7,8-PeCDD	1,2,3,6,7,8-HxCDD	1,2,3,4,6,7,8-HpCDD	1,2,3,4,7,8-HxCDF	1,2,3,4,7,8-HxCDD	1,2,3,4,7,8-HxCDD
	1,2,3,7,8-PeCDF	1,2,3,7,8-PeCDF	1,2,3,7,8-PeCDF	1,2,3,6,7,8-HxCDF	1,2,3,4,6,7,8-HpCDF	1,2,3,6,7,8-HxCDD	1,2,3,6,7,8-HxCDD	1,2,3,6,7,8-HxCDD
	2,3,4,6,7,8-HxCDF	2,3,4,6,7,8-HxCDF	2,3,4,6,7,8-HxCDF	1,2,3,7,8,9-HxCDD	1,2,3,4,7,8,9-HpCDF	1,2,3,6,7,8-HxCDF	1,2,3,6,7,8-HxCDF	1,2,3,6,7,8-HxCDF
	2,3,4,7,8-PeCDF	2,3,4,7,8-PeCDF	2,3,4,7,8-PeCDF	1,2,3,7,8,9-HxCDF	1,2,3,4,7,8-HxCDD	1,2,3,7,8,9-HxCDD	1,2,3,7,8,9-HxCDD	1,2,3,7,8,9-HxCDD
	2,3,7,8-TCDD	2,3,7,8-TCDD	2,3,7,8-TCDF	1,2,3,7,8-PeCDD	1,2,3,4,7,8-HxCDF	1,2,3,7,8,9-HxCDF	1,2,3,7,8,9-HxCDF	1,2,3,7,8,9-HxCDF
	2,3,7,8-TCDF	2,3,7,8-TCDF	Dioxin TEQ	1,2,3,7,8-PeCDF	1,2,3,6,7,8-HxCDD	1,2,3,7,8-PeCDD	1,2,3,7,8-PeCDD	1,2,3,7,8-PeCDD
	Dioxin TEQ	Dioxin TEQ	OCDD	2,3,4,6,7,8-HxCDF	1,2,3,6,7,8-HxCDF	1,2,3,7,8-PeCDF	1,2,3,7,8-PeCDF	1,2,3,7,8-PeCDF
	OCDD	OCDD	OCDF	2,3,4,7,8-PeCDF	1,2,3,7,8,9-HxCDD	2,3,4,6,7,8-HxCDF	2,3,4,6,7,8-HxCDF	2,3,4,6,7,8-HxCDF
	OCDF	OCDF		2,3,7,8-TCDD	1,2,3,7,8,9-HxCDF	2,3,4,7,8-PeCDF	2,3,4,7,8-PeCDF	2,3,4,7,8-PeCDF
	Pentachlorophenol	Pentachlorophenol		2,3,7,8-TCDF	1,2,3,7,8-PeCDD	2,3,7,8-TCDD	2,3,7,8-TCDD	2,3,7,8-TCDD
				Dioxin TEQ	1,2,3,7,8-PeCDF	2,3,7,8-TCDF	2,3,7,8-TCDF	2,3,7,8-TCDF
				OCDD	2,3,4,6,7,8-HxCDF	Dioxin TEQ	Dioxin TEQ	Dioxin TEQ
				OCDF	2,3,4,7,8-PeCDF	OCDD	OCDD	OCDD
				Total VPH	2,3,7,8-TCDD	OCDF	OCDF	OCDF
				Pentachlorophenol	2,3,7,8-TCDF	Pentachlorophenol	Total VPH	Benzidine
					Dioxin TEQ		Pentachlorophenol	Pentachlorophenol
					OCDD			
					OCDF			
					Pentachlorophenol			

Table 2-1c

Contaminants of Potential Concern for Off-Facility Surface Soil
The Oeser Company
Bellingham, Washington

Open Area-Residential	Open Area-Background	Residential	Residential Background	South Slope	Foot Path	Soil Spoils Piles
B(a)P Equivalent	B(a)P Equivalent	B(a)P Equivalent	B(a)P Equivalent	B(a)P Equivalent	7,12-Dimethylbenz(a)anthracene	7,12-Dimethylbenz(a)anthracene
Benzo(a)pyrene	Benzo(a)pyrene	Benzo(a)anthracene	Benzo(a)pyrene	Benzo(a)pyrene	B(a)P Equivalent	7H-Dibenzo(c,g)carbazole
Benzo(b)fluoranthene	Dioxin TEQ	Benzo(a)pyrene	Benzo(b)fluoranthene	1,2,3,4,6,7,8-HpCDD	Benzo(a)pyrene	B(a)P Equivalent
Dibenzo(a,h)anthracene		Benzo(b)fluoranthene	Dibenzo(a,h)anthracene	1,2,3,4,6,7,8-HpCDF	Dibenzo(a,e)pyrene	Benzo(a)anthracene
Indeno(1,2,3-cd)pyrene		Dibenzo(a,h)anthracene	1,2,3,4,6,7,8-HpCDD	1,2,3,4,7,8-HxCDD	Dibenzo(a,h)pyrene	Benzo(a)pyrene
1,2,3,4,6,7,8-HpCDD		Indeno(1,2,3-cd)pyrene	2,3,7,8-TCDD	1,2,3,6,7,8-HxCDD	Dibenzo(a,i)pyrene	Benzo(b)fluoranthene
1,2,3,4,6,7,8-HpCDF		1,2,3,4,6,7,8-HpCDD	Dioxin TEQ	1,2,3,7,8,9-HxCDD	1,2,3,4,6,7,8-HpCDD	Benzo(j)fluoranthene
1,2,3,4,7,8-HxCDD		1,2,3,6,7,8-HxCDD		1,2,3,7,8-PeCDD	1,2,3,4,6,7,8-HpCDF	Benzo(k)fluoranthene
1,2,3,4,7,8-HxCDF		1,2,3,7,8,9-HxCDD		2,3,4,7,8-PeCDF	1,2,3,6,7,8-HxCDD	Chrysene
1,2,3,6,7,8-HxCDD		1,2,3,7,8-PeCDD		Dioxin TEQ	1,2,3,7,8-PeCDD	Dibenzo(a,e)pyrene
1,2,3,7,8,9-HxCDD		Dioxin TEQ		OCDD	Dioxin TEQ	Dibenzo(a,h)anthracene
1,2,3,7,8,9-HxCDF					OCDD	Dibenzo(a,h)pyrene
1,2,3,7,8-PeCDD						Dibenzo(a,i)pyrene
2,3,4,6,7,8-HxCDF						Dibenzo(a,l)pyrene
2,3,4,7,8-PeCDF						Indeno(1,2,3-cd)pyrene
Dioxin TEQ						1,2,3,4,6,7,8-HpCDD
OCDD						1,2,3,4,6,7,8-HpCDF
						1,2,3,4,7,8,9-HpCDF
						1,2,3,4,7,8-HxCDD
						1,2,3,6,7,8-HxCDD
						1,2,3,7,8,9-HxCDD
						1,2,3,7,8-PeCDD
						2,3,4,6,7,8-HxCDF
						2,3,4,7,8-PeCDF
						2,3,7,8-TCDD
						Dioxin TEQ
						OCDD
						OCDF
						Total EPH
						Total VPH
						2-Methylnaphthalene

Table 2-1d

Contaminants of Potential Concern for Subsurface Soil (0 to 6 feet bgs)
The Oeser Company
Bellingham, Washington

East and West Treatment Area	North Pole Yard	North Treatment Area	South Pole Yard	Treated Pole Area	Wood Storage Area	South Slope	Path	Trench
B(a)P Equivalent	7,12-Dimethylbenz(a)anthracene	7,12-Dimethylbenz(a)anthracene	B(a)P Equivalent	B(a)P Equivalent	B(a)P Equivalent	B(a)P Equivalent	None	B(a)P Equivalent
Benzo(a)anthracene	B(a)P Equivalent	B(a)P Equivalent	Benzo(a)pyrene	Benzo(a)anthracene	Benzo(a)pyrene			
Benzo(a)pyrene	Benzo(a)anthracene	Benzo(a)anthracene	Dibenzo(a,e)pyrene	Benzo(a)pyrene	Dibenzo(a,h)pyrene			
Benzo(b)fluoranthene	Benzo(a)pyrene	Benzo(a)pyrene	Dibenzo(a,h)pyrene	Benzo(b)fluoranthene	1,2,3,4,6,7,8-HpCDD			
Benzo(k)fluoranthene	Benzo(b)fluoranthene	Benzo(b)fluoranthene	Dibenzo(a,i)pyrene	Benzo(k)fluoranthene	1,2,3,4,6,7,8-HpCDF			
Chrysene	Benzo(j)fluoranthene	Benzo(j)fluoranthene	Pentachlorophenol	Dibenzo(a,h)anthracene	1,2,3,4,7,8-HxCDD			
Dibenzo(a,h)anthracene	Dibenzo(a,e)pyrene	Benzo(k)fluoranthene		Indeno(1,2,3-cd)pyrene	1,2,3,6,7,8-HxCDD			
Indeno(1,2,3-cd)pyrene	Dibenzo(a,h)anthracene	Dibenzo(a,e)pyrene		1,2,3,4,6,7,8-HpCDD	1,2,3,7,8,9-HxCDD			
1,2,3,4,6,7,8-HpCDD	Dibenzo(a,h)pyrene	Dibenzo(a,h)anthracene		Dioxin TEQ	1,2,3,7,8-PeCDD			
1,2,3,4,6,7,8-HpCDF	Indeno(1,2,3-cd)pyrene	Dibenzo(a,h)pyrene		2-Methylnaphthalene	2,3,4,7,8-PeCDF			
1,2,3,4,7,8,9-HpCDF	1,2,3,4,6,7,8-HpCDD	Dibenzo(a,i)pyrene		Naphthalene	Dioxin TEQ			
1,2,3,4,7,8-HxCDD	1,2,3,4,6,7,8-HpCDF	Dibenzo(a,j)acridine			OCDD			
1,2,3,4,7,8-HxCDF	1,2,3,4,7,8,9-HpCDF	Indeno(1,2,3-cd)pyrene			2-Methylnaphthalene			
1,2,3,6,7,8-HxCDD	1,2,3,4,7,8-HxCDF	1,2,3,4,6,7,8-HpCDD			Pentachlorophenol			
1,2,3,6,7,8-HxCDF	1,2,3,6,7,8-HxCDD	1,2,3,4,6,7,8-HpCDF						
1,2,3,7,8,9-HxCDD	1,2,3,6,7,8-HxCDF	1,2,3,4,7,8,9-HpCDF						
1,2,3,7,8-PeCDF	1,2,3,7,8,9-HxCDD	1,2,3,4,7,8-HxCDF						
2,3,4,6,7,8-HxCDF	1,2,3,7,8,9-HxCDF	1,2,3,6,7,8-HxCDD						
2,3,4,7,8-PeCDF	1,2,3,7,8-PeCDD	1,2,3,6,7,8-HxCDF						
2,3,7,8-TCDF	1,2,3,7,8-PeCDF	1,2,3,7,8,9-HxCDD						
Dioxin TEQ	2,3,4,6,7,8-HxCDF	1,2,3,7,8,9-HxCDF						
OCDD	2,3,4,7,8-PeCDF	1,2,3,7,8-PeCDF						
Total EPH	2,3,7,8-TCDF	2,3,4,6,7,8-HxCDF						
2-Methylnaphthalene	Dioxin TEQ	2,3,4,7,8-PeCDF						
Acenaphthene	OCDD	2,3,7,8-TCDF						
Fluoranthene	Total EPH	Dioxin TEQ						
Fluorene	Total VPH	OCDD						
Naphthalene	2-Methylnaphthalene	OCDF						
Phenanthrene	Pentachlorophenol	Total EPH						
Pyrene		Total VPH						
Pentachlorophenol		2-Methylnaphthalene						
		Naphthalene						
		Pentachlorophenol						

Table 2-1e

Contaminants of Potential Concern for Subsurface Soil (6 to 12 feet bgs)
 The Oeser Company
 Bellingham, Washington

East and West Treatment Area	North Pole Yard	North Treatment Area	South Pole Yard	Treated Pole Area	Wood Storage Area	Foot Path	South Slope
B(a)P Equivalent	B(a)P Equivalent	7,12-Dimethylbenz(a)anthracene	7,12-Dimethylbenz(a)anthracene	B(a)P Equivalent	B(a)P Equivalent	None	B(a)P Equivalent
Benzo(a)anthracene	1,2,3,4,6,7,8-HpCDD	B(a)P Equivalent	7H-Dibenzo(c,g)carbazole	Benzo(a)anthracene	Benzo(a)anthracene		Dioxin TEQ
Benzo(a)pyrene	1,2,3,6,7,8-HxCDD	Benzo(a)anthracene	B(a)P Equivalent	Benzo(a)pyrene	Benzo(a)pyrene		
Benzo(b)fluoranthene	1,2,3,7,8,9-HxCDD	Benzo(a)pyrene	Benzo(a)anthracene	Benzo(b)fluoranthene	Benzo(b)fluoranthene		
Benzo(k)fluoranthene	1,2,3,7,8-PeCDD	Benzo(b)fluoranthene	Benzo(a)pyrene	Dioxin TEQ	Total VPH		
Chrysene	Dioxin TEQ	Benzo(j)fluoranthene	Benzo(b)fluoranthene	TPH-Gas	2-Methylnaphthalene		
Dibenzo(a,h)anthracene	Total VPH	Dibenzo(a,e)pyrene	Benzo(j)fluoranthene	2-Methylnaphthalene	Naphthalene		
Indeno(1,2,3-cd)pyrene	2-Methylnaphthalene	Dibenzo(a,h)anthracene	Benzo(k)fluoranthene	Naphthalene	Pentachlorophenol		
2-Methylnaphthalene		Dibenzo(a,h)pyrene	Dibenzo(a,e)pyrene	Pentachlorophenol			
Fluoranthene		Dibenzo(a,i)pyrene	Dibenzo(a,h)anthracene				
Naphthalene		Indeno(1,2,3-cd)pyrene	Dibenzo(a,h)pyrene				
Carbazole		1,2,3,4,6,7,8-HpCDD	Dibenzo(a,i)pyrene				
Dibenzofuran		1,2,3,4,6,7,8-HpCDF	Indeno(1,2,3-cd)pyrene				
Pentachlorophenol		1,2,3,4,7,8-HxCDF	1,2,3,4,6,7,8-HpCDD				
		1,2,3,6,7,8-HxCDD	1,2,3,4,6,7,8-HpCDF				
		1,2,3,6,7,8-HxCDF	1,2,3,6,7,8-HxCDD				
		1,2,3,7,8,9-HxCDD	1,2,3,6,7,8-HxCDF				
		1,2,3,7,8-PeCDF	1,2,3,7,8,9-HxCDD				
		2,3,4,6,7,8-HxCDF	1,2,3,7,8,9-HxCDF				
		2,3,4,7,8-PeCDF	1,2,3,7,8-PeCDF				
		Dioxin TEQ	2,3,4,6,7,8-HxCDF				
		OCDD	2,3,4,7,8-PeCDF				
		Total EPH	2,3,7,8-TCDF				
		Total VPH	Dioxin TEQ				
		TPH-Diesel	OCDD				
		2-Methylnaphthalene	Total EPH				
		Naphthalene	Total VPH				
		Dibenzofuran	2-Methylnaphthalene				
		Pentachlorophenol	Fluoranthene				
			Naphthalene				
			Pentachlorophenol				

Table 2-1f

Contaminants of Potential Concern for Subsurface Soil (12 to 18 feet bgs)
 The Oeser Company
 Bellingham, Washington

East and West Treatment Area	North Pole Yard	North Treatment Area	South Pole Yard	Treated Pole Area	Wood Storage Area	Foot Path	South Slope
B(a)P Equivalent	B(a)P Equivalent	B(a)P Equivalent	B(a)P Equivalent	None	B(a)P Equivalent	None	B(a)P Equivalent
Benzo(a)anthracene		Benzo(a)anthracene					
Benzo(a)pyrene		Benzo(a)pyrene					
Benzo(b)fluoranthene		Benzo(b)fluoranthene					
Benzo(k)fluoranthene		Benzo(k)fluoranthene					
Chrysene		Dibenzo(a,h)anthracene					
Dibenzo(a,h)anthracene		Indeno(1,2,3-cd)pyrene					
Indeno(1,2,3-cd)pyrene		Total EPH					
1,2,3,4,6,7,8-HpCDD		TPH-Diesel					
1,2,3,4,6,7,8-HpCDF		2-Methylnaphthalene					
1,2,3,4,7,8,9-HpCDF		Naphthalene					
1,2,3,4,7,8-HxCDD		Dibenzofuran					
1,2,3,4,7,8-HxCDF		Pentachlorophenol					
1,2,3,6,7,8-HxCDD							
1,2,3,6,7,8-HxCDF							
1,2,3,7,8,9-HxCDD							
1,2,3,7,8,9-HxCDF							
1,2,3,7,8-PeCDF							
2,3,4,6,7,8-HxCDF							
2,3,4,7,8-PeCDF							
2,3,7,8-TCDF							
Dioxin TEQ							
OCDD							
Total EPH							
TPH-Diesel							
2-Methylnaphthalene							
Acenaphthene							
Fluoranthene							
Fluorene							
Naphthalene							
Pyrene							
Carbazole							
Dibenzofuran							
Pentachlorophenol							

Table 2-2a

**Air, Berry, Groundwater, Sediment, and Surface Water Samples Used in the Human Health Risk Assessment^a
The Oeser Company
Bellingham, Washington**

Air	Berries	Groundwater	Sediment	Surface Water
AS24	Berry1-Washed	Ershigs-1a	SD01	SW01
AS25	Berry1-Unwashed	Ershigs-4a	SD02	SW02
AS26	Berry2-Washed	MW01-D	SD03	SW04
AS27	Berry2-Unwashed	MW02-D	SD04	SW05
AS28	Berry3-Washed	MW03-D	SD05	SW07
AS29	Berry3-Unwashed	MW05-D	SD06	
AS30	Berry4-Washed	MW06-D	SD07	
AS32	Berry4-Unwashed	MW17-D	SD08	
AS33		MW18-D	SD09	
		MW20-D	SD10	
		MW23-D	SD11	
		MW24-D		
		MW25-D		
		MW30-D		
		MW33-D		
		MW34-D		
		MW35-D		
		TC-6		
		TC-5		

^aAir, groundwater, and surface water values represent locations that were sampled multiple times.

Table 2-2b

**On-Facility Surface Soil Samples Used in the Human Health Risk Assessment
The Oeser Company
Bellingham, Washington**

East and West Treatment Area	North and South Pole Yard (Current)	North Pole Yard (Future)	North Treatment Area (Current)	North Treatment Area (Future)	South Pole Yard	Treated Pole Area	Wood Storage Area (Current)	Wood Storage Area (Future)
NA	B-B13	B-B13	B-L30	B-L17	B-N3	B-B16	B-Q20	B-Q20
	B-D7	B-D7	SS26	B-L25	B-O13	B-J20	B-Q26	B-R18
	B-F3	B-F3	SS57	B-L30	B-O7	OS08	B-R18	B-R28
	B-J2	B-J2	SS66	B-Q26	MW28-S	SI-OS01	B-R28	B-U19
	SS23	SS17		B-Q27	MW29-S	SI-OS02	B-U19	MW02-D
		SS23		MW32-S	MW35-S	SI-OS03	MW02-D	MW33-S
		SS35		MW36-S	SS11	SS19	MW33-S	SI-OS04
				SI-OS05	SS45	SS20	SI-OS04	
				SI-SD2	SS50	SS22		
				SS01				
				SS04				
				SS06				
				SS24				
				SS26				
			SS57					
			SS66					

Key:

NA = Not applicable (Surface soil samples were not collected.).

Table 2-2c

**Off-Facility Surface Soil Samples Used in the Human Health Risk Assessment
The Oeser Company
Bellingham, Washington**

Foot Path	South Slope	Soil Spoil Piles	Open Area Residential	Open Area Background	Residential	Residential Background
MWLSC01	B-AA2	SP01	RES-02	RES-B-01A	RES-01	RES-B-01
MWLSC02	B-AA4	SP02	RES-05	RES-B-02	RES-03	RES-B-04
MWLSC03	B-AA6	SP03	RES-07	RES-B-03	RES-04	RES-B-05
MWLSC04	B-BB3	SP04	RES-08	RES-B-06	RES-06	RES-B-08
	B-BB5	SP05	RES-11	RES-B-07	RES-09	RES-B-09
	MW03-D	SP06	RES-12	RES-B-12	RES-10	RES-B-10
	OS60	SP07	RES-16	RES-B-13	RES-13	RES-B-11
	RES-41		RES-18	RES-B-18	RES-14	RES-B-15
	RES-42		RES-28	RES-B-19	RES-15	RES-B-16
	RES-42A		RES-32A	RES-B-20	RES-17	RES-B-17
	RES-43		RES-36		RES-19	
	RES-46		RES-38		RES-20	
			RES-41		RES-21A	
			RES-42		RES-22	
			RES-42A		RES-23	
			RES-43		RES-24	
			RES-46		RES-25	
			RES-47A		RES-26	
			RES-48		RES-29	
			RES-49		RES-30	
			RES-50		RES-31	
			RES-51		RES-33	
			RES-52		RES-34	
			RES-53		RES-35	
			RES-54		RES-37	
			RES-55		RES-44A	
			RES-56		RES-58	
			RES-57			

Table 2-3

**Detected Analytes Lacking EPA-Verified Toxicity Values
The Oeser Company
Bellingham, Washington**

Air	Berries	Groundwater	Sediment	Soil	Surface Water
Benzo(e)pyrene	None	Tetrachlorophenols	Tetrachlorophenols	1,3,5-Tribromophenol	Tetrachlorophenols
Perylene				2-Nitrophenol	
p-Isopropyltoluene				4,6-Dinitro-2-methylphenol	
				4-Chloro-3-Methylphenol	
				4-Chlorophenyl-phenylether	
				Retene	
				Tetrachlorophenols	

Table 2-4

**Summary of Analytes
Where Detection Limits Exceeded Screening Levels
The Oeser Company
Bellingham, Washington**

Air	Berries	Groundwater	Sediment	Soil	Surface Water
1,2,4-Trimethylbenzene	1,2,3,7,8-PeCDD	1,1-Dichloroethene	1,2,3,4,7,8-HxCDF	1,1-Dichloroethene	1,1-Dichloroethene
1,2,3,4,6,7,8-HpCDD	2,3,4,7,8-PeCDF	1,2,3,4,6,7,8-HpCDD	1,2,3,6,7,8-HxCDF	1,2,3,4,7,8,9-HpCDF	1,2,3,4,7,8-HxCDD
1,2,3,4,6,7,8-HpCDF	2,3,7,8-TCDD	1,2,3,4,7,8-HxCDD	1,2,3,7,8,9-HxCDF	1,2,3,4,7,8-HxCDD	1,2,3,4,7,8-HxCDF
1,2,3,4,7,8,9-HpCDF	Dibenzo(a,h)pyrene	1,2,3,4,7,8-HxCDF	1,2,3,7,8-PeCDD	1,2,3,4,7,8-HxCDF	1,2,3,6,7,8-HxCDD
1,2,3,4,7,8-HxCDD	Dibenzo(a,i)pyrene	1,2,3,6,7,8-HxCDD	1,3-Dichlorobenzene	1,2,3,6,7,8-HxCDF	1,2,3,6,7,8-HxCDF
1,2,3,4,7,8-HxCDF	Dibenzo(a,l)pyrene	1,2,3,6,7,8-HxCDF	1,4-Dichlorobenzene	1,2,3,7,8,9-HxCDD	1,2,3,7,8,9-HxCDD
1,2,3,6,7,8-HxCDD		1,2,3,7,8,9-HxCDD	2,3,4,6,7,8-HxCDF	1,2,3,7,8,9-HxCDF	1,2,3,7,8,9-HxCDF
1,2,3,6,7,8-HxCDF		1,2,3,7,8,9-HxCDF	2,3,4,7,8-PeCDF	1,2,3,7,8-PeCDD	1,2,3,7,8-PeCDD
1,2,3,7,8,9-HxCDD		1,2,3,7,8-PeCDD	2,4-Dichlorophenol	1,2,3,7,8-PeCDF	1,2,3,7,8-PeCDF
1,2,3,7,8,9-HxCDF		1,2,3,7,8-PeCDF	2,4-Dimethylphenol	1,3-Dichlorobenzene	1,3-Dichlorobenzene
1,2,3,7,8-PeCDD		1,3-Dichlorobenzene	2,4-Dinitrophenol	1,4-Dichlorobenzene	1,4-Dichlorobenzene
1,2,3,7,8-PeCDF		1,4-Dichlorobenzene	2,4-Dinitrotoluene	2,3,4,6,7,8-HxCDF	2,3,4,6,7,8-HxCDF
1,3,5-Trimethylbenzene		2,3,4,6,7,8-HxCDF	2,6-Dinitrotoluene	2,3,4,7,8-PeCDF	2,3,4,7,8-PeCDF
2,3,4,6,7,8-HxCDF		2,3,4,7,8-PeCDF	2-Chloronaphthalene	2,3,7,8-TCDD	2,3,7,8-TCDD
2,3,4,7,8-PeCDD		2,3,7,8-TCDD	2-Chlorophenol	2,3,7,8-TCDF	2,3,7,8-TCDF
2,3,7,8-TCDD		2,3,7,8-TCDF	2-Methylnaphthalene	2,4-Dinitrophenol	bis(2-Chloroethyl)ether
2,3,7,8-TCDF		2-Nitroaniline	2-Methylphenol	2,4-Dinitrotoluene	C10-C12 Aliphatics
2,4,6-Trichlorophenol		3,3'-Dichlorobenzidine	2-Nitroaniline	2,6-Dinitrotoluene	C10-C12 Aromatics
2,4-Dichlorophenol		bis(2-Chloroethyl)ether	3,3'-Dichlorobenzidine	2-Nitroaniline	C12-C16 Aliphatics
2,4-Dimethylphenol		C10-C12 Aliphatics	4-Chloroaniline	3,3'-Dichlorobenzidine	C12-C16 Aromatics
2,4-Dinitrophenol		C10-C12 Aromatics	4-Methylphenol	Benzidine	C16-C21 Aliphatics
2-Chloronaphthalene		C12-C16 Aliphatics	4-Nitrophenol	Benzo(a)anthracene	C16-C21 Aromatics
2-Chlorophenol		C12-C16 Aromatics	Benzidine	Benzo(a)pyrene	C21-C34 Aliphatics
4-Methylphenol		C16-C21 Aliphatics	Benzo(a)anthracene	Benzo(b)fluoranthene	C21-C34 Aromatics
4-Nitrophenol		C16-C21 Aromatics	Benzo(a)pyrene	bis(2-Chloroethyl)ether	C8-C10 Aliphatics
Benzo(a)pyrene		C21-C34 Aliphatics	Benzo(b)fluoranthene	C10-C12 Aromatics	Chloromethane
Carbazole		C21-C34 Aromatics	Benzo(k)fluoranthene	C12-C16 Aromatics	Dibenzo(a,h)anthracene
Dibenzo(a,h)anthracene		C8-C10 Aliphatics	bis(2-Chloroethyl)ether	Dibenzo(a,h)anthracene	Hexachlorobenzene
Dibenzofuran		Chloromethane	C10-C12 Aromatics	Hexachlorobenzene	Naphthalene
n-Butylbenzene		Hexachlorobenzene	C12-C16 Aromatics	Indeno(1,2,3-cd)pyrene	n-Nitroso-di-n-propylamine
Pentachlorophenol		Naphthalene	Dibenzo(a,h)anthracene	Nitrobenzene	
sec-Butylbenzene		Nitrobenzene	Dibenzofuran	n-Nitroso-di-n-propylamine	
tert-Butylbenzene		n-Nitroso-di-n-propylamine	Hexachlorobenzene	Pentachlorophenol	
			Indeno(1,2,3-cd)pyrene		
			Naphthalene		
			Nitrobenzene		
			n-Nitroso-di-n-propylamine		
			Pentachlorophenol		

3. EXPOSURE ASSESSMENT

The purpose of the exposure assessment is to estimate the magnitude of human exposures, the frequency and duration of these exposures, and the pathways by which humans potentially are exposed (EPA 1989). The following section describes the exposure assessment.

3.1 CHARACTERIZING EXPOSURE SETTING

3.1.1 Human Health Conceptual Site Model

COPCs were detected in on-facility surface and subsurface soil, groundwater, and air. In addition, similar contaminants were detected in off-facility soil, groundwater, air, sediments, surface water, and berries. Following is a narrative describing the human health conceptual site model (CSM; Figure 3-1).

Contaminants in surface soil may remain bound to surface soil; may volatilize; may be dispersed by wind as particulates; may be transported over the surface by runoff or overland flow to off-facility surface soils; and/or may infiltrate subsurface media, including subsurface soil and groundwater. The EPA's removal activities, including removal and capping of contaminated surface soil, have reduced potential future contaminant releases. Contaminated groundwater flowing toward Little Squalicum Creek may be released through seeps to creek surface water, to creek sediment, and to soils adjacent to the creek. Vapors and particulates released from facility processes and vapors released from treated logs may be transported as volatiles or particulates by wind. The CSM includes current and historical sources because it is not possible to differentiate contamination in air due to past releases or ongoing wood-treating operations. However, this does not imply that air releases from an operating facility fall under the jurisdiction of CERCLA.

Media that have been or may be impacted as a result of these transport processes include:

- On- and off-facility surface soil;
- On- and off-facility fugitive dust;
- On- and off-facility subsurface soil;
- Surface water and sediment in Little Squalicum Creek;

- Groundwater underlying the facility and downgradient of the facility; and
- On- and off-facility air.

Foods that may be impacted by facility-related contaminants include berries growing along recreational trails (near the facility and Little Squalicum Creek) and home-grown produce (from nearby residences). Berries were determined not to have been impacted by facility-related COPCs. Blackberry samples were collected near Little Squalicum Creek, and COPC concentrations were not above conservative health-based screening levels (Section 2.1.2). Based on the blackberry sampling, home-grown vegetables initially were assumed not to have been impacted; however, the EPA requested that home-grown vegetables be evaluated for dioxin and furan congeners in this baseline HHRA.

A City of Bellingham ordinance prohibits hunting in the creek vicinity (Bryson 2001). Because the City of Bellingham diverted much of the natural drainage from Little Squalicum Creek to Squalicum Creek nearly 100 years ago, virtually eliminating the flow of natural water within the creek, the presence of fish in Little Squalicum Creek is very limited (EPA 1997c). Wahl (1998) occasionally has observed salmon fingerlings in a small pool that forms where the creek meets the beach at Bellingham Bay. The fish enter the creek from the bay (during unusually high tides and/or storm surges) and remain there for a short time before returning to the bay (Wahl 1998). The creek does not support fish upstream from this terminal pool. The Washington Department of Fish and Wildlife has shock-fished Little Squalicum Creek on several occasions, but has not recovered fish (McGowen 1998). The presence of fish upstream from the terminal pool is unlikely due to the creek's shallow depth, limited flow, and tendency to run nearly dry at times.

No information on historical fisheries in the creek was identified. Documents indicate that in addition to the diversion of drainage from the creek in 1907 by the City of Bellingham, going back to the 1920s, the creek had been used for disposal of wastes from sugar beet processing. Further, sand and gravel mining in the creek area dating back to the early 1930s through the 1960s led to substantial rerouting and changes to the original creek bed, including creation of surface water pools for gravel washing. Based on this information, it is unlikely that any fishery was present after the turn of the century due to physical impact, rather than chemical contamination, on the creek. (EPA 1997c)

Bellingham Bay serves as a fishery, and a separate investigation, not related to The Oeser Company remedial investigation, is ongoing by the Washington State Department of Ecology (Ecology). The Bellingham Bay Demonstration Pilot was established to address the need for sediment cleanup in the

bay. The project addresses sediment cleanup and source control, sediment disposal siting, habitat, and land use. (Ecology 2001b)

The sediments underlying The Oeser Company are Pleistocene glacial outwash deposits resulting from a series of glaciations that occurred between 2 million and 10,000 years ago. Three geologic zones comprise the outwash deposits. An “upper sandy zone” typically occurs from land surface to 20 or 25 feet below ground surface (bgs) and is predominantly fine-to-medium sand with lenses of silt and clay. A “gravelly zone” occurs below the upper sandy zone and comprises gravel and sand with minor pure sand, silt, and clay lenses. The gravelly zone is 25 to 40 feet thick where it has been penetrated locally. A “lower sandy zone” is present below the gravelly zone. The top of the lower sandy zone is encountered at 40 to 50 feet bgs and comprises poorly graded fine-to-medium sand with silt and clay (Easterbrook 1999).

Groundwater occurs in two zones beneath the site. Shallow groundwater occurs at 4 to 15 feet bgs in the upper sandy zone. Shallow groundwater is perched on fine-grained material and discharges downward to the deep aquifer. Deep groundwater generally occurs at 30 to 45 feet bgs in the gravelly zone and lower sandy zone. The deep aquifer comprises coarser, more permeable material and occurs as a continuously saturated aquifer. Deep groundwater likely discharges to Little Squalicum Creek and Bellingham Bay. Little Squalicum Creek is the dominant surface water feature near the site and flows intermittently throughout the year (URS 1994). The creek flows from northeast to southwest and is located 200 feet south of the site at its closest point. In its downstream reaches, Little Squalicum Creek likely serves as a discharge point for the deep aquifer.

Groundwater from the deep aquifer currently is not known to be used for drinking water, except at the nearby cement plant (Tilbury Cement Company) wells, approximately 1,500 feet southwest of the facility. The plant wells also draw water from the deep aquifer for industrial purposes. Although groundwater is not used or planned as a source of drinking water at The Oeser Company facility, federal guidance (EPA 1990) includes groundwater as a potential exposure medium for future receptors. Based on the facility geology, shallow, perched groundwater is unlikely to be developed as a drinking water source in the future because it is discontinuous across the facility and would be unreliable. Only some monitoring wells within the shallow perched groundwater zone beneath the facility yield sufficient groundwater to meet the Washington Administrative Code (WAC) 173-340-720 criteria for a future drinking water source (specifically, sustained pumping at 0.5 gallon per minute), and it is unlikely that this zone would produce such quantities over a sustained period during all seasons. Because hydraulic connectivity of the shallow perched zone and deep aquifer has not been ruled out, and because the deeper

aquifer may be usable as a potential future source of domestic water, protection of the deep aquifer from contamination in the shallow perched aquifer must be considered.

Receptors that potentially are exposed to facility-related contamination, or that may be exposed if current land uses change, include:

- Current and future residents (adults and children) living adjacent to the facility or nearby and potential future on-site residents;
- Current and future on-facility workers;
- Current and future on-facility construction and utility workers;
- Current and future on-facility trespassers; and
- Current and future recreational users who visit Little Squaticum Creek and the adjacent trail.

Each potential receptor and the media to which it may be exposed are discussed in the following paragraphs. All identified exposure pathways are included; however, some may be insignificant in terms of the total risk to each receptor group. Consequently, all of the pathways may not be included in the quantitative risk assessment. TARA Table 1 (Appendix A) presents the rationale behind selection or exclusion of each exposure pathway.

If contaminants migrate off facility to residential areas, residents may inhale airborne contaminants transported as particulates and vapors. Residents may ingest, inhale, or have dermal contact with contaminants in surface soil or may ingest potentially contaminated home-grown produce if contaminants have migrated off the facility. Although there are no current plans for development of the deep aquifer, if it were developed for future domestic use, residents potentially could ingest contaminated groundwater, or be exposed dermally to contaminants in groundwater during household use. As shown in Table 2-1a, volatile COPCs were not identified in groundwater. If the facility were developed for residential use in the future, these same residential exposure pathways may be appropriate for on-facility receptors. In addition, if excavation activities were to occur on the facility as a result of residential development, subsurface soils then could be exposed as surface soils, and future on-facility residents could have direct contact with contaminants currently found in subsurface soils.

On-facility workers, under current and future conditions, may inhale particulates and vapors in air or have direct contact with exposed facility surface soil. Workers also could be exposed to contaminants in groundwater through direct contact if the deep aquifer were developed for facility use. Volatile organic compounds (VOCs) are present in subsurface soil and groundwater underlying the facility, and on-facility

workers may inhale vapors that migrate from these media to the ground surface. If excavation activities occur on the facility, then construction and utility workers could be exposed to contaminants through the same pathways as on-facility workers and may have direct contact with contaminated subsurface soil and groundwater.

Off-facility recreational visitors to Little Squalicum Creek and nearby trails may have dermal contact with surface water or sediment contaminants from the creek. Near other off-facility areas (e.g., the grassy slope south of the facility), recreational visitors may contact potentially contaminated soil. Recreational visitors who eat berries growing near The Oeser Company and Little Squalicum Creek may ingest contaminants deposited onto plant surfaces through particle deposition or incorporated into plants through root uptake or vapor transport. The City of Bellingham intends to develop Little Squalicum Creek and adjacent areas into a park as soon as feasible. Although this likely will increase the number of recreational users, the potential exposure pathways identified above are not expected to change.

3.2 EXPOSURE PATHWAYS

The following exposure pathways were evaluated quantitatively in this baseline HHRA:

- Incidental ingestion of soil;
- Dermal contact with soil;
- Inhalation of volatilized substances from soil;
- Inhalation of wind-blown dust;
- Ingestion of home-grown produce;
- Dermal contact with surface water and sediments;
- Ingestion of groundwater; and
- Dermal contact with groundwater.

Inhalation of VOCs from indoor use of groundwater was not evaluated because volatile COPCs were not detected in groundwater.

3.2.1 Identifying Exposure Scenarios

Exposure scenarios were developed by examining the major exposure pathways to estimate the overall potential exposure of each receptor. The exposure scenarios and pathways that are evaluated in this baseline HHRA are summarized in the CSM (Figure 3-1) and TARA Table 1. Current and future

land use were evaluated as part of the development of the CSM. Based on information currently available, the following represent current exposure conditions:

- **Residents.** Residents are not likely to live on the facility under future land use conditions; however, a small portion of the facility property is zoned for residential use. In addition, changes in zoning restrictions could result in future residents on the facility. Finally, residences are located immediately north and east of the facility and nearby in all directions. These residences are expected to remain in the future;
- **On-Facility Workers.** Workers are present at the facility. In addition, most of the facility property is zoned for industrial land use; therefore, workers may be on the facility property in the future regardless of any potential changes in property ownership; and
- **Recreational Users.** Although access to the facility is restricted, individuals use the undeveloped area south of the facility near Little Squaticum Creek for recreational purposes. This use likely will continue in the future, particularly if the creek area is developed into a park.

Institutional controls, such as groundwater use restrictions, are not considered in this baseline HHRA, but may be evaluated in the feasibility study (FS). Although The Oeser Company is expected to operate as an industrial facility in the future, residential development of the site was evaluated in this baseline HHRA to provide information for risk management decisions.

3.3 QUANTIFICATION OF EXPOSURE

This section describes the calculation of potential exposure to COPCs through the identified exposure pathways. Estimates of chemical intake are based on EPCs and on the estimated magnitude of exposure to contaminated media. The derivation of these estimates is described below.

3.3.1 Exposure Point Concentrations

Analytical data were grouped in various ways for the purpose of calculating EPCs. The purpose of these groupings is to characterize exposures to receptors that may be exposed to a subset of the contamination at The Oeser Company. The rationale used to group data includes the following considerations:

- **Areas that Receptors Would Be Expected to Come in Contact with Routinely.** Where possible, data were grouped across areas where a receptor would spend a large portion of the time that he/she is exposed at the site. For example, residents living near the

facility are expected to be exposed mostly to soil on their own properties. Consequently, each residence was treated separately for estimation of EPCs. While workers on the facility property may be exposed to media from around the entire facility, they likely would be exposed primarily to a smaller area of the site routinely. Therefore, on-facility data were grouped into several subareas;

- **Sources of Contamination.** Several sources of contamination have been identified at The Oeser Company. Although there is some overlap, the known sources and types of soil contamination generally can be differentiated into the seven on-facility areas. Ultimately, the East and West Treatment areas were combined (because of the relatively small size of these areas and similarities in observed contamination) for a total of six subareas on facility. For example, contamination in the Treated Pole Area is limited mostly to surface soil contamination from drippage from poles, while contamination in the West Treatment Area consists of lenses of PCP and creosote extending at least 25 feet bgs. Grouping data by these areas provides a convenient way to estimate risks associated with each of the known sources and may be useful in the FS for remedial decision making; and
- **Availability of Data.** Past sampling at the site generally has not included analyses for all known site contaminants. Only a limited number of samples has been analyzed for dioxins/furans, while some samples were analyzed only for dioxins/furans and no other constituents. Consequently, estimation of risks associated with individual sample locations would be meaningless because the risk estimates could reflect the types of analyses performed rather than the contamination present. Although samples collected during RI field work have included a broader suite of target analytes, not every sample may have been analyzed for every analyte. Therefore, data were grouped in ways that include an adequate number of results for all known or expected site contaminants in each risk calculation.

The following sections describe how these considerations were used to calculate EPCs for on- and off-facility exposure media at The Oeser Company. TARA Table 3 for each exposure medium and site area presents the EPCs for each COPC, the statistical method used to derive each EPC, and the EPC rationale (E & E 2001).

3.3.1.1 On-Facility Surface Soil (Current Exposure Scenarios)

The on-facility data were grouped into six subareas including the East and West Treatment areas (combined), the North Pole Yard, the North Treatment Area, the South Pole Yard, the Treated Pole Area, and the Wood Storage Area. For each subarea, all of the available surface soil data were used, except for those areas covered by gravel or asphalt caps. Three facility areas not covered by gravel or asphalt caps (the North and South Pole Yards [combined because most of the South Pole Yard is covered with a gravel cap] the Treated Pole Area, and the Wood Storage Area) were evaluated for the current exposure scenario. EPCs for each subarea were calculated as described in *Supplemental Guidance to RAGS: Calculating the Concentration Term* (EPA 1992a). Note that for soil, nondetected results were

replaced with half the DL. The resulting values were included in the statistical analysis of each data set. EPCs for surface soil under current exposure scenarios were selected by first performing a W-test to test the normality of the COPC data set for each subarea. If the data distribution was not normally distributed, it was assumed to be lognormal, and no other distribution test was performed. Then, either the 95% upper confidence limit (UCL) of the arithmetic mean of the untransformed data was calculated for normal data sets or the 95% UCL of the arithmetic mean of the transformed data was calculated for lognormal data sets. The lesser of the 95% UCL or the maximum concentration for each COPC in a data set were selected as the EPC. However, if there were less than 10 samples in a subarea, the maximum concentration for each COPC automatically was used as the EPC.

Concentrations of COPCs in fugitive dust and outdoor vapors were calculated from soil EPCs as follows:

- **Fugitive Dust.** The particulate emission factor (PEF) approach described by EPA (1991a) was used to derive air concentrations resulting from fugitive dust emissions from soil data. A site-specific PEF was derived for use in this baseline HHRA using site meteorological data and methodologies defined in the EPA's *Soil Screening Guidance* (1996a). A PEF of 2.10×10^9 cubic meters per kilogram was derived using a site-specific dispersion model input for the inverse of the mean concentration at the center of a square source (Q/C) for Seattle, Washington (0.5 acre; a Q/C was not available for Bellingham, Washington), and a mean annual windspeed of 3.89 meters per second reported at Bellingham Airport. The mean annual windspeed was provided by the Western Regional Climate Center (Ashby 2000). The PEF was applied to the soil EPCs to calculate the air concentration of particulates; and
- **Vapors in Outdoor Air.** The soil-to-air volatilization factor (VF) approach described by EPA (1991a) was used to derive outdoor air EPCs resulting from volatilization of VOCs from soil. VOCs are defined as those compounds having a molecular weight of less than 200 grams per mole and a unitless Henry's Law constant greater than 1×10^{-5} (EPA 1998a). The chemical-specific VF is applied to the soil EPCs to calculate air concentrations of vapors for volatile COPCs (Table 3-1). VFs reported in the EPA, Region 9, PRGs were used (EPA 2000d).

3.3.1.2 On-Facility Surface and Subsurface Soil (Future Exposure Scenarios)

EPCs were calculated for on-facility soil for potential future exposure scenarios similar to those described above for current exposure to surface soil; however, any institutional controls and soil caps were assumed to be no longer present for future scenarios. Subsurface soil to a depth of 18 feet bgs was included in EPC calculations because future excavation activities could bring contaminated subsurface soil to the surface. Three sets of EPCs were generated for each area. The first was calculated using sample

results from 0 to 6 feet bgs, the second using results from 6 to 12 feet bgs, and the last using results from 12 to 18 feet bgs. The purpose of calculating separate EPCs for separate depth intervals is to ensure that risks associated with areas having only shallow subsurface contamination (such as from dripping treated poles) are not diluted over greater depths without contamination, while areas with deeper contamination still are characterized adequately in this baseline HHRA. Concentrations of COPCs in fugitive dust and outdoor vapors were calculated from soil EPCs as described in the previous section. The six facility areas were evaluated separately for the on-facility future exposure scenario. Note that no surface soil samples were collected from the East and West Treatment areas because of the existing infrastructure (e.g., buildings and equipment).

3.3.1.3 Off-Facility Residential Area Soils (Current and Future Exposure Scenarios)

A subset of residences and open areas near the facility was selected for sampling. Figure 3-2 shows the locations and sample types of off-facility soil samples. For residences sampled in a biased fashion, the EPC for each consists of the concentration of each detected COPC in the single composite sample collected from each area. This sample was composited from up to five locations in each residential yard. Each of the five subsamples was collected from an area that would be expected to accumulate the highest concentration of COPCs due to long-term deposition of dust from the facility (e.g., roof drip lines). Additional grab samples were collected from open areas and residences located near the facility. The EPC for these samples consisted of the concentration of each detected COPC in the sample.

Ten samples each also were collected from residences and open areas not impacted by the facility. These samples were collected from a typical residential area of Bellingham. Therefore, the samples would be impacted by typical urban sources in addition to industries located within Bellingham (E & E 2000). Biased composite samples, as described above, were collected from 10 residences and 10 open areas. Instead of calculating EPCs for each residence or open area, EPCs were calculated for all 10 residential samples combined and for all 10 open area samples combined.

3.3.1.4 Off-Facility Recreational Area Soils (Current and Future Exposure Scenarios)

Four EPC calculations were performed for the undeveloped area south of the facility. These EPCs represent the dirt footpath running along Little Squalicum Creek to Bellingham Bay, the gridded surface soil samples collected within the South Slope Area, the surface soil for the spoils piles located near Little Squalicum Creek, and the subsurface soil samples collected from the soil sampling trench near

the creek. These EPCs were used to estimate risks associated with current and future exposures to recreational users of the creek area. All EPCs were calculated as described above (EPA 1992a). Concentrations of COPCs in fugitive dust and outdoor vapors were calculated from soil EPCs as described in Section 3.3.1.1.

3.3.1.5 On- and Off-Facility Groundwater (Current and Future Exposure Scenarios)

Separate EPCs were calculated for each monitoring well in the deep aquifer because each well may represent the sole source of domestic water for current (i.e., Tilbury Cement Company wells) and potential future receptors. Use of the average may represent the best estimate of long-term exposure at a single location. The arithmetic average concentration of several rounds of data for each COPC detected in each monitoring well was used as the EPC. To calculate the average for each COPC where there were nondetected results for some of the sampling rounds, one-half the DL was used. However, when the averages using one-half the DL for nondetected values exceeded the value of the average excluding the nondetected values, the average excluding the nondetected values was used.

3.3.1.6 Off-Facility Air (Current and Future Exposure Scenarios)

EPCs for off-facility air consist of arithmetic average results of all quarterly air samples collected from each location. These EPCs were used to calculate inhalation risks for residents and recreational visitors. Air exposures to on-site workers are addressed in Appendix C. To calculate the average for COPCs where there were nondetected results for some of the sampling rounds, one-half the DL was used. However, when the averages using one-half the DL for nondetected values exceeded the value of the average excluding the nondetected values, the average excluding the nondetected values was used.

3.3.1.7 Little Squalicum Creek Surface Water and Sediment (Current and Future Exposure Scenarios)

EPCs for recreational exposures to Little Squalicum Creek sediment were calculated using the same method as was used for on-facility soil samples (EPA 1992a). The creek was divided into two sections, and two background samples also were considered. EPCs were calculated for: the sediment samples located downstream of Marine Drive; the sediment samples collected between The Oeser Company outfall and Marine Drive; a sediment sample collected directly downgradient from the Birchwood outfall (representing urban runoff not associated with The Oeser Company); and a sediment sample collected from a wetland not impacted by The Oeser Company, located northeast of the creek.

For the surface water EPC, the maximum concentration of each COPC from the four samples collected from within the creek was used. Risks and HQs were calculated using four surface water samples: Nos. SW01, SW02, SW04, and SW05. Although a background sample was collected, the results were not compared to the other samples because there were insufficient samples to make a statistically valid comparison. However, the risk associated with the background sample, No. SW07, was calculated for sake of comparison. Seep samples and the tapped spring samples were not used because these samples do not represent areas where a person would be exposed while wading.

3.3.2 Exposure Factors

The objective of this baseline HHRA was to determine the potential risks associated with reasonable maximum and average exposure conditions at The Oeser Company. The exposure factors described in this section were used to calculate reasonable maximum exposure (RME) and central tendency (CT) risk estimates. RME scenarios are intended to represent the highest exposure that could reasonably be expected to occur for a given exposure pathway at a site and are designed to represent a combination of high-end and central estimates of exposure. The CT scenarios are designed to represent average exposures only and are calculated for purposes of comparison to the RME.

To calculate potential exposure to COPCs through the identified exposure pathways, chronic daily intakes (CDIs) and lifetime average daily intakes (LADIs) for evaluating noncarcinogenic and carcinogenic effects, respectively, were calculated for each COPC. CDIs and LADIs are expressed in units of milligrams per kilogram per day (mg/kg-day). The calculation of CDIs and LADIs involves numerous estimated exposure factors, reflecting information about the behavioral characteristics of the population of interest (e.g., how frequently the population engages in an activity, how many years the population is exposed). For the majority of exposure factors used in this assessment, standardized U.S. EPA default exposure parameters were used. Exposure factors are available from the following EPA sources:

- *Interim Final Guidance: Developing Risk-Based Cleanup Levels at Resource Conservation and Recovery Act Sites in Region 10* (EPA 1998a);
- EPA, Region 10, *Supplemental Risk Assessment Guidance for Superfund* (EPA 1991c);
- *Superfund's Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposure* (EPA 1993c);
- *Exposure Factors Handbook* (EPA 1997a); and

- *Risk Assessment Guidance for Superfund, Human Health Evaluation Manual, Part E, Supplemental Guidance for Dermal Risk Assessment, Interim Guidance (EPA 2000b).*

Where possible, exposure factors recommended in *Interim Final Guidance: Developing Risk-Based Cleanup Levels at Resource Conservation and Recovery Act Sites in Region 10* (EPA 1998a) were selected for use at The Oeser Company. In addition, the EPA, Region 10, has recent regional policy documents on soil ingestion rates, dermal absorption factors, and other exposure factors. The Region 10 Resource Conservation and Recovery Act guidance (EPA 1998a) and the regional policy documents are the most current sources of exposure factors provided by the EPA, Region 10.

The potential exposure of any individual to contaminants depends on such factors as activity patterns, location-specific chemical concentrations, and other site-specific factors. The exposure factors employed in this baseline HHRA and the sources of these values are provided in TARA Table 4 (Appendix A). These exposure factors are based on default values provided in the guidance. The following sections provide additional clarification and explanation of exposure assumptions.

3.3.2.1 Residential Scenario

A residential exposure scenario was evaluated to estimate risks to residents living near The Oeser Company. A potential future residential scenario was evaluated for on-facility areas, representing risks to residents in the event that the facility closes and the site is redeveloped for residential use. This potential future scenario does not imply that such land use is likely or even plausible; however, this scenario is included in this baseline HHRA to give risk managers as much information as possible.

The exposure assumptions for the residential scenario are presented in TARA Table 4 (Appendix A). These values typically represent EPA, Region 10, default values for residential exposure scenarios. Exposure assumptions are provided for an adult resident and a child resident. Risks to adults and children were combined for carcinogens, while risks to children only were calculated for noncarcinogens. This results in the most health-protective risk estimates.

Adult and child residents may be exposed to contamination from The Oeser Company through the following exposure pathways:

- Incidental ingestion of soil;
- Dermal contact with soil;
- Ingestion of potentially contaminated home-grown produce;
- Ingestion of groundwater used as a drinking water source;

- Dermal contact with groundwater used for showering;
- Inhalation of volatiles from groundwater used for household purposes; and
- Inhalation of airborne chemicals.

Because groundwater is not used as a drinking water source or for other household needs, exposure to groundwater was evaluated for potential future exposure scenarios only.

Risks associated with inhalation of chemicals in air for off-site residents were evaluated using measured concentrations at air monitoring stations for the current residential exposure scenario. For future on-site residents, fugitive dust and vapor concentrations were modeled from soil.

The RME scenario represents a combined adult-child receptor living at the site for 30 years (24 years as an adult and six years as a child). The average scenario evaluates an adult receptor living at the site for nine years. The complete list of exposure factors for the residential receptor and the sources of these values are provided in TARA Table 4 (Appendix A).

3.3.2.2 Industrial Scenario

An industrial exposure scenario was evaluated to estimate risks to current and future workers at The Oeser Company. The purpose of this scenario is to evaluate risks associated with chemical contamination in soil and water at the facility.

The exposure assumptions for the industrial scenario are presented in TARA Table 4 (Appendix A). These values represent EPA, Region 10, default values for industrial exposure scenarios.

Workers may be exposed to contamination from The Oeser Company through the following exposure pathways:

- Incidental ingestion of soil;
- Dermal contact with soil;
- Ingestion of groundwater used as a drinking water source; and
- Inhalation of airborne chemicals.

Groundwater was evaluated for the current (i.e., Tilbury Cement Company wells) and potential future (on-facility) scenarios.

Risks associated with inhalation of chemicals in air were modeled as fugitive dust and volatiles emitted from soil. Air monitoring data collected on-site was used to evaluate inhalation risks to workers, as shown in Appendix C.

The RME scenario represents an on-site worker exposure of 25 years. The average scenario evaluates an on-site worker exposure of six years. Workers are assumed to receive one-half their daily exposure to water while at work. In addition, one-half of their daily air inhalation is assumed to occur at work. The complete list of exposure factors for the industrial receptor and the sources of these values are provided in TARA Table 4 (Appendix A).

3.3.2.3 Recreational Scenario

A recreational exposure scenario was used to estimate risks to individuals exposed to contamination that may be in undeveloped areas south of The Oeser Company, in and near Little Squalicum Creek.

The exposure assumptions for the recreational scenario are presented in TARA Table 4 (Appendix A). No default exposure factors for recreational exposure scenarios are available from the EPA; consequently, these factors were developed based on best professional judgment regarding site conditions. An explanation of these exposure assumptions is provided below.

Recreational users may be exposed to contamination from The Oeser Company through the following exposure pathways:

- Incidental ingestion of soil;
- Dermal contact with soil;
- Dermal contact with sediment in Little Squalicum Creek;
- Dermal contact with surface water in Little Squalicum Creek; and
- Inhalation of airborne chemicals.

Risks associated with inhalation of chemicals in air were evaluated using measured concentrations at air monitoring stations for the current recreational visitor exposure scenario. In addition, inhalation of particulates and vapors modeled from soil were considered for Little Squalicum Creek areas.

The recreational exposure scenario was used to assess risks to an adolescent-age receptor (8 to 18 years old). Risks to older or younger individuals (i.e., adults or children) likely would be similar. The RME recreational scenario assumes that individuals would be exposed for the entire 11-year period associated with the selected age group. A nine-year exposure duration (ED) was used for the average scenario, consistent with the residential average scenario. A body weight of 49 kg was used, representing the mean body weight for the age group (EPA 1997a).

In the RME recreational scenario, individuals were assumed to visit the site two days per week throughout the year (for a total of 104 days per year) and to spend 4 hours at the site during each visit. These values should be sufficiently conservative to represent high-end estimates of any future recreational land use. In the average scenario, individuals were assumed to visit the site one day per week for six months per year (for a total of 26 days per year) and to spend 2 hours at the site during each visit.

Individuals were assumed to come into contact with Little Squalicum Creek surface water and sediment. Because the creek is very shallow, only exposure to lower legs and feet were considered. Skin surface areas were estimated as the 90th percentile (for RME) and 50th percentile (for average) of whole body skin surface areas for 12- to 13-year-old boys multiplied by 15%, representing “half legs” exposure (EPA 1997a). These ages were selected to represent the middle of the 8- to 18-year-old age group.

Skin surface areas for evaluation of dermal contact with soil were calculated in a manner similar to contact with surface water and sediment, except that the head, hands, arms, and half legs were assumed to be exposed. The soil-to-skin adherence factors recommended by the EPA, Region 10, for child exposures were used for this exposure pathway (EPA 1998a).

3.4 EXPOSURE ASSESSMENT UNCERTAINTIES

Several factors could cause the estimated exposure levels to differ from the actual exposures experienced by an individual at the site. The purpose of this section is to identify these factors; to discuss the potential effects of the factors on the exposure estimates; and, where possible and appropriate, to estimate the degree of confidence that should be placed on the various assumptions and parameter estimates that make up the exposure estimates.

3.4.1 Environmental Sampling

Samples were intended to characterize the nature and extent of contamination at specific subareas on site. As a result, the samples were collected near sources. The samples were collected from biased areas at the residences to represent the worst case. Samples collected in this manner provide considerable information about The Oeser Company and the residences, but are not statistically representative of the contamination that may be present as a whole. The extent to which these samples overestimate or underestimate potential exposures is unknown, but directed collection of environmental samples is most likely to overestimate potential exposure.

3.4.2 Exposure Point Concentrations

The UCL of the arithmetic mean of the COPC concentration at the site may not be appropriate unless individual exposures are equally likely across all parts of a subarea during time periods similar to those during which the samples were collected. The EPA (1993b) recommends that methods that account for the spatial distribution of contamination should be incorporated into the risk assessment process. Another concern regarding the UCL is that many of the statistical assumptions in its calculation are violated. For example, use of the 95% UCL requires that the sample locations be chosen randomly and that the data are either normally or lognormally distributed. The manner in which nondetected results are incorporated into the calculation of the 95% UCL also affects the result. For example, for nondetected dioxin/furan congeners or PAH compounds, one-half the DL times the TEF or RPF was used. In some cases, 2,3,7,8-TCDD TEQs and B(a)P equivalents were calculated when individual dioxins/furans and PAHs were analyzed, but were not detected. This likely overestimates the concentration of 2,3,7,8-TCDD TEQs and B(a)P equivalents; however, it is not possible to assure or to quantify that assumption. It is also possible that some of the compounds were present at concentrations greater than one-half the DL, in which case, the assumption may be nonconservative.

Additionally, for COPCs in various media, there were insufficient data available to calculate UCLs; therefore, EPCs are based on maximum detected concentrations. This overestimates the range of possible exposure concentrations. Overall, the methodology used to calculate EPCs is likely to overestimate potential exposures.

Uncertainties associated with EPCs may be reduced by increased sampling. For example, there is less uncertainty associated with EPC estimates for on-site soil samples than for off-facility soil samples because a larger number of samples were obtained on site versus off site. It is unknown whether this uncertainty would result in a higher or lower risk, although the number of off-site samples is considered sufficient for estimating EPCs with reasonable confidence.

3.4.3 Contaminant Migration Modeling

The models used to evaluate inhalation of fugitive dust and vapors from soil employed EPA default input parameters along with site- or area-specific values designed to represent a conservative screening-level approach. The results are likely to overestimate the potential air pathway exposure.

An additional exposure pathway, which required modeling from soil, was uptake of dioxin into home-grown produce. The model used was taken from recent EPA (1998c) guidance and should provide a conservative estimate of contaminant uptake from soil. This pathway was included to assess the

potential risks to residents near the facility who consume garden produce. The estimated risks for this pathway are discussed in Section 5.3.2.1.

Some specific uncertainties are related to dioxin uptake from soil. Most gardens observed during the field sampling included nonnative material. Therefore, assuming that produce was grown in native soil may overestimate risk. The biased residential samples were collected from high-impact areas; therefore, actual garden soil concentrations, assuming native material was used for gardening, are likely to be lower. Also, surface soil samples were collected from a depth no greater than 2 inches bgs, whereas root vegetables (which were assumed for modeling purposes) likely grow at greater depths to some extent. Therefore, the surface soil may be more highly contaminated than deeper soil; thus, potential uptake may be overestimated. Alternatively, historical deposition may have been greater than recent deposition; this combined with infiltration and leaching over time may result in higher concentrations in soil at lower depths and an underestimation of potential uptake by plants.

3.4.4 Exposure Parameters

The principal uncertainty regarding the exposure estimation calculations is associated with the selection of appropriate parameter values. The values used are included in TARA Table 4 (Appendix A). Individual parameter values were selected so that the overall pathway exposure estimates would approximate RME and average, or CT, exposure. Overall, the use of such exposure parameters serves to overestimate potential exposures. The exposure parameters used in calculating the exposure estimates were obtained primarily from EPA guidance. These values are intentionally conservative and likely overestimate average or typical exposures. Overall, EPA's intention in providing default exposure factors is to err on the health-protective, or conservative, side; however, uncertainties may ultimately over- or underestimate individual exposure.

3.4.5 Future Land Use

Future residential scenarios were assumed at The Oeser Company; however, only a portion of the facility actually is zoned as residential. In addition, uncertainties arise because of the difficulties in predicting future land uses. The assumption of future residential development at The Oeser Company may overestimate the true future risks at the site if future land use does not include residences.

3.4.6 Steady-State Assumption

All exposure calculations used in this baseline HHRA were performed assuming that the chemical concentrations in the affected media remain at a steady state (i.e., remain constant for the duration of the exposure period). The steady-state assumption may be appropriate for highly chlorinated compounds such as dioxins/furans and PCP. However, the steady-state assumption is inappropriate for the more soluble and mobile COPCs, such as benzene and naphthalene. These compounds migrate or may degrade over time. For simplicity, the health-protective steady-state assumption was used, resulting in overestimation of the potential exposures. Steady-state assumptions are common practice in risk assessment because the state of the science does not allow for reliable alternatives. In addition, air concentration data derived from the air monitoring stations probably do not represent steady-state concentrations. These concentrations can vary greatly depending on local atmospheric conditions, such as wind speed, wind direction, and precipitation. Facility operations also will influence contaminant concentrations greatly. The risk attributed to detected air concentrations may be overestimated or underestimated depending on how close these values are to the actual average air concentrations.

3.4.7 Bioavailability

It was conservatively assumed that 100% of COPCs associated with the affected media were bioavailable. This assumption may overestimate risk because the actual amount of a chemical in environmental media that is bioavailable is uncertain, but it is likely to be less than 100%. The exposure assessment estimates the amount of a chemical, in terms of LADI or CDI, that is taken into the body through a variety of exposure pathways. However, intake is not equivalent to bioavailability. Once in the body, the chemical must be released from the medium it is associated with (e.g., soil) and then be absorbed into the bloodstream. These processes vary in efficiency depending on the properties of the medium in question and each individual chemical. Without additional, site-specific data, the assumption of 100% bioavailability is reasonable and appropriate.

Table 3-1

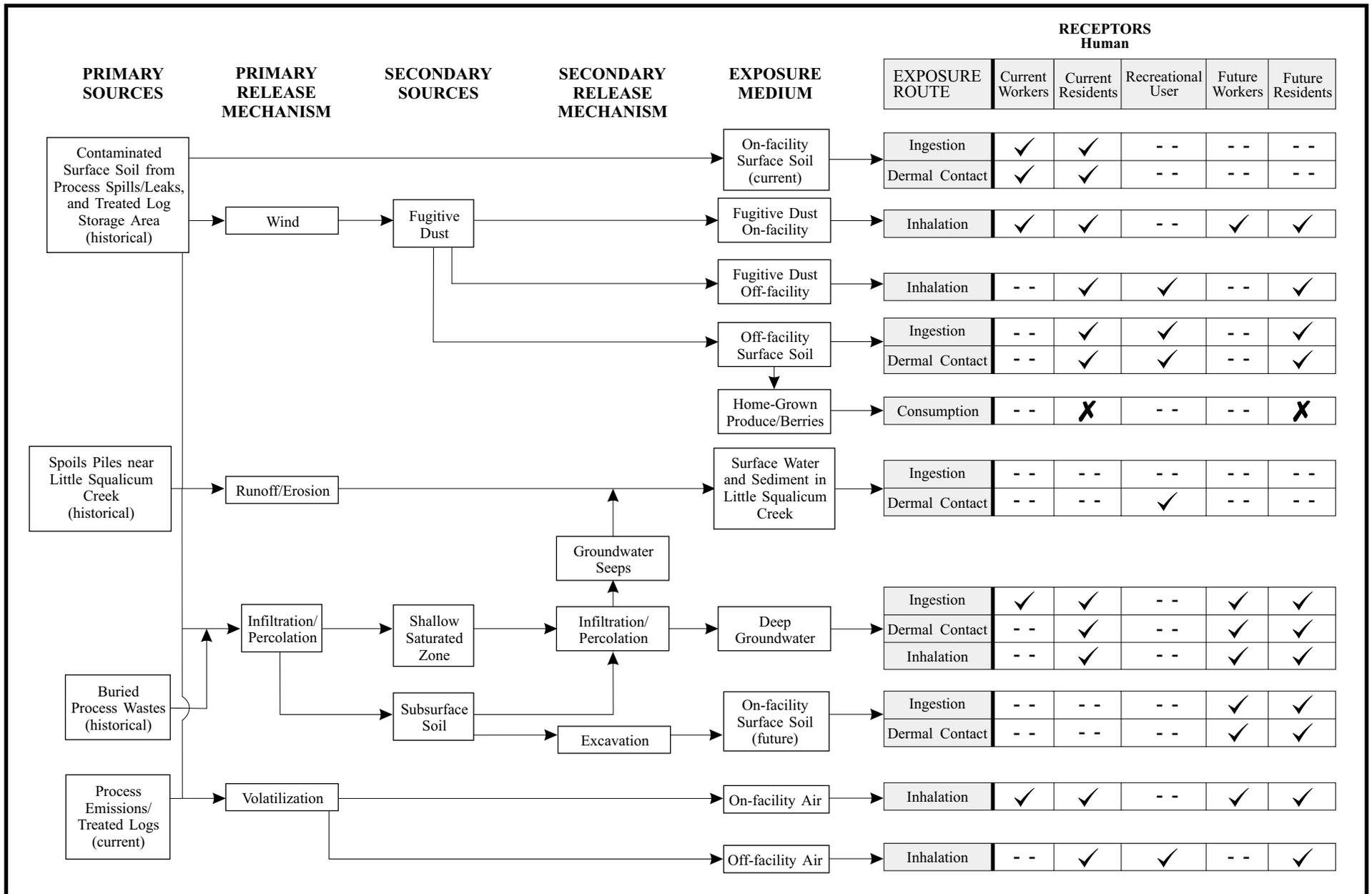
**Chemical-Specific Volatilization
Factors for Soil COPCs
The Oeser Company
Bellingham, Washington**

7,12-Dimethylbenz(a)anthracene	NA
7H-Dibenzo(c,g)carbazole	NA
B(a)P Equivalent	NA
Benzo(a)anthracene	NA
Benzo(a)pyrene	NA
Benzo(b)fluoranthene	NA
Benzo(j)fluoranthene	NA
Benzo(k)fluoranthene	NA
Chrysene	2.70E+06
Dibenzo(a,e)pyrene	NA
Dibenzo(a,h)anthracene	NA
Dibenzo(a,h)pyrene	NA
Dibenzo(a,i)pyrene	NA
Dibenzo(a,j)acridine	NA
Dibenzo(a,l)pyrene	NA
Indeno(1,2,3-cd)pyrene	NA
1,2,3,4,6,7,8-HpCDD	NA
1,2,3,4,6,7,8-HpCDF	NA
1,2,3,4,7,8,9-HpCDF	NA
1,2,3,4,7,8-HxCDD	NA
1,2,3,4,7,8-HxCDF	NA
1,2,3,6,7,8-HxCDD	NA
1,2,3,6,7,8-HxCDF	NA
1,2,3,7,8,9-HxCDD	NA
1,2,3,7,8,9-HxCDF	NA
1,2,3,7,8-PeCDD	NA
1,2,3,7,8-PeCDF	NA
2,3,4,6,7,8-HxCDF	NA
2,3,4,7,8-PeCDF	NA
2,3,7,8-TCDD	NA
2,3,7,8-TCDF	NA
Dioxin TEQ	NA
OCDD	NA
OCDF	NA
Total EPH	NA
Total VPH	NA
TPH-Diesel	NA
TPH-Gas	NA
2-Methylnaphthalene	4.30E+04
Acenaphthene	1.80E+05
Fluoranthene	NA
Fluorene	2.70E+05
Naphthalene	4.30E+04
Phenanthrene	NA
Pyrene	3.10E+06
Benzidine	NA
Carbazole	NA
Dibenzofuran	6.50E+05
Pentachlorophenol	NA

Source: Preliminary Remediation Goals (EPA 2000d)

Key:

NA = Not available.



LEGEND
 ✓ = Complete Pathway Evaluated in the Risk Assessment
 ✗ = Evaluated in the Screening Process, but not quantified in the Risk Assessment
 -- = Incomplete Exposure Pathway

THE OESER COMPANY
 SUPERFUND SITE
 REMEDIAL INVESTIGATION
 Bellingham, Washington
Past and Present Conditions

Figure 3-1
 HUMAN HEALTH
 CONCEPTUAL SITE MODEL

Date: 1/28/02
 Drawn by: AES
 10:START-2\01030016\fig 3-1

Figure 3-2

THE OESER COMPANY
SUPERFUND SITE

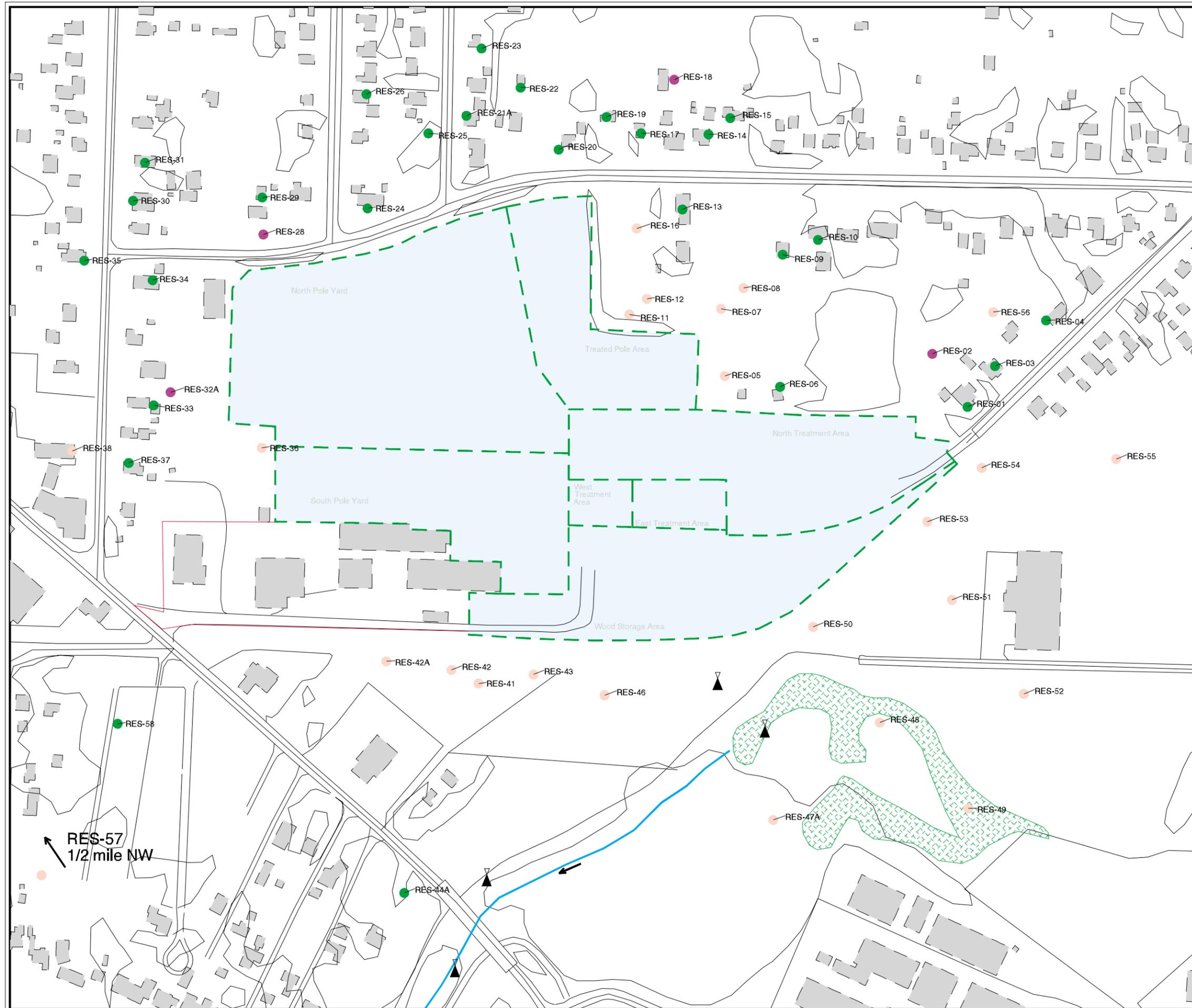
Bellingham, Washington

Human Health Risk Assessment

Classification of Off-Facility
Surface Soil Samples

LEGEND

- Open Area, Residential Properties
- Open Area, Non-Residential Properties
- Biased Sample, Residential Properties



RES-57
1/2 mile NW

4. TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to compile toxicity data for the COPCs identified at The Oeser Company and to estimate the relationship between the amount of exposure to a COPC (i.e., dose level) and the likelihood of adverse effects. Qualitative descriptions of the potential toxic properties of the COPCs are provided in Appendix B.

4.1 TOXICITY VALUES

Toxicity values that describe the relationship between exposure to a contaminant and the associated increased adverse effects were compiled. The following sources were used to obtain toxicity values:

- **Integrated Risk Information System (IRIS) Computer Database (EPA 2000a).** IRIS is the preferred source of information because this database contains the most recent toxicity values reviewed extensively by the EPA;
- **Health Effects Assessment Summary Tables (HEAST; EPA 1997b).** These tables were consulted if a toxicity value was not available in IRIS (EPA 2000a). The EPA compiled these values for use in risk assessments. Toxicity values presented in HEAST are not reviewed as rigorously as those presented in IRIS; and
- **The EPA's National Center for Environmental Assessment (NCEA).** Provisional toxicity values from the NCEA may be used when values are not available in IRIS or HEAST.

Toxicity values are presented in TARA Tables 5.1 and 5.2 for noncarcinogens and TARA Tables 6.1 and 6.2 for carcinogens (Appendix A). Toxicity profiles for the major risk drivers are presented in Appendix B.

4.1.1 Categorization of Chemicals as Carcinogens or Noncarcinogens

Carcinogenic and noncarcinogenic health effects were evaluated quantitatively in this baseline HHRA. The endpoints for these two different types of effects are assessed differently because the mechanism(s) by which chemicals cause cancer is (are) fundamentally different from the process(es) by

which noncarcinogenic effects are caused. The principal difference reflects the assumption that noncancer effects are assumed to exhibit a threshold dose below which no adverse effects occur, whereas no such threshold generally is assumed to exist for carcinogenic effects.

As used here, the term *carcinogen* refers to any chemical for which there is sufficient evidence that exposure may result in continuing uncontrolled cell division with the potential to metastasize or to invade other tissues (i.e., cancer) in humans or animals. Conversely, the term *noncarcinogen* refers to any chemical for which the carcinogenic evidence is negative or insufficient. These definitions are under continuous review by the EPA and are subject to change as new information becomes available and the weight-of-evidence is modified. Because exposure to some chemicals may result in carcinogenic and noncarcinogenic effects, both endpoints associated with a COPC were evaluated quantitatively in this baseline HHRA.

The likelihood that an agent is a human carcinogen is evaluated using the EPA's weight-of-evidence classification (EPA 1986). Data derived from human and animal studies are reviewed as: (1) sufficient, (2) limited, (3) inadequate, (4) no data, or (5) evidence of no effect. The weight-of-evidence classifications are presented in Table 4-1.

4.1.2 Assessment of Carcinogens

To evaluate cancer risks, the EPA provides oral slope factors (SFs), which are expressed as risks per (mg/kg-day)⁻¹. However, toxicity values for carcinogenic effects sometimes are expressed in terms of risk per unit concentration of the substance in the medium where human contact occurs. Inhalation unit risks typically are expressed as risk per microgram per cubic meter (µg/m³). Consequently, inhalation SFs were derived from inhalation unit risks (expressed as [µg/m³]⁻¹) by assuming a body weight of 70 kg and an inhalation rate of 20 cubic meters per day (m³/day). When necessary, oral SFs were derived from drinking water unit risks by assuming a 70-kg body weight and a water ingestion rate of 2 liters per day. The standardized duration assumption for SFs is continuous lifetime exposure. Therefore, when no absorption adjustment is required:

$$\frac{\text{Inhalation SF}}{(\text{mg/kg}\&\text{day})^{\&1}} = \frac{\text{Air Unit Risk } (\mu\text{g}/\text{m}^3)^{\&1} \times 70 \text{ kg} \times 10^3 \mu\text{g}/\text{mg}}{20 \text{ m}^3/\text{day}}$$

$$\frac{\text{Oral SF}}{(\text{mg/kg}\&\text{day})^{\&1}} = \frac{\text{Water Unit Risk } (\mu\text{g}/\text{L})^{\&1} \times 70 \text{ kg} \times 10^3 \mu\text{g}/\text{mg}}{2 \text{ L}/\text{day}}$$

4.1.3 Assessment of Noncarcinogens

The potential for adverse health effects associated with noncarcinogens (e.g., organ damage, adverse immunological effects, birth defects, and respiratory and skin irritation) usually is assessed by comparing the estimated average daily intake (i.e., exposure dose) to a reference dose (RfD). RfDs typically are expressed in terms of a person's daily intake with respect to his or her weight, specifically in units of milligrams of contaminant taken in per kilogram of body weight per day (mg/kg-day). The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily human intake, including sensitive subgroups, which should not result in an appreciable risk of deleterious effects.

RfDs are developed for specific exposure routes (i.e., oral, dermal, and inhalation). The EPA frequently provides noncancer toxicity criteria for inhalation exposure as reference concentrations (RfCs) rather than RfDs. RfCs are derived using the same principles as those for oral RfDs; however, the analysis of inhalation exposures is more complex because of the dynamics of the respiratory system and its diversity across species and because of differences in the physico-chemical properties of contaminants (EPA 1989). RfCs are expressed as a concentration in air (milligrams per cubic meters [mg/m³]) for continuous daily exposure. However, for risk assessment purposes, RfCs were converted to corresponding inhalation RfDs (RfDi). A human adult body weight of 70 kg and an inhalation rate of 20 m³/day are used to convert an RfC to an RfDi:

$$\text{RfDi (mg/kg-day)} = \text{RfC (mg/m}^3\text{)} \times 20 \text{ (m}^3\text{/day)} \times 1/70 \text{ (kg)}$$

RfDs and RfCs may be derived for chronic and subchronic exposures. In this baseline HHRA, chronic RfDs were employed to evaluate all potential noncancer health effects.

4.1.4 Route-to-Route Extrapolation of Reference Doses and Slope Factors

4.1.4.1 Oral-to-Dermal Extrapolation

Because the EPA has not promulgated dermal route toxicity values for most chemicals, oral route RfDs and SFs were modified using gastrointestinal absorption data to evaluate exposures to substances by the dermal route. Oral toxicity values are used as the preferred alternative to not evaluating the potential dermal effects associated with these compounds at all. Such route-to-route extrapolation has a scientific basis because once a chemical is absorbed, its distribution, metabolism, and elimination patterns (biokinetics) are usually similar, regardless of the exposure route. In addition to ultimate absorption into the blood compartment, metabolism and perhaps other factors contribute to receptor responses incurred

by exposure via different routes (e.g., ingestion v. dermal exposures). Therefore, using route-to-route extrapolation introduces uncertainties which may be either conservative or nonconservative. The scientific basis for route-to-route extrapolation fails for chemicals which may have effects at the point of contact. However, for many of these compounds, dermal toxicity values are not available. Uncertainties associated with route-to-route extrapolation are discussed in Section 4.2.3.

4.1.4.2 Oral-to-Inhalation Extrapolation

Oral RfDs and SFs were used as inhalation toxicity values if inhalation route RfDs and SFs were not available for organic COPCs. Uncertainties associated with route-to-route extrapolation are discussed in Section 4.2.3.

4.1.5 Other Issues

Some COPCs associated with The Oeser Company required evaluation using toxicity values or methods not provided in standard sources. Following are the special circumstances used in this baseline HHRA.

4.1.5.1 Petroleum Hydrocarbons

Ecology's policy on total petroleum hydrocarbons is outlined in the newly revised MTCA (Chapter 173-340 WAC) and is considered the most applicable methodology for evaluating petroleum fractions (Ecology 2001a). The toxicity profile in Appendix B summarizes the toxicity of TPH mixtures. Toxicity values are not available for petroleum hydrocarbons, such as creosote, diesel, or gasoline, which are complex mixtures of many structurally related chemicals. Current methodology for evaluating the toxicity of petroleum hydrocarbons recommends assessing risks associated with ranges of hydrocarbon fractions and with specific chemical constituents of petroleum (such as PAHs and benzene, toluene, ethylbenzene, and xylenes [BTEX]). The methods described in MTCA were applied to this risk assessment where fractionated data for TPH were available. When fractionated data were not available, the previous MTCA method for assessing risk to TPH mixtures was used, which involved using surrogate toxicity values for TPH constituents, such as PAHs.

4.1.5.2 Dioxins/Furans

The only polychlorinated dibenzo-p-dioxin (PCDD)/polychlorinated dibenzofuran (PCDF) with a cancer SF listed in IRIS or HEAST is 2,3,7,8-TCDD. For risk assessment purposes, the concentrations of the other dioxins/furans are converted to equivalent concentrations of 2,3,7,8-TCDD using TEFs. The dioxins/furans then are evaluated as if they are the single chemical 2,3,7,8-TCDD.

Internationally accepted TEFs were developed in 1988 (NATO/CCMS 1988). In 1997, a working group organized by the World Health Organization (WHO) reevaluated the TEF values based on existing literature (Vanden Berg et al. 1998). The EPA, Region 10, adopted the revised WHO TEFs for PCDDs and PCDFs. The updated TEFs are presented in Table 4-2. A nonzero value was assigned to all PCDFs and PCDDs with chlorines present in positions 2,3,7, and 8. The TEF assigned to 2,3,7,8-TCDD is 1 because the remaining congeners are less toxic and the assigned TEFs represent a fraction of 2,3,7,8-TCDD toxicity. Congeners without 2,3,7,8-chlorine substitution have a TEF of 0.

In general, assessment of human health risks resulting from exposure to dioxins/furans relies not on individual data for the 210 isomers but on a value derived using congener-specific concentrations and TEFs; the 2,3,7,8-TCDD equivalency value; or the TEQ. The TEQ represents the toxicity-related value that combines the toxicities for all dioxin-like PCDDs/PCDFs and expresses them as a sample concentration term for only 2,3,7,8-TCDD. In other words, the concentration of 17 congeners is expressed as a single value (EPA 1989).

The TEQ is calculated by multiplying the measured concentration of each congener by its assigned TEF. If more than one congener is present, then the TEF-adjusted concentrations are summed, and this value is referred to as the TEQ for the sample. No standard methodology for assessing noncancer health effects of dioxins and furans is available from the EPA.

4.1.5.3 Carcinogenic Polycyclic Aromatic Hydrocarbons

The only PAH with a cancer SF listed in IRIS is B(a)P. Similar to the dioxins/furans, the concentrations of other cPAHs are converted to equivalent concentrations of B(a)P (B[a]P equivalents) using RPFs. RPFs for six additional PAHs are provided by the NCEA (EPA 1993a). These PAHs include:

- Benzo(a)anthracene (RPF = 0.1);
- Benzo(b)fluoranthene (RPF = 0.1);
- Benzo(k)fluoranthene (RPF = 0.01);

- Dibenzo(a,h)anthracene (RPF = 1);
- Indeno(1,2,3-cd)pyrene (RPF = 0.1); and
- Chrysene (RPF = 0.001).

RPFs were used to evaluate potential cancer risks associated with these chemicals.

Additional RPFs are available from the California Environmental Protection Agency (CalEPA) for many other PAHs (CalEPA 1996). These values were used for any of the following PAHs found at The Oeser Company. Although analysis for all of these compounds is not possible because of a lack of reference standards, these PAHs were targeted as TICs:

- Benzo(j)fluoranthene (RPF = 0.1);
- Dibenzo(a,j)acridine (RPF = 0.1);
- Dibenzo(a,h)acridine (RPF = 0.1);
- 7H-Dibenzo(c,g)carbazole (RPF = 1);
- Dibenzo(a,e)pyrene (RPF = 1);
- Dibenzo(a,h)pyrene (RPF = 10);
- Dibenzo(a,i)pyrene (RPF = 10);
- Dibenzo(a,l)pyrene (RPF = 10);
- 7,12-Dimethylbenzo(a)anthracene (RPF = 20);
- 1,6-Dinitropyrene (RPF = 10);
- 1,8-Dinitropyrene (RPF = 1);
- 3-Methylcholanthrene (RPF = 2);
- 5-Methylchrysene (RPF = 1);
- 5-Nitroacenaphthene (RPF = 0.01);
- 6-Nitrochrysene (RPF = 10);
- 2-Nitrofluorene (RPF = 0.01);
- 1-Nitropyrene (RPF = 0.1); and
- 4-Nitropyrene (RPF = 0.1).

The RPFs for 7,12-dimethylbenzo(a)anthracene; 5-methylchloroanthracene; and 5-nitroacenaphthene were calculated as the ratio of the CalEPA expedited potency factors to the CalEPA oral potency factor for B(a)P. B(a)P equivalents are calculated by multiplying the measured concentration of each cPAH by its assigned RPF. If more than one compound is present, then the

RPF-adjusted concentrations are summed. Nitrogen-containing PAHs were not detected in any media except groundwater, where 1-nitropyrene was detected in one sample. All other nitrogen-containing PAHs were not evaluated further in the risk assessment.

4.2 TOXICITY ASSESSMENT UNCERTAINTIES

A degree of uncertainty is inherent in the numerical toxicity values used in any risk assessment, reflecting the large number of assumptions and calculations associated with deriving SFs and RfDs. The principal sources of uncertainty are described below.

4.2.1 Carcinogenic Toxicity Assessment Assumptions

Bioassay and epidemiological studies would require tens of thousands of animals or humans to determine whether a chemical is carcinogenic at low doses. The estimated SF is derived from several critical factors, including the relationship between tumor location, time to appearance, and proportion of animals exhibiting tumors. Because animal bioassay or human epidemiological data usually are insufficient to directly estimate SFs at low doses, carcinogenic extrapolation models are used to estimate low-dose SFs. These models are based on the assumption that a small but finite risk of cancer is associated with any dose above 0. The EPA also assumes that the dose-response relationship is linear at low doses, in contrast to other toxic effects for which thresholds are assumed to exist.

The high-dose-to-low-dose extrapolation model favored by the EPA and other federal regulatory agencies is the linearized multistage model. The EPA uses the statistically derived 95% UCL on the slope of the dose response curve to derive SFs, rather than a maximum likelihood value. Based on human epidemiological and animal data, the EPA considers cancer to follow a series of discrete stages (i.e., initiation, promotion, and progression) that ultimately can result in uncontrolled cell proliferation. Consistent with this conclusion, the use of the linearized multistage model permits an upper-bound estimate of the SF. However, compelling scientific arguments can be made for several other extrapolative models that would result in significantly reduced values for SFs. Therefore, most EPA-promulgated SFs represent upper-bound values based on animal data, which should not necessarily be interpreted as equivalent to actual human cancer potencies. Using these values likely will overestimate risk.

4.2.2 Noncarcinogenic Toxicity Assessment Assumptions

Key assumptions used in assessing the likelihood of noncarcinogenic effects are that threshold doses exist below which various noncarcinogenic effects do not occur and that the occurrence or absence of noncarcinogenic effects can be extrapolated between species and occasionally between routes of exposure and over varying EDs. The threshold assumption appears to be sound for most noncarcinogens, based on reasonably good fits of experimental data to the usual dose response curves.

Other assumptions generally appear to be true to varying degrees. For example, the effects observed in one species or by one route of exposure may not occur in another species or by another route. The noncancer effects may occur at higher or lower doses because of differences in the biokinetics of a compound in different species or when exposure occurs by different routes. The uncertainty in these assumptions is taken into account by using uncertainty factors (UFs), which reflect uncertainty associated with species-to-species extrapolation and include safety factors to protect sensitive individuals. In addition to UFs, a modifying factor (MF) is applied to reflect a qualitative professional assessment of additional scientific uncertainties in the critical study and in the entire database. The UFs and MFs used by the EPA are health-protective; consequently, the resulting RfDs are likely to be health-protective.

A potentially nonconservative uncertainty is the lack of a scientific method for evaluating child-specific risks based on age-related physiological responses to exposure to chemicals in the environment. Such uncertainties are likely to be reduced in the future as scientific understanding increases.

Some uncertainties relate to specific compounds. For example, while it is known that dioxins/furans have noncancer adverse effects (e.g., hormonal disruption) there is no established scientific method for quantifying or predicting such effects or their potential magnitude on receptors.

4.2.3 Route-to-Route Extrapolation of Reference Doses and Slope Factors

Route-to-route extrapolation of RfDs and SFs adds another source of uncertainty to the risk estimates. Such extrapolation may result in either underestimation or overestimation of the true risks for the extrapolated route. Although this practice adds uncertainty to the risk assessment process, it is preferable to omitting exposure to a chemical by a route for which no RfD or SF is available from the quantitative risk assessment, which would lead to underestimation of the overall risks posed by the chemical.

4.2.4 Perspective on Toxicity Assessment Uncertainties

The toxicity assessment process compensates for the basic uncertainties described above by using UFs and MFs when assessing noncarcinogens and by using the 95% UCL from the linearized multistage model for assessing carcinogens. These health-protective approaches ensure that the toxicity values used in the risk estimation process are unlikely to underestimate the true toxicity of a chemical.

Table 4-1

**Weight-Of-Evidence Classifications for Chemical Carcinogenicity
The Oeser Company
Bellingham, Washington**

Group	Description
A	Human Carcinogen
B	Probable Human Carcinogens B1 Limited human data are available B2 Sufficient evidence in animals and no evidence in humans
C	Possible Human Carcinogen
D	Not Classifiable
E	Evidence of Noncarcinogenicity for Humans

Source: EPA 1986.

Table 4-2

**2,3,7,8-TCDD Toxicity Equivalency Factors
The Oeser Company
Bellingham, Washington**

Congener	TEF
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	1
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.0001
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.0001

Source: Vanden Berg et al. 1998.

Key:

HpCDD = Heptachlorodibenzo-p-dioxin.
 HpCDF = Heptachlorodibenzofuran.
 HxCDD = Hexachlorodibenzo-p-dioxin.
 HxCDF = Hexachlorodibenzofuran.
 OCDD = Octachlorodibenzo-p-dioxin.
 OCDF = Octachlorodibenzofuran.
 PeCDD = Pentachlorodibenzo-p-dioxin.
 PeCDF = Pentachlorodibenzofuran.
 TCDD = Tetrachlorodibenzo-p-dioxin.
 TCDF = Tetrachlorodibenzofuran.
 TEF = Toxicity equivalency factor.

5. RISK CHARACTERIZATION

5.1 INTRODUCTION

This section integrates the information developed in the exposure assessment and toxicity assessment sections to identify the COCs and to obtain estimates of the potential risks posed to human health at The Oeser Company Superfund Site. The purpose of this section is to present the key findings of the risk assessment and to put them into perspective with respect to assumptions and uncertainties. This section is organized as follows: first, the methods for obtaining risk estimates for carcinogens and noncarcinogens are presented; next, the risk estimates associated with the various receptors and associated exposure pathways are summarized; finally, uncertainties associated with the risk calculations are presented. Throughout this section, the strengths and weaknesses of various aspects of the risk characterization are highlighted. The central component of this section is somewhat repetitive because risk estimates for so many different site areas, receptors, and exposure media are presented; however, presentation of this information within the main body of the text is critical to expressing the potential human health risks associated with the site.

5.2 RISK ESTIMATION METHODS

5.2.1 Potential Cancer Risks

Cancer risks were assessed by multiplying the estimated LADI or absorbed dose of a carcinogen by its SF. This linear relationship is valid only at low risk levels (i.e., cancer risks less than 1×10^{-2}).¹

The calculated risk is expressed as the probability of an individual developing cancer over a lifetime and is an estimated upper-bound, incremental probability. That is, the actual risk is likely to be no more than, and probably less than, the calculated risk.

¹For risks greater than this level, the one-hit equation is recommended by the EPA (1989):

$$\text{Cancer risk} = 1 - \exp(-\text{LADI} \times \text{SF})$$

Where: exp = The exponential.
LADI = Lifetime average daily intake.
SF = Slope factor.

Cancer risks were estimated separately for exposure to each chemical or range of petroleum hydrocarbon fractions for each exposure pathway and then were summed across all exposure pathways for each medium (i.e., air, water, soil, and groundwater) for each potentially exposed population. This process was performed for each receptor group evaluated at The Oeser Company Superfund Site. A risk calculation spreadsheet provided by the Washington State Department of Ecology was used to estimate risks for exposure to petroleum hydrocarbon fractions and TPH constituents. Concentrations of BTEX were subtracted from the appropriate hydrocarbon fractions to avoid double-counting risk from these chemicals, as per MTCA guidance (Ecology 2001a)..

5.2.2 Noncarcinogenic Effects

The potential for adverse effects resulting from exposure to noncarcinogens was assessed by comparing the COPC-specific CDI to its chronic RfD. This comparison was made by calculating the ratio of the estimated CDI to the corresponding RfD to yield an HQ:

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

For example, if the daily intake of a contaminant is equal to the RfD, then the HQ is 1. If the daily intake is less than or greater than the RfD, then the HQ is less than or greater than 1, respectively. HQs for individual chemicals TPH fraction groupings were summed to yield hazard indices (HIs). HQs are presented separately for each evaluated receptor (e.g., site visitors and workers). The receptor-specific HQs then were summed across chemicals and exposure pathways for each scenario. HQs for petroleum hydrocarbons were determined using fractionated data, when available. Concentrations of BTEX were subtracted from the appropriate hydrocarbon fraction groups to avoid double-counting risk from these chemicals, as per MTCA guidance (Ecology 2001a).

5.3 RISK ESTIMATES

Estimates of exposures and associated risks for the scenarios evaluated in this baseline HHRA are presented in Tables 5-1 to 5-15. These tables summarize the information presented in TARA Tables 7 and 8 (E & E 2001). In this section, the risk estimates are summarized and discussed separately for current and future scenarios for each medium. Risks associated with soil pathways are discussed separately from those associated with groundwater. Risk for air exposure pathways associated with soil pathways (i.e., inhalation of fugitive dust) is presented with soil risks. Risk for air exposure pathways

associated with groundwater (i.e., inhalation of vapors from household groundwater use) is presented with groundwater risks. For scenarios having risk greater than regulatory benchmarks, the COPCs contributing the majority of risk are identified. For the sake of brevity, only the RME scenarios are summarized. CT scenarios, provided for comparison purposes only, are presented in TARA Tables 7 and 8 where available (E & E 2001).

5.3.1 Risk Management

Federal environmental laws and policies recognize that estimates of very small levels of risk are insignificant. The concept of *de minimis* risk refers to levels below which risks are so small that they are not of concern. In risk management, the EPA Superfund program recognizes that estimated excess cancer risks less than 1×10^{-6} are generally *de minimis* and that risks greater than 1×10^{-4} are generally unacceptable. The need for remedies for sites posing cancer risks between 1×10^{-6} and 1×10^{-4} is considered on a site-by-site basis.

For evaluating noncarcinogenic effects, the EPA defines acceptable exposure levels as those to which the human population, including sensitive subgroups, may be exposed without adverse effects during a lifetime, incorporating an adequate margin of safety (EPA 1989, 40 Code of Federal Regulations 300.430(e)(2)(i)(A)). This acceptable exposure level is approximated best by an HI equal to or less than 1. If the HI is less than 1, then adverse effects usually would not be expected. However, adverse effects may occur when the HI exceeds 1. It is not possible to assign mathematical probabilities to the likelihood of the occurrence of adverse effects when the HI exceeds 1 in a deterministic risk assessment.

5.3.2 Potential Excess Lifetime Cancer Risks Associated with Exposure to Surface Soil

Potential excess lifetime cancer risks associated with exposure to surface soil are presented in Table 5-1. Risk estimates are segregated by location, receptor, and exposure pathway (i.e., ingestion, dermal contact, and vegetable ingestion). The primary COPC contributing most to the risk estimate also is included. The following text provides additional explanation of cancer risk estimates associated with potential exposure to surface soil.

5.3.2.1 Current Exposure Scenarios

Off-Facility Properties

The potential excess lifetime cancer risks associated with exposure to surface soil, in open area samples collected from nearby off-facility properties, ranged from 4E-06 (Location 38) to 2E-04

(Location 53). Note that the maximum excess lifetime cancer risk was associated with the sample collected southeast of the facility on property which is not currently a residence. The maximum risk at an actual current open area residence is 2E-05 (Residence 18). Figure 3-2 indicates which of the open area off-facility samples actually are residences. The excess lifetime cancer risk associated with the background open area sample is 7E-06. Figure 5-1 presents open area residential cancer risks and the associated background cancer risk for vegetable ingestion, incidental soil ingestion, inhalation of particulates, and dermal contact with soil.

The potential excess lifetime cancer risks associated with exposure to surface soil, collected from biased composite samples collected at nearby off-facility properties, ranged from 6E-06 (Residences 23, 24, 44A) to 4E-05 (Residence 26). The excess lifetime cancer risk associated with the background biased residential sample is 1E-05. Figure 5-2 presents biased sample residential cancer risks and the associated background cancer risk for vegetable ingestion, incidental soil ingestion, inhalation of particulates, and dermal contact with soil. (Biased samples were collected from locations expected to have elevated concentrations of potential contaminants.) Each biased sample was a composite sample composed of surface soil from a series of locations, including along walkways, underneath eaves or adjacent to downspouts, or along entryways. Portions of yards with nonnative materials were excluded from sampling.)

The COPCs contributing most to these risk estimates are B(a)P equivalents and dioxin TEQs. Part of the reason that the risks associated with these chemical groups are higher is that one-half the DLs were assumed to be present in all “non-detect” samples, of which there were many. These one-half DLs were included in the estimate of the EPC. This was done because there was no basis to assume that the true concentrations were zero. Therefore, the actual risks may be higher, but likely are lower than those presented in this assessment. Section 3.4.2 presents a more detailed discussion of uncertainties associated with EPCs.

As described in Section 3.1.1, dioxin and furan congeners were assessed for the surface soil to vegetable uptake pathway and subsequent ingestion by residents. The risks associated with vegetable ingestion are included in Table 5-1 and TARA Table 8-Vegetation. Note that in no case is vegetable ingestion the pathway responsible for the majority of the overall risk. In most cases it contributes about one-tenth of the overall cancer risk; however, at a few locations vegetable ingestion contributes as much as one-third the overall risk. Section 3.4.3 presents the uncertainties associated with assumptions associated with ingestion of home-grown produce.

On-Facility Worker

The potential excess lifetime cancer risks associated with exposure to on-facility surface soil ranged from 5E-04 to 1E-03 (Table 5-1). The 2,3,7,8-TCDD TEQ was the primary COPC responsible for the risk, and exposure through incidental ingestion contributed greater than 90% to these risk estimates. These estimated risks were associated with conservative assumptions about the magnitude of worker contact with and ingestion of surface soil; specifically, it was assumed that a worker would ingest 200 mg of soil through hand-to-mouth activities every working day throughout their working lifetime. Access to on-facility soil for the current on-site worker was assumed to be limited to areas that are not paved or covered with gravel. An analysis of potential risks associated with on-facility air is presented in Appendix C.

Off-Facility Recreational Visitor

The potential excess lifetime cancer risks associated with exposure to surface soil collected from the path adjacent to Little Squalicum Creek, the South Slope Area, and the spoils piles adjacent to Little Squalicum Creek ranged from 1E-06 to 4E-05 (Table 5-1). The risk estimate associated with the spoils piles is due primarily to TPH. Estimated concentrations of B(a)P equivalents also contributed to elevated risk, which is likely to be somewhat elevated by inclusion of one-half DLs for nondetected compounds. Also, the extent to which recreational users would be exposed solely to media within the spoils piles is uncertain.

5.3.2.2 Future Exposure Scenarios

The on-facility future scenario assumes that all gravel and asphalt caps will be removed and that all surface soil is available for contact. This assumption is unlikely in the foreseeable future because the facility is expected to remain in operation.

On-Facility Resident

The potential excess lifetime cancer risks associated with exposure to surface soil at the facility ranged from 2E-03 to 7E-03 (Table 5-1). The primary COPC responsible for the excess lifetime cancer risks was the 2,3,7,8-TCDD TEQ. These risk estimate assume that a resident lives on the facility year-round, contacts contaminated soil on a daily basis, and consumes vegetables grown in on-facility soil.

On-Facility Worker

The potential excess lifetime cancer risks associated with exposure to surface soil at the facility ranged from 6E-04 to 2E-03 (Table 5-1). The primary COPC responsible for the risk was the 2,3,7,8-TCDD TEQ. These estimated risks were associated with conservative assumptions about the magnitude of worker contact with and ingestion of surface soil; specifically, it was assumed that a worker would ingest 200 mg of soil through hand-to-mouth activities every working day throughout their working lifetime.

5.3.3 Potential Noncarcinogenic Effects Associated with Surface Soil

Potential noncancer HIs associated with exposure to surface soil are presented in Table 5-2. Risk estimates are segregated by location, receptor, and exposure pathway (i.e., ingestion, dermal contact, and inhalation). The primary COPC contributing most to the risk estimate also is included. Note that HIs for off-site residents are not included because no noncarcinogenic COPCs were identified in off-facility surface soil samples at concentrations exceeding risk-based screening levels. The following text provides additional explanation of surface soil noncancer hazard estimates.

5.3.3.1 Current Exposure Scenarios

On-Facility Worker

Total HIs for the on-facility worker associated with exposure to surface soil ranged from 5E-04 to 1E-03 (Table 5-2).

Off-Facility Recreational Visitor

The total HI associated with exposure to the spoils piles located adjacent to Little Squalicum Creek was 0.5 (Table 5-2). The HI is primarily due to TPH.

5.3.3.2 Future Exposure Scenarios

The on-facility future scenario assumes that all gravel and asphalt caps will be removed and that all surface soil is available for contact. This assumption is unlikely in the foreseeable future because the facility is expected to remain in operation.

On-Facility Resident

Total HIs associated with exposure to surface soil on the facility ranged from 0.007 to 0.08 (Table 5-2).

On-Facility Worker

Total HIs associated with exposure to surface soil on the facility ranged from 0.001 to 0.01 (Table 5-2).

5.3.4 Potential Excess Lifetime Cancer Risks Associated with Exposure to Subsurface Soil (0 to 6 Feet bgs)

Potential excess lifetime cancer risks associated with exposure to subsurface soil (0 to 6 feet bgs) are presented in Table 5-3. Risk estimates are segregated by location, receptor, and exposure pathway (i.e., ingestion, dermal contact, and inhalation). Note that under current exposure conditions, exposure to subsurface soil represents an incomplete exposure pathway. However, this scenario was included to evaluate the risks in the future if excavation resulted in exposure of soils currently 0 to 6 feet bgs. The primary COPC contributing most to the risk estimate also is included. The following text provides additional explanation of cancer risk estimates associated with potential future exposure to subsurface soil (0 to 6 feet bgs).

On-Facility Resident

The potential excess lifetime cancer risks associated with exposure to subsurface soil (0 to 6 feet bgs) at the facility ranged from 3E-05 to 5E-03 (Table 5-3). The primary COPCs responsible for the estimated risks were the 2,3,7,8-TCDD TEQ and B(a)P equivalents.

On-Facility Worker

The potential excess lifetime cancer risks associated with exposure to subsurface soil (0 to 6 feet bgs) at the facility ranged from 1E-05 to 2E-03 (Table 5-3). The estimated risks for the East and West Treatment areas and the North Treatment Area were the highest, and the estimated risks for the South Pole Yard were the lowest. The primary COPCs responsible for the risks were B(a)P equivalents and 2,3,7,8-TCDD TEQ.

Off-Facility Recreational Visitor

The potential excess lifetime cancer risks associated with exposure to subsurface soil (0 to 6 feet bgs) collected from the soil sample trench adjacent to Little Squalicum Creek and the South Slope Area were 1E-08 and 2E-08, respectively (Table 5-3).

5.3.5 Potential Noncarcinogenic Effects Associated with Subsurface Soil (0 to 6 feet bgs)

Potential noncancer HIs associated with exposure to subsurface soil (0 to 6 feet bgs) are presented in Table 5-4. Risk estimates are segregated by location, receptor, and exposure pathway (i.e., ingestion, dermal contact, and inhalation). Note that under current exposure conditions, exposure to subsurface soil represents an incomplete exposure pathway. However, this scenario was included to evaluate the risks in the future if on-site excavation resulted in exposure of soils currently 0 to 6 feet bgs. The primary COPC contributing most to the risk estimate also is included. The following text provides additional explanation of noncancer HIs associated with exposure to subsurface soil (0 to 6 feet bgs).

On-Facility Resident

Total HIs for the on-facility resident associated with exposure to subsurface soil (0 to 6 feet bgs) ranged from 0.006 to 70 (Table 5-4). The East and West Treatment areas and the North Treatment Area had HIs of 70 (primarily due to naphthalene) and 5 (primarily due to TPH), respectively. These hazard estimates assume that a resident lives on the facility year-round and contacts contaminated subsurface soil on a daily basis.

On-Facility Worker

Total HIs associated with exposure to subsurface soil (0 to 6 feet bgs) ranged from 1E-03 to 11 (Table 5-4). The East and West Treatment areas had an HI of 11 (primarily due to naphthalene).

Off-Facility Recreational Visitor

No noncarcinogenic COPCs were identified in off-facility subsurface soil.

5.3.6 Potential Excess Lifetime Cancer Risks Associated with Exposure to Subsurface Soil (6 to 12 Feet bgs)

Potential excess lifetime cancer risks associated with exposure to subsurface soil (6 to 12 feet bgs) are presented in Table 5-5. Risk estimates are segregated by location, receptor, and

exposure pathway (i.e., ingestion, dermal contact, and inhalation). Note that under current exposure conditions, exposure to subsurface soil represents an incomplete exposure pathway. However, this scenario was included to evaluate the risks in the future if excavation resulted in exposure of soils currently 6 to 12 feet bgs. The primary COPC contributing most to the risk estimate also is included. The following text provides additional explanation of cancer risk estimates associated with future exposure to subsurface soil (6 to 12 feet bgs).

On-Facility Resident

The potential excess lifetime cancer risks associated with exposure to subsurface soil (6 to 12 feet bgs) at the facility ranged from 2E-05 to 1E-03 (Table 5-5). The risks for the East and West Treatment areas, the North Treatment Area, and the South Pole Yard were the highest. The primary COPCs responsible for the risks were the 2,3,7,8-TCDD TEQ and B(a)P equivalents. These risk estimates assume that a resident lives on the facility year-round and contacts contaminated subsurface soil on a daily basis.

On-Facility Worker

The potential excess lifetime cancer risks associated with exposure to subsurface soil (6 to 12 feet bgs) at the facility ranged from 7E-06 to 7E-04 (Table 5-5). The risks for the East and West Treatment areas, North Treatment Area, and the South Pole Yard were the highest. The primary COPCs responsible for the risk were the B(a)P equivalents, TPH, and 2,3,7,8-TCDD TEQ.

Off-Facility Recreational Visitor

The potential excess lifetime cancer risk associated with exposure to subsurface soil (6 to 12 feet bgs) at the South Slope Area was 7E-08 (Table 5-5).

5.3.7 Potential Noncarcinogenic Effects Associated with Subsurface Soil (6 to 12 Feet bgs)

Potential noncancer HIs associated with exposure to subsurface soil (6 to 12 feet bgs) are presented in Table 5-6. Risk estimates are segregated by location, receptor, and exposure pathway (i.e., ingestion, dermal contact, and inhalation). Note that under current exposure conditions, exposure to subsurface soil represents an incomplete exposure pathway. However, this scenario was included to evaluate the risks in the future if on-site excavation resulted in exposure of soils currently 6 to 12 feet bgs. The primary COPC contributing most to the risk estimate also is included. The following text provides

additional explanation of noncancer HIs associated with potential future exposure to subsurface soil (6 to 12 feet bgs).

On-Facility Resident

Total HIs for a potential future on-facility resident associated with exposure to subsurface soil (6 to 12 feet bgs) ranged from 1 to 20 (Table 5-6). The East and West Treatment areas, the North Treatment Area, the South Pole Yard, and the Treated Pole Area had the greatest HIs. Naphthalene and TPH were the primary COPCs. These hazard estimates assume that a resident lives on the facility year-round and contacts contaminated subsurface soil on a daily basis.

On-Facility Worker

Total HIs for a potential future on-facility worker associated with exposure to subsurface soil (6 to 12 feet bgs) ranged from 0.2 to 3 (Table 5-6). The East and West Treatment areas, the North Treatment Area, and the South Pole Yard had the greatest HIs. Naphthalene and TPH were the primary COPCs.

Off-Facility Recreational Visitor

No noncarcinogenic COPCs were identified in off-facility subsurface soil.

5.3.8 Potential Excess Lifetime Cancer Risks Associated with Exposure to Subsurface Soil (12 to 18 Feet bgs)

Potential excess lifetime cancer risks associated with exposure to subsurface soil (12 to 18 feet bgs) are presented in Table 5-7. Risk estimates are segregated by location, receptor, and exposure pathway (i.e., ingestion, dermal contact, and inhalation). Note that under current exposure conditions, exposure to subsurface soil represents an incomplete exposure pathway. However, this scenario was included to evaluate the risks in the future if excavation resulted in exposure of soils currently 12 to 18 feet bgs. The primary COPC contributing most to the risk estimate also is included. The following text provides additional explanation of cancer risk estimates associated with potential future exposure to subsurface soil (12 to 18 feet bgs).

On-Facility Resident

The potential excess lifetime cancer risks associated with exposure to subsurface soil (12 to 18 feet bgs) at the facility ranged from 1E-06 to 3E-03 (Table 5-7). The risks for the East and West Treatment areas and the North Treatment Area were the highest. The primary COPCs responsible for the risks were B(a)P equivalents. These risk estimates assume that a resident lives on the facility year-round and contacts contaminated subsurface soil on a daily basis.

On-Facility Worker

The potential excess lifetime cancer risks associated with exposure to subsurface soil (12 to 18 feet bgs) at the facility ranged from 5E-07 to 1E-03 (Table 5-7). The risks for the East and West Treatment areas and the North Treatment Area were the greatest. The primary COPC responsible for the risks were B(a)P equivalents.

Off-Facility Recreational Visitor

The potential excess lifetime cancer risk associated with exposure to subsurface soil (12 to 18 feet bgs) at the South Slope Area was 4E-08 (Table 5-7).

5.3.9 Potential Noncarcinogenic Effects Associated with Subsurface Soil (12 to 18 feet bgs)

Potential noncancer HIs associated with exposure to subsurface soil (12 to 18 feet bgs) are presented in Table 5-8. Risk estimates are segregated by location, receptor, and exposure pathway (i.e., ingestion, dermal contact, and inhalation). Note that under current exposure conditions, exposure to subsurface soil represents an incomplete exposure pathway. However, this scenario was included to evaluate the risks in the future if on-site excavation resulted in exposure of soils currently 12 to 18 feet bgs. The primary COPC contributing most to the risk estimate also is included. The following text provides additional explanation of noncancer HIs associated with potential future exposure to subsurface soil (12 to 18 feet bgs).

On-Facility Resident

Total HIs for the on-facility resident associated with exposure to subsurface soil (12 to 18 feet bgs) ranged from 20 to 50 (Table 5-8). The East and West Treatment areas and the North Treatment Area had the highest HIs. Naphthalene and TPH were the primary COPCs. These hazard

estimates assume that a resident lives on the facility year-round and contacts contaminated subsurface soil on a daily basis.

On-Facility Worker

Total HIs for the on-facility worker associated with exposure to subsurface soil (12 to 18 feet bgs) ranged from 3 to 8 (Table 5-8). The East and West Treatment areas and the North Treatment Area had the highest HIs. Naphthalene was the primary COPC.

5.3.10 Potential Excess Lifetime Cancer Risks Associated with Exposure to Sediment

Potential excess lifetime cancer risks associated with exposure to sediment in Little Squalicum Creek are presented in Table 5-9. Risk estimates are segregated by location and are based on potential dermal contact only. Note that no noncarcinogenic compounds were detected at concentrations greater than screening levels; therefore, risks for sediment are presented only for carcinogens. The following text provides additional explanation of cancer risk estimates associated with exposure to sediment.

Off-Facility Recreational Visitor

The potential excess lifetime cancer risks associated with exposure to sediment in Little Squalicum Creek were 5E-07 downstream of Marine Drive and 8E-07 upstream of Marine Drive. The potential excess lifetime cancer risks associated with exposure to the background sediment samples were 2E-08 (in the sample collected from the wetland northeast of Little Squalicum Creek) and 1E-08 (in the sample collected from the Birchwood outfall located upgradient of The Oeser Company; Table 5-9).

5.3.11 Potential Excess Lifetime Cancer Risks Associated with Groundwater

Potential excess lifetime cancer risks associated with exposure to groundwater are presented in Table 5-10. Risk estimates are segregated by well, receptor, and exposure pathway (i.e., ingestion and dermal contact). Note that under current exposure conditions, exposure to groundwater on site represents an incomplete exposure pathway because the only wells on site currently are monitoring wells. Consequently, under normal exposure conditions, no risks to workers or residents associated with contact with groundwater are expected. However, this scenario was included to evaluate the risks in the future if deep groundwater is developed for domestic and/or industrial uses. Shallow groundwater is not plentiful enough to be a reliable source of drinking or industrial process water in the future. The primary COPC

contributing most to the risk estimate also is included. The following text provides additional explanation of cancer risk estimates associated with exposure to groundwater.

5.3.11.1 Current Exposure Scenarios

On-Facility Worker

The potential excess lifetime cancer risks associated with the groundwater wells at the Tilbury Cement Company (TC-5 and TC-6) ranged from 2E-04 to 4E-04 for the on-facility worker (Table 5-10). However, no carcinogenic COPCC was ever detected in these wells; the potential excess lifetime cancer risks for on-facility worker exposure to groundwater is based solely on the use of one-half DLs for nondetected compounds. Consequently, actual risks to on-facility workers are likely to be significantly less than the estimated risks.

5.3.11.2 Future Exposure Scenarios

On-Facility Resident

The potential excess lifetime cancer risks associated with the deep groundwater wells ranged from 8E-04 to 1E-03 for the potential future on-facility resident (Table 5-10). The risk calculated for the background well, MW06-D, was 8E-04. The primary COPCs are 2,3,7,8-TCDD TEQ and B(a)P equivalents.

On-Facility Worker

The potential excess lifetime cancer risks associated with the deep groundwater wells ranged from 6E-06 to 1E-05 for the on-facility worker (Table 5-10). The risk calculated for the background well, MW06-D, was 8E-06. These risk estimates assume that a resident ingests and contacts groundwater on a daily basis.

The COPCs that have contributed to risks for each well are 2,3,7,8-TCDD TEQ and B(a)P equivalents. PCP also is a COPC for several wells, including MW05-D and MW25-D. No other compounds were identified as COPCs. Only two cPAHs were detected in one deep well, MW05-D. No other cPAHs were detected in any of the deep wells; therefore, the calculation of B(a)P equivalents for these wells was based solely on the use of one-half DLs for nondetected compounds. In contrast, at least one dioxin congener was detected in every deep well, however, no concentrations of dioxin/furan congeners exceeded the respective screening toxicity values, and like the calculation of the B(a)P equivalents, the calculation of the 2,3,7,8-TCDD TEQ is based largely on the use of one-half DLs for

nondetected compounds. Given that the risk levels in the background well exceed the EPA acceptable risk levels, and that primary COPCs are calculated values based on one-half DLs, the risks associated with use of groundwater likely are overestimated.

5.3.12 Potential Noncarcinogenic Effects Associated with Groundwater

Potential noncancer HIs associated with exposure to groundwater are presented in Table 5-11. Risk estimates are segregated by well, receptor, and exposure pathway (i.e., ingestion and dermal contact). Note that under current exposure conditions, exposure to groundwater on site represents an incomplete exposure pathway. However, this scenario was included to evaluate the risks in the future if deep groundwater is developed for domestic/industrial uses. Shallow groundwater is not plentiful enough to be a reliable source of drinking or industrial process water in the future. The primary COPC contributing most to the risk estimate also is included. The following text provides additional explanation of HIs associated with exposure to groundwater.

5.3.12.1 Current Exposure Scenarios

There were no noncarcinogenic COPCs in the samples obtained from the Tilbury Cement Company wells.

5.3.12.2 Future Exposure Scenarios

HIs for each well ranged from 0.01 to 0.5 for the on-facility resident and 0.0001 to 0.0002 for the on-facility worker (Table 5-11). Evaluated COPCs include naphthalene, PCP, and EPHs. These hazard estimates assume that a resident or worker ingests and contacts groundwater on a daily basis.

5.3.13 Potential Excess Lifetime Cancer Risks Associated with Surface Water

Potential excess lifetime cancer risks associated with exposure to surface water are presented in Table 5-12. Risk estimates are presented for Little Squalicum Creek and a background location and are based on potential dermal contact only. The primary COPC contributing most to the risk estimate also is included. The following text provides additional explanation of cancer risk estimates associated with exposure to surface water.

5.3.13.1 Current and Future Exposure Scenarios

Off-Facility Recreational Visitor

The potential excess lifetime cancer risk for dermal exposure of the recreational visitor to the surface water in Little Squalicum Creek was 5E-04 (Table 5-12). The 2,3,7,8-TCDD TEQ contributed the highest percentage of the risk (approximately 80%). Multiple dioxin congeners were detected in surface water as COPCs, as well as B(a)P. The risks associated with the detected dioxin/furan congeners and B(a)P were 3E-04 and 1E-05, respectively. Therefore, in this case, DLs are not driving risk. The risk associated with the background surface water location, SW02, was 1E-04; however, this is based primarily on one-half DLs for nondetected compounds.

5.3.14 Potential Noncarcinogenic Effects Associated with Surface Water

The potential noncancer HI associated with exposure to Little Squalicum Creek surface water is presented in Table 5-13. No COPCs were identified in the background location; therefore, an HI is included only for the creek downgradient of the Oeser outfall. The HI is based on potential dermal contact only. The primary COPC contributing most to the HI also is included.

5.3.14.1 Current and Future Exposure Scenarios

Off-Facility Recreational Visitor

The HI for dermal exposure of the recreational visitor to the surface water in Little Squalicum Creek was 0.05, based on exposure to the only noncarcinogenic COPC, PCP (Table 5-13). COPCs were not identified in the background sample; therefore, an HI was not calculated.

5.3.15 Potential Excess Lifetime Cancer Risks Associated with Air

Potential excess lifetime cancer risks associated with exposure to air are presented in Table 5-14. Risk estimates are presented for each sampling station with chemicals detected at concentrations greater than screening levels. The primary COPC contributing most to the risk estimate also is included. Note that risks were calculated only for current receptors; risks to future receptors may be similar, but air concentrations in the future are difficult to predict. Furthermore, air concentrations do not necessarily represent a long-term exposure level because air concentrations are more likely to change over time than concentrations in other media. Therefore, future risks may be higher or lower than those estimated here. The following text provides additional explanation of cancer risk estimates associated with inhalation exposure to air.

Off-Facility Resident

Potential excess lifetime cancer risks were calculated individually for each air sampling station. Risks ranged from 3E-06 to 3E-5 for the off-facility resident (Table 5-14). Only one sampling station, AS29, exceeded MTCA's ceiling. AS29 was located at The Oeser Company's northeast fence line. The risk of 1E-05 at the background air sampling location, AS30, was equal to or higher than the risk at all sampling locations other than AS29. This suggests that other urban sources may be contributing to the risk. The primary COPCs included dioxins/furans, benzene, and PCP. While detections of dioxins/furans and benzene are likely due to other urban sources, the PCP detected at AS29 is likely due to site-related activities.

Off-Facility Recreational Visitor

Potential excess lifetime cancer risks for the off-facility recreational visitor ranged from 8E-08 to 1E-06 (Table 5-14). No risks exceeded the EPA's acceptable range of 1E-06 to 1E-04 or the MTCA ceiling of 1E-05. The background air sampling location had a risk of 4E-07, which was higher than the risk at all other sampling locations besides AS29.

5.3.16 Potential Noncarcinogenic Effects Associated with Air

Potential noncancer HIs associated with exposure to air are presented in Table 5-15. Risk estimates are presented for each sampling station with chemicals detected at concentrations greater than screening levels. The primary COPC contributing most to the risk estimate also is included. Note that HIs were calculated only for current receptors; noncancer hazards to future receptors may be similar, but air concentrations in the future are difficult to predict. Furthermore, air concentrations do not necessarily represent a long-term exposure level because air concentrations are more likely to change over time than concentrations in other media. Future noncancer HIs may be higher or lower than those presented here. The following text provides additional explanation of HIs associated with inhalation exposure to air.

Off-Facility Resident

HIs were calculated individually for each air sampling station. Total HIs ranged from 0.06 to 5 for the on-facility resident (Table 5-15). Air sampling stations AS25 and AS29 had total HIs of 3 and 5, respectively. The HIs at these two stations exceeded the EPA's and MTCA's acceptable level for noncarcinogens. Both of these sampling stations were located near The Oeser Company's northeast fence line. The background air sampling station, AS30, had a total HI of 2. COPCs included

1,2,4-trimethylbenzene; 1,3,5-trimethylbenzene; benzene; 2-methylnaphthalene; n-propylbenzene; naphthalene; PCP; sec-butylbenzene; and dibenzofuran.

Off-Facility Recreational Visitor

Total HIs for the off-facility recreational visitor ranged from 0.003 to 0.2 (Table 5-15). These HIs are below EPA and MTCA acceptable levels.

5.4 RISK CHARACTERIZATION UNCERTAINTIES

The risk characterization combines and integrates the information developed in the COPC selection process, as well as in the exposure and toxicity assessments. Therefore, uncertainties associated with these aspects of this baseline HHRA also may affect the degree of confidence that can be placed in risk characterization results. Sections 2.2, 3.4, and 4.2 provide full discussions of uncertainties associated with COPC selection, exposure assessment, and toxicity assessment, respectively.

5.4.1 Assumption of Future Residential Land Use

The most conservative exposure scenarios evaluated in this baseline HHRA involved residential exposure assumptions. This assumption is plausible considering current residential locations; however, future residential development of The Oeser Company is not expected. Therefore, potential future residential risks on facility may not be representative of actual future risks.

5.4.2 Exposure Assessment Uncertainties

There are specific exposure assessment uncertainties associated with estimating risks and hazards due to dermal contact with surface water. Dermal absorption depends on numerous factors including the area of exposed skin, anatomical location of exposed skin, length of contact, concentration of chemical on skin, chemical-specific permeability, medium in which the chemical is applied, and skin condition and integrity. The permeability of the skin to a chemical in water is influenced by the physicochemical properties of the substance, including its molecular weight (size and shape), electrostatic charge, lipophilicity, and solubility in water.

The dermal permeability coefficients of highly lipophilic molecules in water, such as those found in the samples from Little Squalicum Creek, are outside of the effective predictive domain of these factors, which renders the estimates of risk highly uncertain. For example, the difference between the

lower and upper confidence limits on the estimate of the mean dermal permeability coefficients for TCDD, PCP and B(a)P spans approximately three orders of magnitude (EPA 2000b). For each chemical, an average predicted permeability coefficient which lies between the two confidence limits was used to estimate dermal dose for this risk assessment. However, until experimental studies are conducted which better define the dermal permeability coefficients, the estimated hazards and risks associated with skin contact in water with highly lipophilic substances should not be considered to be reliably quantitative, and are likely to be significantly overestimated (Hoang 2002).

In addition, calculation of the EPC for surface water is uncertain, because it is unlikely that the concentrations of COPCs in the creek are constant over time.

5.4.3 Perspectives on Risk Characterization Uncertainties

Because numerous conservative assumptions were used in the selection of COPCs and the exposure and toxicity assessments, the risk characterization results likely overestimate risks associated with COPCs at The Oeser Company. One of the major items that likely overestimates risk at The Oeser Company is the use of one-half the DLs for nondetected dioxins/furans and cPAHs. For example, the potential excess lifetime cancer risks for on-facility residential exposure to groundwater exceeded EPA criteria based solely on the use of one-half DLs for nondetected compounds.

However, use of one half the DL likely underestimates the variability in actual sample results. This can affect the derivation of the EPC used for estimating cancer risks. Using of one half the DL is assumed to be conservative.

Table 5-1

**Excess Lifetime Cancer Risks Associated with Surface Soil
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Current	Worker	Dermal	NPY/SPY	1.74E-04	Dioxin TEQ	1.72E-04
Current	Worker	Ingestion	NPY/SPY	2.87E-04	Dioxin TEQ	2.86E-04
Current	Worker	Inhalation	NPY/SPY	2.72E-08	Dioxin TEQ	2.72E-08
	Total			5.E-04		
Current	Worker	Dermal	NTA	1.26E-04	Dioxin TEQ	1.25E-04
Current	Worker	Ingestion	NTA	8.35E-04	Dioxin TEQ	8.31E-04
Current	Worker	Inhalation	NTA	1.98E-08	Dioxin TEQ	1.98E-08
	Total			1.E-03		
Current	Worker	Dermal	WSA	8.24E-05	Dioxin TEQ	6.47E-05
Current	Worker	Ingestion	WSA	4.86E-04	Dioxin TEQ	4.31E-04
Current	Worker	Inhalation	WSA	1.12E-08	Dioxin TEQ	1.03E-08
	Total			6.E-04		
Current/Future	Resident	Dermal	OPEN BKGD	1.51E-06	B(a)P Equivalent	1.42E-06
Current/Future	Resident	Ingestion	OPEN BKGD	4.88E-06	B(a)P Equivalent	4.24E-06
Current/Future	Resident	Veg. Ingestion	OPEN BKGD	2.92E-07	Dioxin TEQ	2.92E-07
	Total			7.E-06		
Current/Future	Resident	Dermal	OPEN-RES-02	1.91E-06	B(a)P Equivalent	1.91E-06
Current/Future	Resident	Ingestion	OPEN-RES-02	5.70E-06	B(a)P Equivalent	5.70E-06
	Total			8.E-06		
Current/Future	Resident	Dermal	OPEN-RES-05	2.85E-06	B(a)P Equivalent	2.23E-06
Current/Future	Resident	Ingestion	OPEN-RES-05	1.11E-05	B(a)P Equivalent	6.68E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-05	2.05E-06	Dioxin TEQ	2.05E-06
	Total			2.E-05		
Current/Future	Resident	Dermal	OPEN-RES-07	2.39E-06	B(a)P Equivalent	2.10E-06
Current/Future	Resident	Ingestion	OPEN-RES-07	8.40E-06	B(a)P Equivalent	6.26E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-07	9.83E-07	Dioxin TEQ	9.83E-07
	Total			1.E-05		
Current/Future	Resident	Dermal	OPEN-RES-08	2.43E-06	B(a)P Equivalent	1.96E-06
Current/Future	Resident	Ingestion	OPEN-RES-08	9.27E-06	B(a)P Equivalent	5.86E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-08	1.56E-06	Dioxin TEQ	1.56E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	OPEN-RES-11	3.02E-06	B(a)P Equivalent	1.97E-06
Current/Future	Resident	Ingestion	OPEN-RES-11	1.35E-05	Dioxin TEQ	7.63E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-11	3.51E-06	Dioxin TEQ	3.51E-06
	Total			2.E-05		
Current/Future	Resident	Dermal	OPEN-RES-12	2.35E-06	B(a)P Equivalent	1.94E-06
Current/Future	Resident	Ingestion	OPEN-RES-12	8.82E-06	B(a)P Equivalent	5.80E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-12	1.39E-06	Dioxin TEQ	1.39E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	OPEN-RES-16	1.78E-06	B(a)P Equivalent	1.61E-06
Current/Future	Resident	Ingestion	OPEN-RES-16	6.02E-06	B(a)P Equivalent	4.81E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-16	5.54E-07	Dioxin TEQ	5.54E-07
	Total			8.E-06		

Table 5-1

**Excess Lifetime Cancer Risks Associated with Surface Soil
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Current/Future	Resident	Dermal	OPEN-RES-18	2.38E-06	B(a)P Equivalent	1.57E-06
Current/Future	Resident	Ingestion	OPEN-RES-18	1.06E-05	Dioxin TEQ	5.89E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-18	2.71E-06	Dioxin TEQ	2.71E-06
	Total			2.E-05		
Current/Future	Resident	Dermal	OPEN-RES-28	1.26E-06	B(a)P Equivalent	1.26E-06
Current/Future	Resident	Ingestion	OPEN-RES-28	3.76E-06	B(a)P Equivalent	3.76E-06
	Total			5.E-06		
Current/Future	Resident	Dermal	OPEN-RES-32A	1.53E-06	B(a)P Equivalent	1.53E-06
Current/Future	Resident	Ingestion	OPEN-RES-32A	4.57E-06	B(a)P Equivalent	4.57E-06
	Total			6.E-06		
Current/Future	Resident	Dermal	OPEN-RES-36	1.42E-06	B(a)P Equivalent	1.42E-06
Current/Future	Resident	Ingestion	OPEN-RES-36	4.25E-06	B(a)P Equivalent	4.25E-06
	Total			6.E-06		
Current/Future	Resident	Dermal	OPEN-RES-38	1.09E-06	B(a)P Equivalent	1.09E-06
Current/Future	Resident	Ingestion	OPEN-RES-38	3.25E-06	B(a)P Equivalent	3.25E-06
	Total			4.E-06		
Current/Future	Resident	Dermal	OPEN-RES-41	2.28E-06	B(a)P Equivalent	1.85E-06
Current/Future	Resident	Ingestion	OPEN-RES-41	8.70E-06	B(a)P Equivalent	5.52E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-41	1.46E-06	Dioxin TEQ	1.46E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	OPEN-RES-42	2.04E-06	B(a)P Equivalent	1.69E-06
Current/Future	Resident	Ingestion	OPEN-RES-42	7.60E-06	B(a)P Equivalent	5.07E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-42	1.16E-06	Dioxin TEQ	1.16E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	OPEN-RES-42A	7.96E-06	Dioxin TEQ	6.78E-06
Current/Future	Resident	Ingestion	OPEN-RES-42A	5.29E-05	Dioxin TEQ	4.93E-05
Current/Future	Resident	Veg. Ingestion	OPEN-RES-42A	2.27E-05	Dioxin TEQ	2.27E-05
	Total			8.E-05		
Current/Future	Resident	Dermal	OPEN-RES-43	3.43E-06	Dioxin TEQ	2.26E-06
Current/Future	Resident	Ingestion	OPEN-RES-43	1.99E-05	Dioxin TEQ	1.64E-05
Current/Future	Resident	Veg. Ingestion	OPEN-RES-43	7.55E-06	Dioxin TEQ	7.55E-06
	Total			3.E-05		
Current/Future	Resident	Dermal	OPEN-RES-46	1.76E-06	B(a)P Equivalent	1.61E-06
Current/Future	Resident	Ingestion	OPEN-RES-46	5.94E-06	B(a)P Equivalent	4.80E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-46	5.21E-07	Dioxin TEQ	5.21E-07
	Total			8.E-06		
Current/Future	Resident	Dermal	OPEN-RES-47A	1.27E-06	B(a)P Equivalent	1.02E-06
Current/Future	Resident	Ingestion	OPEN-RES-47A	3.80E-06	B(a)P Equivalent	3.05E-06
	Total			5.E-06		
Current/Future	Resident	Dermal	OPEN-RES-48	1.54E-06	B(a)P Equivalent	1.32E-06
Current/Future	Resident	Ingestion	OPEN-RES-48	5.56E-06	B(a)P Equivalent	3.93E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-48	7.48E-07	Dioxin TEQ	7.48E-07
	Total			8.E-06		

Table 5-1

**Excess Lifetime Cancer Risks Associated with Surface Soil
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Current/Future	Resident	Dermal	OPEN-RES-49	1.39E-06	B(a)P Equivalent	1.19E-06
Current/Future	Resident	Ingestion	OPEN-RES-49	5.00E-06	B(a)P Equivalent	3.57E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-49	6.54E-07	Dioxin TEQ	6.54E-07
	Total			7.E-06		
Current/Future	Resident	Dermal	OPEN-RES-50	8.17E-06	Dioxin TEQ	4.72E-06
Current/Future	Resident	Ingestion	OPEN-RES-50	4.47E-05	Dioxin TEQ	3.43E-05
Current/Future	Resident	Veg. Ingestion	OPEN-RES-50	1.58E-05	Dioxin TEQ	1.58E-05
	Total			7.E-05		
Current/Future	Resident	Dermal	OPEN-RES-51	1.77E-06	B(a)P Equivalent	1.77E-06
Current/Future	Resident	Ingestion	OPEN-RES-51	5.28E-06	B(a)P Equivalent	5.28E-06
	Total			7.E-06		
Current/Future	Resident	Dermal	OPEN-RES-52	2.00E-06	B(a)P Equivalent	1.51E-06
Current/Future	Resident	Ingestion	OPEN-RES-52	8.12E-06	B(a)P Equivalent	4.51E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-52	1.66E-06	Dioxin TEQ	1.66E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	OPEN-RES-53	1.74E-05	Dioxin TEQ	1.40E-05
Current/Future	Resident	Ingestion	OPEN-RES-53	1.12E-04	Dioxin TEQ	1.02E-04
Current/Future	Resident	Veg. Ingestion	OPEN-RES-53	4.69E-05	Dioxin TEQ	4.69E-05
	Total			2.E-04		
Current/Future	Resident	Dermal	OPEN-RES-54	7.32E-06	Dioxin TEQ	4.78E-06
Current/Future	Resident	Ingestion	OPEN-RES-54	4.24E-05	Dioxin TEQ	3.48E-05
Current/Future	Resident	Veg. Ingestion	OPEN-RES-54	1.60E-05	Dioxin TEQ	1.60E-05
	Total			7.E-05		
Current/Future	Resident	Dermal	OPEN-RES-55	1.86E-06	B(a)P Equivalent	1.86E-06
Current/Future	Resident	Ingestion	OPEN-RES-55	5.57E-06	B(a)P Equivalent	5.57E-06
	Total			7.E-06		
Current/Future	Resident	Dermal	OPEN-RES-56	2.04E-06	B(a)P Equivalent	1.84E-06
Current/Future	Resident	Ingestion	OPEN-RES-56	6.96E-06	B(a)P Equivalent	5.51E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-56	6.65E-07	Dioxin TEQ	6.65E-07
	Total			1.E-05		
Current/Future	Resident	Dermal	OPEN-RES-57	1.81E-06	B(a)P Equivalent	1.43E-06
Current/Future	Resident	Ingestion	OPEN-RES-57	6.99E-06	B(a)P Equivalent	4.28E-06
Current/Future	Resident	Veg. Ingestion	OPEN-RES-57	1.24E-06	Dioxin TEQ	1.24E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	RES BKGD	2.75E-06	B(a)P Equivalent	2.00E-06
Current/Future	Resident	Ingestion	RES BKGD	1.14E-05	B(a)P Equivalent	5.97E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	RES-01	2.15E-06	B(a)P Equivalent	1.83E-06
Current/Future	Resident	Ingestion	RES-01	7.77E-06	B(a)P Equivalent	5.47E-06
Current/Future	Resident	Veg. Ingestion	RES-01	1.06E-06	Dioxin TEQ	1.06E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	RES-03	2.29E-06	B(a)P Equivalent	1.45E-06
Current/Future	Resident	Ingestion	RES-03	1.04E-05	Dioxin TEQ	6.06E-06
Current/Future	Resident	Veg. Ingestion	RES-03	2.78E-06	Dioxin TEQ	2.78E-06
	Total			2.E-05		

Table 5-1

**Excess Lifetime Cancer Risks Associated with Surface Soil
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Current/Future	Resident	Dermal	RES-04	1.58E-06	B(a)P Equivalent	1.26E-06
Current/Future	Resident	Ingestion	RES-04	6.10E-06	B(a)P Equivalent	3.76E-06
Current/Future	Resident	Veg. Ingestion	RES-04	1.08E-06	Dioxin TEQ	2.78E-06
	Total			9.E-06		
Current/Future	Resident	Dermal	RES-06	3.16E-06	B(a)P Equivalent	2.15E-06
Current/Future	Resident	Ingestion	RES-06	1.38E-05	Dioxin TEQ	7.36E-06
Current/Future	Resident	Veg. Ingestion	RES-06	3.38E-06	Dioxin TEQ	3.38E-06
	Total			2.E-05		
Current/Future	Resident	Dermal	RES-09	2.06E-06	B(a)P Equivalent	1.86E-06
Current/Future	Resident	Ingestion	RES-09	7.00E-06	B(a)P Equivalent	5.56E-06
Current/Future	Resident	Veg. Ingestion	RES-09	6.62E-07	Dioxin TEQ	6.62E-07
	Total			1.E-05		
Current/Future	Resident	Dermal	RES-10	1.78E-06	B(a)P Equivalent	1.64E-06
Current/Future	Resident	Ingestion	RES-10	5.90E-06	B(a)P Equivalent	4.90E-06
Current/Future	Resident	Veg. Ingestion	RES-10	4.59E-07	Dioxin TEQ	4.59E-07
	Total			8.E-06		
Current/Future	Resident	Dermal	RES-13	1.67E-06	B(a)P Equivalent	1.29E-06
Current/Future	Resident	Ingestion	RES-13	6.59E-06	B(a)P Equivalent	3.87E-06
Current/Future	Resident	Veg. Ingestion	RES-13	1.25E-06	Dioxin TEQ	1.25E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	RES-14	2.21E-06	B(a)P Equivalent	1.77E-06
Current/Future	Resident	Ingestion	RES-14	8.51E-06	B(a)P Equivalent	5.30E-06
Current/Future	Resident	Veg. Ingestion	RES-14	1.47E-06	Dioxin TEQ	1.47E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	RES-15	1.78E-06	B(a)P Equivalent	1.57E-06
Current/Future	Resident	Ingestion	RES-15	6.26E-06	B(a)P Equivalent	4.69E-06
Current/Future	Resident	Veg. Ingestion	RES-15	7.23E-07	Dioxin TEQ	7.23E-07
	Total			9.E-06		
Current/Future	Resident	Dermal	RES-17	2.43E-06	B(a)P Equivalent	1.68E-06
Current/Future	Resident	Ingestion	RES-17	1.05E-05	Dioxin TEQ	5.46E-06
Current/Future	Resident	Veg. Ingestion	RES-17	2.51E-06	Dioxin TEQ	2.51E-06
	Total			2.E-05		
Current/Future	Resident	Dermal	RES-19	1.96E-06	B(a)P Equivalent	1.59E-06
Current/Future	Resident	Ingestion	RES-19	7.45E-06	B(a)P Equivalent	4.74E-06
Current/Future	Resident	Veg. Ingestion	RES-19	1.24E-06	Dioxin TEQ	1.24E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	RES-20	1.67E-06	B(a)P Equivalent	1.27E-06
Current/Future	Resident	Ingestion	RES-20	6.68E-06	B(a)P Equivalent	3.79E-06
Current/Future	Resident	Veg. Ingestion	RES-20	1.32E-06	Dioxin TEQ	1.32E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	RES-21A	1.58E-06	B(a)P Equivalent	1.24E-06
Current/Future	Resident	Ingestion	RES-21A	6.21E-06	B(a)P Equivalent	3.71E-06
Current/Future	Resident	Veg. Ingestion	RES-21A	1.15E-06	Dioxin TEQ	1.15E-06
	Total			9.E-06		

Table 5-1

**Excess Lifetime Cancer Risks Associated with Surface Soil
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Current/Future	Resident	Dermal	RES-22	3.53E-06	Dioxin TEQ	1.78E-06
Current/Future	Resident	Ingestion	RES-22	1.82E-05	Dioxin TEQ	1.30E-05
Current/Future	Resident	Veg. Ingestion	RES-22	5.95E-06	Dioxin TEQ	5.95E-06
	Total			3.E-05		
Current/Future	Resident	Dermal	RES-23	1.44E-06	B(a)P Equivalent	1.44E-06
Current/Future	Resident	Ingestion	RES-23	4.31E-06	B(a)P Equivalent	4.31E-06
	Total			6.E-06		
Current/Future	Resident	Dermal	RES-24	1.54E-06	B(a)P Equivalent	1.54E-06
Current/Future	Resident	Ingestion	RES-24	4.61E-06	B(a)P Equivalent	4.61E-06
	Total			6.E-06		
Current/Future	Resident	Dermal	RES-25	1.58E-06	B(a)P Equivalent	1.39E-06
Current/Future	Resident	Ingestion	RES-25	5.56E-06	B(a)P Equivalent	4.17E-06
Current/Future	Resident	Veg. Ingestion	RES-25	6.37E-07	Dioxin TEQ	6.37E-07
	Total			8.E-06		
Current/Future	Resident	Dermal	RES-26	8.83E-06	B(a)P Equivalent	8.45E-06
Current/Future	Resident	Ingestion	RES-26	2.80E-05	B(a)P Equivalent	2.53E-05
Current/Future	Resident	Veg. Ingestion	RES-26	1.25E-06	Dioxin TEQ	1.25E-06
	Total			4.E-05		
Current/Future	Resident	Dermal	RES-29	1.54E-06	B(a)P Equivalent	1.39E-06
Current/Future	Resident	Ingestion	RES-29	5.24E-06	B(a)P Equivalent	4.16E-06
Current/Future	Resident	Veg. Ingestion	RES-29	4.96E-07	Dioxin TEQ	4.96E-07
	Total			7.E-06		
Current/Future	Resident	Dermal	RES-30	1.68E-06	B(a)P Equivalent	1.50E-06
Current/Future	Resident	Ingestion	RES-30	5.80E-06	B(a)P Equivalent	4.47E-06
Current/Future	Resident	Veg. Ingestion	RES-30	6.08E-07	Dioxin TEQ	6.08E-07
	Total			8.E-06		
Current/Future	Resident	Dermal	RES-31	1.74E-06	B(a)P Equivalent	1.54E-06
Current/Future	Resident	Ingestion	RES-31	6.11E-06	B(a)P Equivalent	4.59E-06
Current/Future	Resident	Veg. Ingestion	RES-31	6.97E-07	Dioxin TEQ	6.97E-07
	Total			9.E-06		
Current/Future	Resident	Dermal	RES-33	1.75E-06	B(a)P Equivalent	1.56E-06
Current/Future	Resident	Ingestion	RES-33	6.06E-06	B(a)P Equivalent	4.66E-06
Current/Future	Resident	Veg. Ingestion	RES-33	6.40E-07	Dioxin TEQ	6.40E-07
	Total			8.E-06		
Current/Future	Resident	Dermal	RES-34	7.20E-06	B(a)P Equivalent	7.20E-06
Current/Future	Resident	Ingestion	RES-34	2.15E-05	B(a)P Equivalent	2.15E-05
	Total			3.E-05		
Current/Future	Resident	Dermal	RES-35	1.90E-06	B(a)P Equivalent	1.53E-06
Current/Future	Resident	Ingestion	RES-35	7.26E-06	B(a)P Equivalent	4.56E-06
Current/Future	Resident	Veg. Ingestion	RES-35	1.24E-06	Dioxin TEQ	1.24E-06
	Total			1.E-05		
Current/Future	Resident	Dermal	RES-37	1.74E-06	B(a)P Equivalent	1.42E-06
Current/Future	Resident	Ingestion	RES-37	6.60E-06	B(a)P Equivalent	4.23E-06
Current/Future	Resident	Veg. Ingestion	RES-37	1.09E-06	Dioxin TEQ	1.09E-06
	Total			9.E-06		

Table 5-1

**Excess Lifetime Cancer Risks Associated with Surface Soil
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Current/Future	Resident	Dermal	RES-44A	1.48E-06	B(a)P Equivalent	1.48E-06
Current/Future	Resident	Ingestion	RES-44A	4.42E-06	B(a)P Equivalent	4.42E-06
	Total			6.E-06		
Current/Future	Resident	Dermal	RES-58	1.49E-06	B(a)P Equivalent	1.49E-06
Current/Future	Resident	Ingestion	RES-58	4.45E-06	B(a)P Equivalent	4.45E-06
	Total			6.E-06		
Current/Future	Recreational Visitor	Dermal	PATH	1.25E-07	B(a)P Equivalent	6.68E-08
Current/Future	Recreational Visitor	Ingestion	PATH	5.73E-07	Dioxin TEQ	3.90E-07
Current/Future	Recreational Visitor	Inhalation	PATH	4.46E-11	Dioxin TEQ	3.72E-11
	Total			1.E-06		
Current/Future	Recreational Visitor	Dermal	SLOPE	9.53E-08	Dioxin TEQ	6.60E-08
Current/Future	Recreational Visitor	Ingestion	SLOPE	5.20E-07	Dioxin TEQ	4.40E-07
Current/Future	Recreational Visitor	Inhalation	SLOPE	4.52E-11	Dioxin TEQ	4.19E-11
	Total			1.E-06		
Current/Future	Recreational Visitor	Dermal	SPOILS	9.16E-06	B(a)P Equivalent	8.28E-06
Current/Future	Recreational Visitor	Ingestion	SPOILS	2.86E-05	B(a)P Equivalent	2.27E-05
Current/Future	Recreational Visitor	Inhalation	SPOILS	1.48E-09	B(a)P Equivalent	9.17E-10
	Total			4.E-05		
Future	Resident	Dermal	NPY	6.32E-04	Dioxin TEQ	6.16E-04
Future	Resident	Ingestion	NPY	4.50E-03	Dioxin TEQ	4.48E-03
Future	Resident	Inhalation	NPY	2.04E-07	Dioxin TEQ	2.03E-07
Future	Resident	Veg. Ingestion	NPY	2.06E-03	Dioxin TEQ	2.06E-03
	Total			7.E-03		
Future	Resident	Dermal	NTA	5.75E-04	Dioxin TEQ	5.48E-04
Future	Resident	Ingestion	NTA	4.05E-03	Dioxin TEQ	3.99E-03
Future	Resident	Inhalation	NTA	1.82E-07	Dioxin TEQ	1.81E-07
Future	Resident	Veg. Ingestion	NTA	1.83E-03	Dioxin TEQ	1.83E-03
	Total			6.E-03		
Future	Resident	Dermal	SPY	3.86E-04	Dioxin TEQ	2.70E-04
Future	Resident	Ingestion	SPY	2.31E-03	Dioxin TEQ	1.97E-03
Future	Resident	Inhalation	SPY	9.56E-08	Dioxin TEQ	8.89E-08
Future	Resident	Veg. Ingestion	SPY	9.03E-04	Dioxin TEQ	9.03E-04
	Total			4.E-03		
Future	Resident	Dermal	TPA	2.77E-04	Dioxin TEQ	2.23E-04
Future	Resident	Ingestion	TPA	1.78E-03	Dioxin TEQ	1.62E-03
Future	Resident	Inhalation	TPA	7.65E-08	Dioxin TEQ	7.34E-08
Future	Resident	Veg. Ingestion	TPA	7.45E-04	Dioxin TEQ	7.45E-04
	Total			3.E-03		
Future	Resident	Dermal	WSA	1.69E-04	Dioxin TEQ	1.33E-04
Future	Resident	Ingestion	WSA	1.09E-03	Dioxin TEQ	9.66E-04
Future	Resident	Inhalation	WSA	4.76E-08	Dioxin TEQ	4.37E-08
Future	Resident	Veg. Ingestion	WSA	4.43E-04	Dioxin TEQ	4.43E-04
	Total			2.E-03		
Future	Worker	Dermal	NPY	3.08E-04	Dioxin TEQ	3.00E-04
Future	Worker	Ingestion	NPY	2.01E-03	Dioxin TEQ	2.00E-03
Future	Worker	Inhalation	NPY	4.79E-08	Dioxin TEQ	4.76E-08
	Total			2.E-03		

Table 5-1

**Excess Lifetime Cancer Risks Associated with Surface Soil
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Future	Worker	Dermal	NTA	2.80E-04	Dioxin TEQ	2.67E-04
Future	Worker	Ingestion	NTA	1.81E-03	Dioxin TEQ	1.78E-03
Future	Worker	Inhalation	NTA	4.28E-08	Dioxin TEQ	4.24E-08
	Total			2.E-03		
Future	Worker	Dermal	SPY	1.88E-04	Dioxin TEQ	1.32E-04
Future	Worker	Ingestion	SPY	1.03E-03	Dioxin TEQ	8.78E-04
Future	Worker	Inhalation	SPY	2.25E-08	Dioxin TEQ	2.09E-08
	Total			1.E-03		
Future	Worker	Dermal	TPA	1.35E-04	Dioxin TEQ	1.09E-04
Future	Worker	Ingestion	TPA	7.93E-04	Dioxin TEQ	7.25E-04
Future	Worker	Inhalation	TPA	1.80E-08	Dioxin TEQ	1.73E-08
	Total			9.E-04		
Future	Worker	Dermal	WSA	8.24E-05	Dioxin TEQ	6.47E-05
Future	Worker	Ingestion	WSA	4.86E-04	Dioxin TEQ	4.31E-04
Future	Worker	Inhalation	WSA	1.12E-08	Dioxin TEQ	1.03E-08
	Total			6.E-04		

Key:

- COPC = Contaminant of potential concern.
- NPY = North Pole Yard.
- NTA = North Treatment Area.
- OPEN BKGD = Open area background sample location.
- OPEN-RES = Open area composite sample location.
- PATH = Footpath near Little Squalicum Creek.
- RES = Residential composite sample location.
- RES BKGD = Residential background sample location.
- SLOPE = South Slope.
- SPOILS = Soil spoils piles near Little Squalicum Creek.
- SPY = South Pole Yard.
- TEQ = Toxic equivalency quotient.
- TPA = Treated Pole Area.
- Veg. = Vegetation.
- WSA = Wood Storage Area.

Table 5-2

**Hazard Indices Associated with Surface Soil
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Hazard Index	Primary COPC	Primary COPC HQ
Current	Worker	Dermal	NPY/SPY	2.47E-04	Pentachlorophenol	2.47E-04
Current	Worker	Ingestion	NPY/SPY	3.00E-04	Pentachlorophenol	3.00E-04
Current	Worker	Inhalation	NPY/SPY	7.14E-09	Pentachlorophenol	7.14E-09
	Total			5.E-04		
Current	Worker	Dermal	WSA	4.47E-04	Pentachlorophenol	4.22E-04
Current	Worker	Ingestion	WSA	6.12E-04	Pentachlorophenol	5.13E-04
Current	Worker	Inhalation	WSA	1.46E-08	Pentachlorophenol	1.22E-08
	Total			1.E-03		
Current/Future	Recreational	Dermal	SPOILS	2.92E-05	2-Methylnaphthalene	2.92E-05
Current/Future	Recreational	Ingestion	SPOILS	4.66E-01	TPH Mixture	4.66E-01
Current/Future	Recreational	Inhalation	SPOILS	8.70E-03	2-Methylnaphthalene	8.70E-03
	Total			5.E-01		
Future	Resident	Dermal	NPY	3.39E-02	Pentachlorophenol	3.39E-02
Future	Resident	Ingestion	NPY	4.69E-02	Pentachlorophenol	4.69E-02
Future	Resident	Inhalation	NPY	1.12E-06	Pentachlorophenol	1.12E-06
	Total			8.E-02		
Future	Resident	Dermal	NTA	2.22E-02	Pentachlorophenol	2.22E-02
Future	Resident	Ingestion	NTA	3.07E-02	Pentachlorophenol	3.07E-02
Future	Resident	Inhalation	NTA	7.31E-07	Pentachlorophenol	7.31E-07
	Total			5.E-02		
Future	Resident	Dermal	SPY	5.55E-03	Pentachlorophenol	5.55E-03
Future	Resident	Ingestion	SPY	7.67E-03	Pentachlorophenol	7.67E-03
Future	Resident	Inhalation	SPY	1.83E-07	Pentachlorophenol	1.83E-07
	Total			1.E-02		
Future	Resident	Dermal	TPA	1.05E-02	Pentachlorophenol	1.05E-02
Future	Resident	Ingestion	TPA	1.45E-02	Pentachlorophenol	1.45E-02
Future	Resident	Inhalation	TPA	3.45E-07	Pentachlorophenol	3.45E-07
	Total			2.E-02		
Future	Resident	Dermal	WSA	2.57E-03	Pentachlorophenol	2.43E-03
Future	Resident	Ingestion	WSA	4.00E-03	Pentachlorophenol	3.35E-03
Future	Resident	Inhalation	WSA	9.51E-08	Pentachlorophenol	7.99E-08
	Total			7.E-03		
Future	Worker	Dermal	NPY	5.90E-03	Pentachlorophenol	5.90E-03
Future	Worker	Ingestion	NPY	7.18E-03	Pentachlorophenol	7.18E-03
Future	Worker	Inhalation	NPY	1.71E-07	Pentachlorophenol	1.71E-07
	Total			1.E-02		

Table 5-2

**Hazard Indices Associated with Surface Soil
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Hazard Index	Primary COPC	Primary COPC HQ
Future	Worker	Dermal	NTA	3.86E-03	Pentachlorophenol	3.86E-03
Future	Worker	Ingestion	NTA	4.70E-03	Pentachlorophenol	4.70E-03
Future	Worker	Inhalation	NTA	1.12E-07	Pentachlorophenol	1.12E-07
	Total			9.E-03		
Future	Worker	Dermal	SPY	9.66E-04	Pentachlorophenol	9.66E-04
Future	Worker	Ingestion	SPY	1.17E-03	Pentachlorophenol	1.17E-03
Future	Worker	Inhalation	SPY	2.80E-08	Pentachlorophenol	2.80E-08
	Total			2.E-03		
Future	Worker	Dermal	TPA	1.82E-03	Pentachlorophenol	1.82E-03
Future	Worker	Ingestion	TPA	2.22E-03	Pentachlorophenol	2.22E-03
Future	Worker	Inhalation	TPA	5.28E-08	Pentachlorophenol	5.28E-08
	Total			4.E-03		
Future	Worker	Dermal	WSA	4.47E-04	Pentachlorophenol	4.22E-04
Future	Worker	Ingestion	WSA	6.12E-04	Pentachlorophenol	5.13E-04
Future	Worker	Inhalation	WSA	1.46E-08	Pentachlorophenol	1.22E-08
	Total			1.E-03		

Key:

- COPC = Contaminant of potential concern.
- HI = Hazard index.
- NPY = North Pole Yard.
- NTA = North Treatment Area.
- SPOILS = Soil spoils piles near Little Squaticum Creek.
- SPY = South Pole Yard.
- TPA = Treated Pole Area.
- TPH = Total petroleum hydrocarbons.
- VPH = Volatile petroleum hydrocarbons.
- WSA = Wood Storage Area.

Table 5-3

**Excess Lifetime Cancer Risks Associated with Subsurface Soil (0 to 6 feet bgs)
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Future	Resident	Dermal	ETA/WTA	7.05E-04	B(a)P Equivalent	5.96E-04
Future	Resident	Ingestion	ETA/WTA	2.17E-03	B(a)P Equivalent	1.78E-03
Future	Resident	Inhalation	ETA/WTA	5.19E-08	Chrysene	1.62E-07
	Total			3.E-03		
Future	Resident	Dermal	NPY	2.05E-04	B(a)P Equivalent	9.86E-05
Future	Resident	Ingestion	NPY	6.54E-04	B(a)P Equivalent	2.95E-04
Future	Resident	Inhalation	NPY	2.19E-08	Dioxin TEQ	1.21E-08
	Total			9.E-04		
Future	Resident	Dermal	NTA	7.72E-04	B(a)P Equivalent	6.43E-04
Future	Resident	Ingestion	NTA	2.42E-03	B(a)P Equivalent	1.92E-03
Future	Resident	Inhalation	NTA	5.96E-08	B(a)P Equivalent	3.69E-08
	Total			3.E-03		
Future	Resident	Dermal	SPY	7.20E-06	B(a)P Equivalent	5.99E-06
Future	Resident	Ingestion	SPY	1.95E-05	B(a)P Equivalent	1.79E-05
Future	Resident	Inhalation	SPY	4.17E-10	B(a)P Equivalent	3.44E-10
	Total			3.E-05		
Future	Resident	Dermal	TPA	4.05E-05	B(a)P Equivalent	4.02E-05
Future	Resident	Ingestion	TPA	1.22E-04	B(a)P Equivalent	1.20E-04
Future	Resident	Inhalation	TPA	2.41E-09	B(a)P Equivalent	2.31E-09
	Total			2.E-04		
Future	Resident	Dermal	WSA	2.79E-05	Dioxin TEQ	2.12E-05
Future	Resident	Ingestion	WSA	1.70E-04	Dioxin TEQ	1.54E-04
Future	Resident	Inhalation	WSA	7.38E-09	Dioxin TEQ	6.98E-09
	Total			2.E-04		
Future	Worker	Dermal	ETA/WTA	3.44E-04	B(a)P Equivalent	2.90E-04
Future	Worker	Ingestion	ETA/WTA	9.70E-04	B(a)P Equivalent	7.95E-04
Future	Worker	Inhalation	ETA/WTA	1.22E-08	Chrysene	3.81E-08
	Total			1.E-03		
Future	Worker	Dermal	NPY	9.98E-05	B(a)P Equivalent	4.80E-05
Future	Worker	Ingestion	NPY	2.92E-04	B(a)P Equivalent	1.32E-04
Future	Worker	Inhalation	NPY	5.15E-09	Dioxin TEQ	2.84E-09
	Total			4.E-04		

Table 5-3

**Excess Lifetime Cancer Risks Associated with Subsurface Soil (0 to 6 feet bgs)
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Future	Worker	Dermal	NTA	3.76E-04	B(a)P Equivalent	3.13E-04
Future	Worker	Ingestion	NTA	1.08E-03	B(a)P Equivalent	8.58E-04
Future	Worker	Inhalation	NTA	1.40E-08	B(a)P Equivalent	8.68E-09
	Total			1.E-03		
Future	Worker	Dermal	SPY	3.51E-06	B(a)P Equivalent	2.92E-06
Future	Worker	Ingestion	SPY	8.71E-06	B(a)P Equivalent	7.99E-06
Future	Worker	Inhalation	SPY	9.80E-11	B(a)P Equivalent	8.08E-11
	Total			1.E-05		
Future	Worker	Dermal	TPA	1.97E-05	B(a)P Equivalent	1.96E-05
Future	Worker	Ingestion	TPA	5.47E-05	B(a)P Equivalent	5.37E-05
Future	Worker	Inhalation	TPA	5.66E-10	B(a)P Equivalent	5.43E-10
	Total			7.E-05		
Future	Worker	Dermal	WSA	1.36E-05	Dioxin TEQ	1.03E-05
Future	Worker	Ingestion	WSA	7.59E-05	Dioxin TEQ	6.89E-05
Future	Worker	Inhalation	WSA	1.73E-09	Dioxin TEQ	1.64E-09
	Total			9.E-05		
Future	Recreational Visitor	Dermal	SLOPE	4.23E-09	B(a)P Equivalent	4.23E-09
Future	Recreational Visitor	Ingestion	SLOPE	1.16E-08	B(a)P Equivalent	1.16E-08
Future	Recreational Visitor	Inhalation	SLOPE	4.68E-13	B(a)P Equivalent	4.68E-13
	Total			2.E-08		
Future	Recreational Visitor	Dermal	TRENCH	3.95E-09	B(a)P Equivalent	3.95E-09
Future	Recreational Visitor	Ingestion	TRENCH	1.08E-08	B(a)P Equivalent	1.08E-08
Future	Recreational Visitor	Inhalation	TRENCH	4.38E-13	B(a)P Equivalent	4.38E-13
	Total			1.E-08		

Key:

- COPC = Contaminant of potential concern.
- ETA = East Treatment Area.
- NPY = North Pole Yard.
- NTA = North Treatment Area.
- SLOPE = South Slope.
- SPY = South Pole Yard.
- TEQ = Toxic equivalency quotient.
- TPA = Treated Pole Area.
- TRENCH = Soil sample trench near Little Squaticum Creek.
- WSA = Wood Storage Area.
- WTA = West Treatment Area.

Table 5-4

**Hazard Indices Associated with Subsurface Soil (0 to 6 feet bgs)
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Hazard Index	Primary COPC	Primary COPC HQ
Future	Resident	Dermal	ETA/WTA	1.37E+00	Naphthalene	5.96E-01
Future	Resident	Ingestion	ETA/WTA	4.01E+00	Naphthalene	1.85E+00
Future	Resident	Inhalation	ETA/WTA	6.67E+01	Naphthalene	5.04E+01
	Total			7.E+01		
Future	Resident	Dermal	NPY	1.53E-01	Pentachlorophenol	1.51E-01
Future	Resident	Ingestion	NPY	1.21E+00	TPH Mixture	9.93E-01
Future	Resident	Inhalation	NPY	1.20E-01	2-Methylnaphthalene	1.20E-01
	Total			1.E+00		
Future	Resident	Dermal	NTA	1.68E-01	Pentachlorophenol	1.60E-01
Future	Resident	Ingestion	NTA	4.20E+00	TPH Mixture	3.96E+00
Future	Resident	Inhalation	NTA	6.60E-01	2-Methylnaphthalene	3.65E-01
	Total			5.E+00		
Future	Resident	Dermal	SPY	2.65E-03	Pentachlorophenol	2.65E-03
Future	Resident	Ingestion	SPY	3.67E-03	Pentachlorophenol	3.67E-03
Future	Resident	Inhalation	SPY	8.73E-08	Pentachlorophenol	8.73E-08
	Total			6.E-03		
Future	Resident	Dermal	TPA	8.01E-03	Naphthalene	5.14E-03
Future	Resident	Ingestion	TPA	2.49E-02	Naphthalene	1.60E-02
Future	Resident	Inhalation	TPA	6.78E-01	Naphthalene	4.35E-01
	Total			7.E-01		
Future	Resident	Dermal	WSA	8.53E-03	Pentachlorophenol	5.86E-03
Future	Resident	Ingestion	WSA	1.64E-02	2-Methylnaphthalene	8.31E-03
Future	Resident	Inhalation	WSA	2.26E-01	2-Methylnaphthalene	2.26E-01
	Total			3.E-01		
Future	Worker	Dermal	ETA/WTA	2.39E-01	Naphthalene	1.04E-01
Future	Worker	Ingestion	ETA/WTA	6.14E-01	Naphthalene	2.84E-01
Future	Worker	Inhalation	ETA/WTA	1.02E+01	Naphthalene	7.71E+00
	Total			11		
Future	Worker	Dermal	NPY	2.65E-02	Pentachlorophenol	2.63E-02
Future	Worker	Ingestion	NPY	1.42E-01	TPH Mixture	1.10E-01
Future	Worker	Inhalation	NPY	1.84E-02	2-Methylnaphthalene	1.84E-02
	Total			2.E-01		

Table 5-4

**Hazard Indices Associated with Subsurface Soil (0 to 6 feet bgs)
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Hazard Index	Primary COPC	Primary COPC HQ
Future	Worker	Dermal	NTA	2.93E-02	Pentachlorophenol	2.79E-02
Future	Worker	Ingestion	NTA	4.78E-01	TPH Mixture	4.41E-01
Future	Worker	Inhalation	NTA	1.01E-01	2-Methylnaphthalene	5.59E-02
	Total			6.E-01		
Future	Worker	Dermal	SPY	4.61E-04	Pentachlorophenol	4.61E-04
Future	Worker	Ingestion	SPY	5.61E-04	Pentachlorophenol	5.61E-04
Future	Worker	Inhalation	SPY	1.34E-08	Pentachlorophenol	1.34E-08
	Total			1.E-03		
Future	Worker	Dermal	TPA	1.39E-03	Naphthalene	8.93E-04
Future	Worker	Ingestion	TPA	3.82E-03	Naphthalene	2.45E-03
Future	Worker	Inhalation	TPA	1.04E-01	Naphthalene	6.65E-02
	Total			1.E-01		
Future	Worker	Dermal	WSA	1.48E-03	Pentachlorophenol	1.02E-03
Future	Worker	Ingestion	WSA	2.51E-03	2-Methylnaphthalene	1.27E-03
Future	Worker	Inhalation	WSA	3.46E-02	2-Methylnaphthalene	3.46E-02
	Total			4.E-02		

Key:

- COPC = Contaminant of potential concern.
- EPH = Extractable petroleum hydrocarbons.
- ETA = East Treatment Area.
- HI = Hazard index.
- NPY = North Pole Yard.
- NTA = North Treatment Area.
- SPY = South Pole Yard.
- TPA = Treated Pole Area.
- VPH = Volatile petroleum hydrocarbons.
- WSA = Wood Storage Area.
- WTA = West Treatment Area.

Table 5-5

**Excess Lifetime Cancer Risks Associated with Subsurface Soil (6 to 12 feet bgs)
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Future	Resident	Dermal	ETA/WTA	2.89E-04	B(a)P Equivalent	2.46E-04
Future	Resident	Ingestion	ETA/WTA	7.93E-04	B(a)P Equivalent	7.35E-04
Future	Resident	Inhalation	ETA/WTA	1.68E-08	Chrysene	4.10E-08
	Total			1E-03		
Future	Resident	Dermal	NPY	2.09E-06	Dioxin TEQ	1.82E-06
Future	Resident	Ingestion	NPY	1.41E-05	Dioxin TEQ	1.33E-05
Future	Resident	Inhalation	NPY	6.15E-10	Dioxin TEQ	6.00E-10
	Total			2E-05		
Future	Resident	Dermal	NTA	6.71E-05	Dioxin TEQ	2.97E-05
Future	Resident	Ingestion	NTA	3.14E-04	Dioxin TEQ	2.16E-04
Future	Resident	Inhalation	NTA	1.19E-08	Dioxin TEQ	9.76E-09
	Total			4E-04		
Future	Resident	Dermal	SPY	3.40E-04	B(a)P Equivalent	2.74E-04
Future	Resident	Ingestion	SPY	1.14E-03	B(a)P Equivalent	8.18E-04
Future	Resident	Inhalation	SPY	3.01E-08	B(a)P Equivalent	1.57E-08
	Total			1E-03		
Future	Resident	Dermal	TPA	5.65E-06	B(a)P Equivalent	3.74E-06
Future	Resident	Ingestion	TPA	1.49E-05	B(a)P Equivalent	1.12E-05
Future	Resident	Inhalation	TPA	3.84E-10	B(a)P Equivalent	2.15E-10
	Total			2E-05		
Future	Resident	Dermal	WSA	6.43E-06	Pentachlorophenol	5.52E-06
Future	Resident	Ingestion	WSA	1.00E-05	Benzo(a)pyrene	1.14E-05
Future	Resident	Inhalation	WSA	3.83E-10	Pentachlorophenol	3.31E-10
	Total			2E-05		
Future	Worker	Dermal	ETA/WTA	1.41E-04	B(a)P Equivalent	1.20E-04
Future	Worker	Ingestion	ETA/WTA	3.54E-04	B(a)P Equivalent	3.28E-04
Future	Worker	Inhalation	ETA/WTA	3.94E-09	Chrysene	9.63E-09
	Total			5E-04		
Future	Worker	Dermal	NPY	1.02E-06	Dioxin TEQ	8.88E-07
Future	Worker	Ingestion	NPY	6.28E-06	Dioxin TEQ	5.92E-06
Future	Worker	Inhalation	NPY	1.45E-10	Dioxin TEQ	1.41E-10
	Total			7E-06		

Table 5-5

**Excess Lifetime Cancer Risks Associated with Subsurface Soil (6 to 12 feet bgs)
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Future	Worker	Dermal	NTA	3.27E-05	Benzo(a)pyrene	1.51E-05
Future	Worker	Ingestion	NTA	1.40E-04	Dioxin TEQ	9.63E-05
Future	Worker	Inhalation	NTA	2.80E-09	Dioxin TEQ	2.29E-09
	Total			2E-04		
Future	Worker	Dermal	SPY	1.66E-04	B(a)P Equivalent	1.33E-04
Future	Worker	Ingestion	SPY	5.07E-04	B(a)P Equivalent	9.13E-05
Future	Worker	Inhalation	SPY	7.08E-09	B(a)P Equivalent	3.69E-09
	Total			7E-04		
Future	Worker	Dermal	TPA	2.75E-06	B(a)P Equivalent	1.82E-06
Future	Worker	Ingestion	TPA	6.67E-06	B(a)P Equivalent	5.00E-06
Future	Worker	Inhalation	TPA	9.03E-11	B(a)P Equivalent	5.05E-11
	Total			9E-06		
Future	Worker	Dermal	WSA	3.13E-06	Pentachlorophenol	2.69E-06
Future	Worker	Ingestion	WSA	4.48E-06	Benzo(a)pyrene	5.10E-06
Future	Worker	Inhalation	WSA	9.01E-11	Pentachlorophenol	7.79E-11
	Total			8E-06		
Future	Recreational Visitor	Dermal	SLOPE	1.56E-08	B(a)P Equivalent	1.27E-08
Future	Recreational Visitor	Ingestion	SLOPE	5.43E-08	B(a)P Equivalent	3.47E-08
Future	Recreational Visitor	Inhalation	SLOPE	3.27E-12	Dioxin TEQ	1.87E-12
	Total			7E-08		

Key:

- COPC = Contaminant of potential concern.
- ETA = East Treatment Area.
- NPY = North Pole Yard.
- NTA = North Treatment Area.
- SLOPE = South Slope.
- SPY = South Pole Yard.
- TEQ = Toxic equivalency quotient.
- TPA = Treated Pole Area.
- WSA = Wood Storage Area.
- WTA = West Treatment Area.

Table 5-6

**Hazard Indices Associated with Subsurface Soil (6 to 12 feet bgs)
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Hazard Index	Primary COPC	Primary COPC HQ
Future	Resident	Dermal	ETA/WTA	3.20E-01	Naphthalene	1.44E-01
Future	Resident	Ingestion	ETA/WTA	9.94E-01	Naphthalene	4.47E-01
Future	Resident	Inhalation	ETA/WTA	1.62E+01	Naphthalene	1.22E+01
	Total			1.7.E+01		
Future	Resident	Dermal	NPY	4.52E-03	2-Methylnaphthalene	4.52E-03
Future	Resident	Ingestion	NPY	8.52E-01	TPH Mixture	8.38E-01
Future	Resident	Inhalation	NPY	3.82E-01	2-Methylnaphthalene	3.82E-01
	Total			1.E+00		
Future	Resident	Dermal	NTA	1.54E-01	Naphthalene	8.42E-02
Future	Resident	Ingestion	NTA	3.61E+00	TPH Mixture	3.00E+00
Future	Resident	Inhalation	NTA	1.03E+01	Naphthalene	7.13E+00
	Total			1.E+01		
Future	Resident	Dermal	SPY	2.70E-01	Naphthalene	1.05E-01
Future	Resident	Ingestion	SPY	3.71E+00	TPH Mixture	2.98E+00
Future	Resident	Inhalation	SPY	1.51E+01	Naphthalene	8.86E+00
	Total			2.0.E+01		
Future	Resident	Dermal	TPA	8.42E-02	Naphthalene	6.37E-02
Future	Resident	Ingestion	TPA	2.56E-01	Naphthalene	1.98E-01
Future	Resident	Inhalation	TPA	6.81E+00	Naphthalene	5.39E+00
	Total			7.E+00		
Future	Resident	Dermal	WSA	2.27E-02	Pentachlorophenol	1.20E-02
Future	Resident	Ingestion	WSA	4.13E-01	TPH Mixture	3.63E-01
Future	Resident	Inhalation	WSA	9.04E-01	2-Methylnaphthalene	7.13E-01
	Total			1.E+00		
Future	Worker	Dermal	ETA/WTA	5.57E-02	Naphthalene	1.25E-02
Future	Worker	Ingestion	ETA/WTA	1.52E-01	Naphthalene	6.85E-02
Future	Worker	Inhalation	ETA/WTA	2.48E+00	Naphthalene	1.86E+00
	Total			3.E+00		
Future	Worker	Dermal	NPY	7.86E-04	2-Methylnaphthalene	7.86E-04
Future	Worker	Ingestion	NPY	9.32E-02	TPH Mixture	9.10E-02
Future	Worker	Inhalation	NPY	5.85E-02	2-Methylnaphthalene	5.85E-02
	Total			2.E-01		

Table 5-6

**Hazard Indices Associated with Subsurface Soil (6 to 12 feet bgs)
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Hazard Index	Primary COPC	Primary COPC HQ
Future	Worker	Dermal	NTA	2.68E-02	Naphthalene	1.46E-02
Future	Worker	Ingestion	NTA	4.15E-01	TPH Mixture	3.20E-01
Future	Worker	Inhalation	NTA	1.57E+00	Naphthalene	1.09E+00
	Total			2.E+00		
Future	Worker	Dermal	SPY	4.70E-02	Naphthalene	1.82E-02
Future	Worker	Ingestion	SPY	4.42E-01	TPH Mixture	3.30E-01
Future	Worker	Inhalation	SPY	2.31E+00	Naphthalene	1.36E+00
	Total			3.E+00		
Future	Worker	Dermal	TPA	1.47E-02	Naphthalene	1.11E-02
Future	Worker	Ingestion	TPA	3.91E-02	Naphthalene	3.03E-02
Future	Worker	Inhalation	TPA	1.04E+00	Naphthalene	8.25E-01
	Total			1.E+00		
Future	Worker	Dermal	WSA	3.95E-03	Pentachlorophenol	2.09E-03
Future	Worker	Ingestion	WSA	4.81E-02	TPH Mixture	4.05E-02
Future	Worker	Inhalation	WSA	1.38E-01	2-Methylnaphthalene	1.09E-01
	Total			2.E-01		

Key:

- COPC** = Contaminant of potential concern.
- EPH** = Extractable petroleum hydrocarbons.
- ETA** = East Treatment Area.
- HI** = Hazard index.
- NPY** = North Pole Yard.
- NTA** = North Treatment Area.
- SPY** = South Pole Yard.
- TPA** = Treated Pole Area.
- TPH** = Total petroleum hydrocarbons.
- VPH** = Volatile petroleum hydrocarbons.
- WSA** = Wood Storage Area.
- WTA** = West Treatment Area.

Table 5-7

**Excess Lifetime Cancer Risks Associated with Subsurface Soil (12 to 18 feet bgs)
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Future	Resident	Dermal	ETA/WTA	6.98E-04	B(a)P Equivalent	5.60E-04
Future	Resident	Ingestion	ETA/WTA	2.55E-03	B(a)P Equivalent	1.68E-03
Future	Resident	Inhalation	ETA/WTA	7.19E-08	Dioxin TEQ	3.85E-08
	Total			3.E-03		
Future	Resident	Dermal	NPY	3.08E-07	B(a)P Equivalent	3.08E-07
Future	Resident	Ingestion	NPY	9.20E-07	B(a)P Equivalent	9.20E-07
Future	Resident	Inhalation	NPY	1.77E-11	B(a)P Equivalent	1.77E-11
	Total			1.E-06		
Future	Resident	Dermal	NTA	9.13E-05	B(a)P Equivalent	7.58E-05
Future	Resident	Ingestion	NTA	2.47E-04	B(a)P Equivalent	2.27E-04
Future	Resident	Inhalation	NTA	5.29E-09	B(a)P Equivalent	4.35E-09
	Total			3.E-04		
Future	Resident	Dermal	SPY	2.55E-07	B(a)P Equivalent	2.55E-07
Future	Resident	Ingestion	SPY	7.61E-07	B(a)P Equivalent	7.61E-07
Future	Resident	Inhalation	SPY	1.46E-11	B(a)P Equivalent	1.46E-11
	Total			1.E-06		
Future	Resident	Dermal	WSA	8.04E-07	B(a)P Equivalent	8.04E-07
Future	Resident	Ingestion	WSA	2.40E-06	B(a)P Equivalent	2.40E-06
Future	Resident	Inhalation	WSA	4.62E-11	B(a)P Equivalent	4.62E-11
	Total			3.E-06		
Future	Worker	Dermal	ETA/WTA	3.40E-04	B(a)P Equivalent	2.73E-04
Future	Worker	Ingestion	ETA/WTA	1.14E-03	B(a)P Equivalent	7.48E-04
Future	Worker	Inhalation	ETA/WTA	1.69E-08	Dioxin TEQ	9.04E-09
	Total			1.E-03		
Future	Worker	Dermal	NPY	1.50E-07	B(a)P Equivalent	1.50E-07
Future	Worker	Ingestion	NPY	4.11E-07	B(a)P Equivalent	4.11E-07
Future	Worker	Inhalation	NPY	4.15E-12	B(a)P Equivalent	4.15E-12
	Total			6.E-07		
Future	Worker	Dermal	NTA	4.45E-05	B(a)P Equivalent	3.69E-05
Future	Worker	Ingestion	NTA	1.10E-04	B(a)P Equivalent	1.01E-04
Future	Worker	Inhalation	NTA		B(a)P Equivalent	1.02E-09
	Total			2.E-04		

Table 5-7

**Excess Lifetime Cancer Risks Associated with Subsurface Soil (12 to 18 feet bgs)
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Future	Worker	Dermal	SPY	1.24E-07	B(a)P Equivalent	1.24E-07
Future	Worker	Ingestion	SPY	3.40E-07	B(a)P Equivalent	3.40E-07
Future	Worker	Inhalation	SPY	3.44E-12	B(a)P Equivalent	3.44E-12
	Total			5.E-07		
Future	Worker	Dermal	WSA	3.92E-07	B(a)P Equivalent	3.92E-07
Future	Worker	Ingestion	WSA	1.07E-06	B(a)P Equivalent	1.07E-06
Future	Worker	Inhalation	WSA	1.08E-11	B(a)P Equivalent	1.08E-11
	Total			1.E-06		
Future	Recreational Visitor	Dermal	Slope	1.11E-08	B(a)P Equivalent	1.11E-08
Future	Recreational Visitor	Ingestion	Slope	3.04E-08	B(a)P Equivalent	3.04E-08
Future	Recreational Visitor	Inhalation	Slope	1.23E-12	B(a)P Equivalent	1.23E-12
	Total			4.E-08		

Key:

- COPC = Contaminant of potential concern.
- ETA = East Treatment Area.
- NPY = North Pole Yard.
- NTA = North Treatment Area.
- SLOPE = South Slope.
- SPY = South Pole Yard.
- TEQ = Toxic equivalency quotient.
- WSA = Wood Storage Area.
- WTA = West Treatment Area.

Table 5-8

**Hazard Indices Associated with Subsurface Soil (12 to 18 feet bgs)
Reasonable Maximum Exposure
Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Hazard Index	Primary COPC	Primary COPC HQ
Future	Resident	Dermal	ETA/WTA	8.63E-01	Naphthalene	3.70E-01
Future	Resident	Ingestion	ETA/WTA	3.37E+00	Naphthalene	1.15E+00
Future	Resident	Inhalation	ETA/WTA	4.50E+01	Naphthalene	3.13E+01
	Total			5.E+01		
Future	Resident	Dermal	NTA	2.89E-01	Naphthalene	1.13E-01
Future	Resident	Ingestion	NTA	1.37E+00	TPH Mixture	1.76E-01
Future	Resident	Inhalation	NTA	1.92E+01	Naphthalene	9.56E+00
	Total			2.E+01		
Future	Worker	Dermal	ETA/WTA	1.50E-01	Naphthalene	6.43E-02
Future	Worker	Ingestion	ETA/WTA	5.16E-01	Naphthalene	1.76E-01
Future	Worker	Inhalation	ETA/WTA	6.89E+00	Naphthalene	4.79E+00
	Total			8.E+00		
Future	Worker	Dermal	NTA	5.03E-02	Naphthalene	1.97E-02
Future	Worker	Ingestion	NTA	3.59E-01	TPH Mixture	1.76E-01
Future	Worker	Inhalation	NTA	2.93E+00	Naphthalene	1.46E+00
	Total			3.E+00		

Key:

- COPC = Contaminant of potential concern.
- EPH = Extractable petroleum hydrocarbons.
- ETA = East Treatment Area.
- HI = Hazard index.
- NTA = North Treatment Area.
- TPH = Total petroleum hydrocarbons.
- WTA = West Treatment Area.

Table 5-9

**Excess Lifetime Cancer Risks Associated with Sediment
Reasonable Maximum Exposure
Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Current/Future	Recreational Visitor	Dermal	SED WET	2E-08	B(a)P Equivalent	1.78E-08
Current/Future	Recreational Visitor	Dermal	SET OUT	1E-08	B(a)P Equivalent	8.76E-09
Current/Future	Recreational Visitor	Dermal	SED DOWN	5E-07	B(a)P Equivalent	3.46E-07
Current/Future	Recreational Visitor	Dermal	SED UP	8E-07	B(a)P Equivalent	4.06E-07

Key:

- COPC = Contaminant of potential concern.
- SED DOWN = Sediment sample collected downstream from Marine Drive.
- SED OUT = Sediment sample collected from the Birchwood Outfall upgradient of The Oeser Company.
- SED UP = Sediment sample collected upstream from Marine Drive.
- SED WET = Sediment sample collected from a wetland northeast of Little Squalicum Creek.

Table 5-10

**Excess Lifetime Cancer Risks Associated with Groundwater
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Current/Future	Worker	Ingestion	TC-5	8.E-06	2,3,7,8-TCDD TEQ	4.67E-06
Current/Future	Worker	Dermal	TC-5	4.E-04	2,3,7,8-TCDD TEQ	2.69E-04
	Total			4.E-04		
Current/Future	Worker	Ingestion	TC-6	5.E-06	B(a)P Equivalent	3.48E-06
Current/Future	Worker	Dermal	TC-6	2.E-04	B(a)P Equivalent	1.22E-04
	Total			2.E-04		
Future	Resident	Dermal	BKGD-MW06-D	7.3E-04	2,3,7,8-TCDD TEQ	5.12E-04
Future	Resident	Ingestion	BKGD-MW06-D	3.4E-05	2,3,7,8-TCDD TEQ	1.97E-05
	Total			8.E-04		
Future	Worker	Ingestion	BKGD-MW06-D	8.E-06	2,3,7,8-TCDD TEQ	4.63E-06
Future	Resident	Dermal	TC-5	7.4E-04	2,3,7,8-TCDD TEQ	5.17E-04
Future	Resident	Ingestion	TC-5	3.4E-05	2,3,7,8-TCDD TEQ	1.99E-04
	Total			8E-04		
Future	Resident	Dermal	TC-6	4.0E-04	B(a)P Equivalent	2.32E-04
Future	Resident	Ingestion	TC-6	2.2E-05	B(a)P Equivalent	1.48E-05
	Total			4E-04		
Future	Resident	Dermal	Ershigs-1a	7.4E-04	2,3,7,8-TCDD TEQ	5.57E-04
Future	Resident	Ingestion	Ershigs-1a	3.3E-05	2,3,7,8-TCDD TEQ	2.14E-05
	Total			8.E-04		
Future	Worker	Ingestion	Ershigs-1a	8.E-06	2,3,7,8-TCDD TEQ	5.04E-06
Future	Resident	Dermal	Ershigs-4a	5.7E-04	2,3,7,8-TCDD TEQ	3.44E-04
Future	Resident	Ingestion	Ershigs-4a	2.8E-05	B(a)P Equivalent	1.44E-05
	Total			6.E-04		
Future	Worker	Ingestion	Ershigs-4a	8.E-06	B(a)P Equivalent	3.38E-06
Future	Resident	Dermal	MW01-D	7.5E-04	2,3,7,8-TCDD TEQ	5.30E-04
Future	Resident	Ingestion	MW01-D	3.5E-05	2,3,7,8-TCDD TEQ	2.04E-05
	Total			8.E-04		
Future	Worker	Ingestion	MW01-D	8.E-06	2,3,7,8-TCDD TEQ	4.80E-06
Future	Resident	Dermal	MW02-D	4.6E-04	2,3,7,8-TCDD TEQ	2.38E-04
Future	Resident	Ingestion	MW02-D	2.3E-05	B(a)P Equivalent	1.42E-05
	Total			5.E-04		
Future	Worker	Ingestion	MW02-D	8.E-06	B(a)P Equivalent	3.34E-06

Table 5-10

**Excess Lifetime Cancer Risks Associated with Groundwater
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Future	Resident	Dermal	MW03-D	9.6E-04	2,3,7,8-TCDD TEQ	7.96E-04
Future	Resident	Ingestion	MW03-D	4.1E-05	2,3,7,8-TCDD TEQ	3.06E-05
	Total			1.E-03		
Future	Worker	Ingestion	MW03-D	8.E-06	2,3,7,8-TCDD TEQ	7.20E-06
Future	Resident	Dermal	MW05-D	7.8E-04	2,3,7,8-TCDD TEQ	4.50E-04
Future	Resident	Ingestion	MW05-D	4.2E-05	2,3,7,8-TCDD TEQ	1.73E-05
	Total			8.E-04		
Future	Worker	Ingestion	MW05-D	8.E-06	2,3,7,8-TCDD TEQ	4.07E-06
Future	Resident	Dermal	MW17-D	6.6E-04	2,3,7,8-TCDD TEQ	4.23E-04
Future	Resident	Ingestion	MW17-D	3.1E-05	2,3,7,8-TCDD TEQ	1.63E-05
	Total			7.E-04		
Future	Worker	Ingestion	MW17-D	8.E-06	2,3,7,8-TCDD TEQ	3.82E-06
Future	Resident	Dermal	MW18-D	5.5E-04	2,3,7,8-TCDD TEQ	3.21E-04
Future	Resident	Ingestion	MW18-D	2.7E-05	B(a)P Equivalent	1.43E-05
	Total			6.E-04		
Future	Worker	Ingestion	MW18-D	8.E-06	B(a)P Equivalent	3.36E-06
Future	Resident	Dermal	MW20-D	5.5E-04	2,3,7,8-TCDD TEQ	3.25E-04
Future	Resident	Ingestion	MW20-D	2.7E-05	B(a)P Equivalent	1.44E-05
	Total			6.E-04		
Future	Worker	Ingestion	MW20-D	8.E-06	B(a)P Equivalent	3.39E-06
Future	Resident	Dermal	MW23-D	7.0E-04	2,3,7,8-TCDD TEQ	4.68E-04
Future	Resident	Ingestion	MW23-D	3.3E-05	2,3,7,8-TCDD TEQ	1.80E-05
	Total			7.E-04		
Future	Worker	Ingestion	MW23-D	8.E-06	2,3,7,8-TCDD TEQ	4.23E-06
Future	Resident	Dermal	MW24-D	6.3E-04	2,3,7,8-TCDD TEQ	4.01E-04
Future	Resident	Ingestion	MW24-D	3.0E-05	2,3,7,8-TCDD TEQ	1.54E-05
	Total			7.E-04		
Future	Worker	Ingestion	MW24-D	8.E-06	2,3,7,8-TCDD TEQ	3.62E-06
Future	Resident	Dermal	MW25-D	6.4E-04	2,3,7,8-TCDD TEQ	3.83E-04
Future	Resident	Ingestion	MW25-D	3.2E-05	2,3,7,8-TCDD TEQ	1.47E-05
	Total			7.E-04		
Future	Worker	Ingestion	MW25-D	8.E-06	2,3,7,8-TCDD TEQ	3.46E-06

Table 5-10

**Excess Lifetime Cancer Risks Associated with Groundwater
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Future	Resident	Dermal	MW30-D	7.5E-04	2,3,7,8-TCDD TEQ	5.34E-04
Future	Resident	Ingestion	MW30-D	3.5E-05	2,3,7,8-TCDD TEQ	2.06E-05
	Total			8.E-04		
Future	Worker	Ingestion	MW30-D	8.E-06	2,3,7,8-TCDD TEQ	4.83E-06
Future	Resident	Dermal	MW33-D	5.3E-04	2,3,7,8-TCDD TEQ	3.01E-04
Future	Resident	Ingestion	MW33-D	2.6E-05	B(a)P Equivalent	1.43E-05
	Total			6.E-04		
Future	Worker	Ingestion	MW33-D	8.E-06	B(a)P Equivalent	3.36E-06
Future	Resident	Dermal	MW34-D	8.7E-04	2,3,7,8-TCDD TEQ	6.34E-04
Future	Resident	Ingestion	MW34-D	4.0E-05	2,3,7,8-TCDD TEQ	2.44E-05
	Total			9.E-04		
Future	Worker	Ingestion	MW34-D	8.E-06	2,3,7,8-TCDD TEQ	5.73E-06
Future	Resident	Dermal	MW35-D	5.8E-04	2,3,7,8-TCDD TEQ	3.54E-04
Future	Resident	Ingestion	MW35-D	2.8E-05	B(a)P Equivalent	1.44E-05
	Total			6.E-04		
Future	Worker	Ingestion	MW35-D	8.E-06	B(a)P Equivalent	3.38E-06

Key:

BKGD = Background.
 COPC = Chemical of potential concern.
 TEQ = Toxic equivalency quotient.

<p align="center">Table 5-11</p> <p align="center">Total Hazard Indices Associated with Groundwater</p> <p align="center">Reasonable Maximum Exposure</p> <p align="center">The Oeser Company</p> <p align="center">Bellingham, Washington</p>						
Scenario	Receptor Population	Pathway	Location	Hazard Index	Primary COPC	Primary COPC HQ
Future	Resident	Dermal	MW02-D	7.7E-05	Naphthalene	7.74E-05
Future	Resident	Ingestion	MW02-D	8.4E-04	Naphthalene	8.44E-04
Future	Resident	Inhalation	MW02-D	9.8E-02	Naphthalene	9.81E-02
	Total			1.E-01		
Future	Worker	Ingestion	MW02-D	1.E-04	Naphthalene	1.29E-04
Future	Resident	Ingestion/ Inhalation	MW03-D	5.E-01	EPH	4.98E-01
Future	Resident	Dermal	MW05-D	2.3E-02	Pentachlorophenol	2.30E-02
Future	Resident	Ingestion	MW05-D	1.1E-02	Pentachlorophenol	1.11E-02
	Total			3.E-02		
Future	Worker	Ingestion	MW05-D	2.E-03	Pentachlorophenol	1.70E-03
Future	Resident	Dermal	MW20-D	6.4E-05	Naphthalene	6.39E-05
Future	Resident	Ingestion	MW20-D	7.0E-04	Naphthalene	6.97E-04
Future	Resident	Inhalation	MW20-D	8.1E-02	Naphthalene	8.11E-02
	Total			8.E-02		
Future	Worker	Ingestion	MW20-D	1.E-04	Naphthalene	1.07E-04
Future	Resident	Dermal	MW25-D	7.8E-03	Pentachlorophenol	7.82E-03
Future	Resident	Ingestion	MW25-D	3.8E-03	Pentachlorophenol	3.79E-03
	Total			1.E-02		
Future	Worker	Ingestion	MW25-D	6.E-04	Pentachlorophenol	5.80E-04

Key:

- COPC = Contaminant of potential concern.
- EPH = Extractable petroleum hydrocarbon.
- HI = Hazard index.

Table 5-12

**Excess Lifetime Cancer Risks Associated with Surface Water
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington**

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Current/Future	Recreational Visitor	Dermal	BKGD-CREEK	1.E-04	2,3,7,8-TCDD TEQ	8.4E-05
Current/Future	Recreational Visitor	Dermal	CREEK	5.E-04	2,3,7,8-TCDD TEQ	4.6E-04

Key:

- BKGD = Background sample collected from Little Squalicum Creek.
- COPC = Chemical of potential concern.
- CREEK = Little Squalicum Creek.
- TEQ = Toxic equivalency quotient.

Table 5-13

Total Hazard Indices Associated with Surface Water
 Reasonable Maximum Exposure
 The Oeser Company
 Bellingham, Washington

Scenario	Receptor Population	Pathway	Location	Hazard Index	Primary COPC	Primary COPC HQ
Current/Future	Recreational Visitor	Dermal	CREEK	5.E-02	Pentachlorophenol	4.97E-02

Key:

- COPC = Chemical of potential concern.
- CREEK = Little Squalicum Creek.
- HI = Hazard index.

Table 5-14

Excess Lifetime Cancer Risks Associated with Air
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington

Scenario	Receptor Population	Pathway	Location	Risk	Primary COPC	Primary COPC Risk
Current	Recreational Visitor	Inhalation	BKGD-AS30	4.E-07	Benzene	2.58E-07
Current	Resident	Inhalation	BKGD-AS30	1.E-05	Benzene	8.41E-06
Current	Recreational Visitor	Inhalation	AS24	2.E-07	Benzene	1.63E-07
Current	Resident	Inhalation	AS24	7.E-06	Benzene	5.32E-06
Current	Recreational Visitor	Inhalation	AS25	3.E-07	Benzene	1.72E-07
Current	Resident	Inhalation	AS25	1.E-05	Benzene	5.60E-06
Current	Recreational Visitor	Inhalation	AS26	8.E-08	2,3,7,8-TCDD TEQ	5.16E-08
Current	Resident	Inhalation	AS26	3.E-06	2,3,7,8-TCDD TEQ	1.68E-06
Current	Recreational Visitor	Inhalation	AS27	2.E-07	2,3,7,8-TCDD TEQ	1.28E-07
Current	Resident	Inhalation	AS27	7.E-06	2,3,7,8-TCDD TEQ	4.15E-06
Current	Recreational Visitor	Inhalation	AS28	3.E-07	Benzene	1.77E-07
Current	Resident	Inhalation	AS28	8.E-06	Benzene	5.75E-06
Current	Recreational Visitor	Inhalation	AS29	1.E-06	Pentachlorophenol	6.03E-07
Current	Resident	Inhalation	AS29	3.E-05	Pentachlorophenol	1.96E-05
Current	Recreational Visitor	Inhalation	AS32	3.E-07	Benzene	1.41E-07
Current	Resident	Inhalation	AS32	9.E-06	Benzene	4.60E-06
Current	Recreational Visitor	Inhalation	AS33	9.E-08	2,3,7,8-TCDD TEQ	8.54E-08
Current	Resident	Inhalation	AS33	3.E-06	2,3,7,8-TCDD TEQ	2.78E-06

Key:

BKGD = Background.
COPC = Chemical of potential concern.
TEQ = Toxic equivalency quotient.

Table 5-15

Total Hazard Indices Associated with Air
Reasonable Maximum Exposure
The Oeser Company
Bellingham, Washington

Scenario	Receptor Population	Pathway	Location	Hazard Index	Primary COPC	Primary COPC HQ
Current	Resident	Inhalation	BKGD-AS30	2.E+00	1,2,4-Trimethylbenzene	9.16E-01
Current	Recreational Visitor	Inhalation	BKGD-AS30	8.E-02	1,2,4-Trimethylbenzene	4.17E-02
Current	Resident	Inhalation	AS24	1.E+00	1,2,4-Trimethylbenzene	4.49E-01
Current	Recreational Visitor	Inhalation	AS24	4.E-02	1,2,4-Trimethylbenzene	2.04E-02
Current	Resident	Inhalation	AS25	3.E+00	1,2,4-Trimethylbenzene	1.83E+00
Current	Recreational Visitor	Inhalation	AS25	1.E-01	1,2,4-Trimethylbenzene	8.31E-02
Current	Resident	Inhalation	AS26	6.E-02	Benzene	5.70E-02
Current	Recreational Visitor	Inhalation	AS26	3.E-03	Benzene	2.59E-03
Current	Resident	Inhalation	AS27	8.E-01	1,2,4-Trimethylbenzene	3.32E-01
Current	Recreational Visitor	Inhalation	AS27	4.E-02	1,2,4-Trimethylbenzene	1.51E-02
Current	Resident	Inhalation	AS28	1.E+00	1,2,4-Trimethylbenzene	6.56E-01
Current	Recreational Visitor	Inhalation	AS28	7.E-02	1,2,4-Trimethylbenzene	2.99E-02
Current	Resident	Inhalation	AS29	5.E+00	1,2,4-Trimethylbenzene	1.89E+00
Current	Recreational Visitor	Inhalation	AS29	2.E-01	1,2,4-Trimethylbenzene	8.61E-02
Current	Resident	Inhalation	AS32	1.E+00	1,2,4-Trimethylbenzene	5.14E-01
Current	Recreational Visitor	Inhalation	AS32	5.E-02	1,2,4-Trimethylbenzene	2.34E-02

Key:

BKGD = Background.
COPC = Chemical of potential concern.
HI = Hazard index.

Figure 5-1
Excess Lifetime Cancer Risks for Residents
Open Area Samples
The Oeser Company
Bellingham, Washington

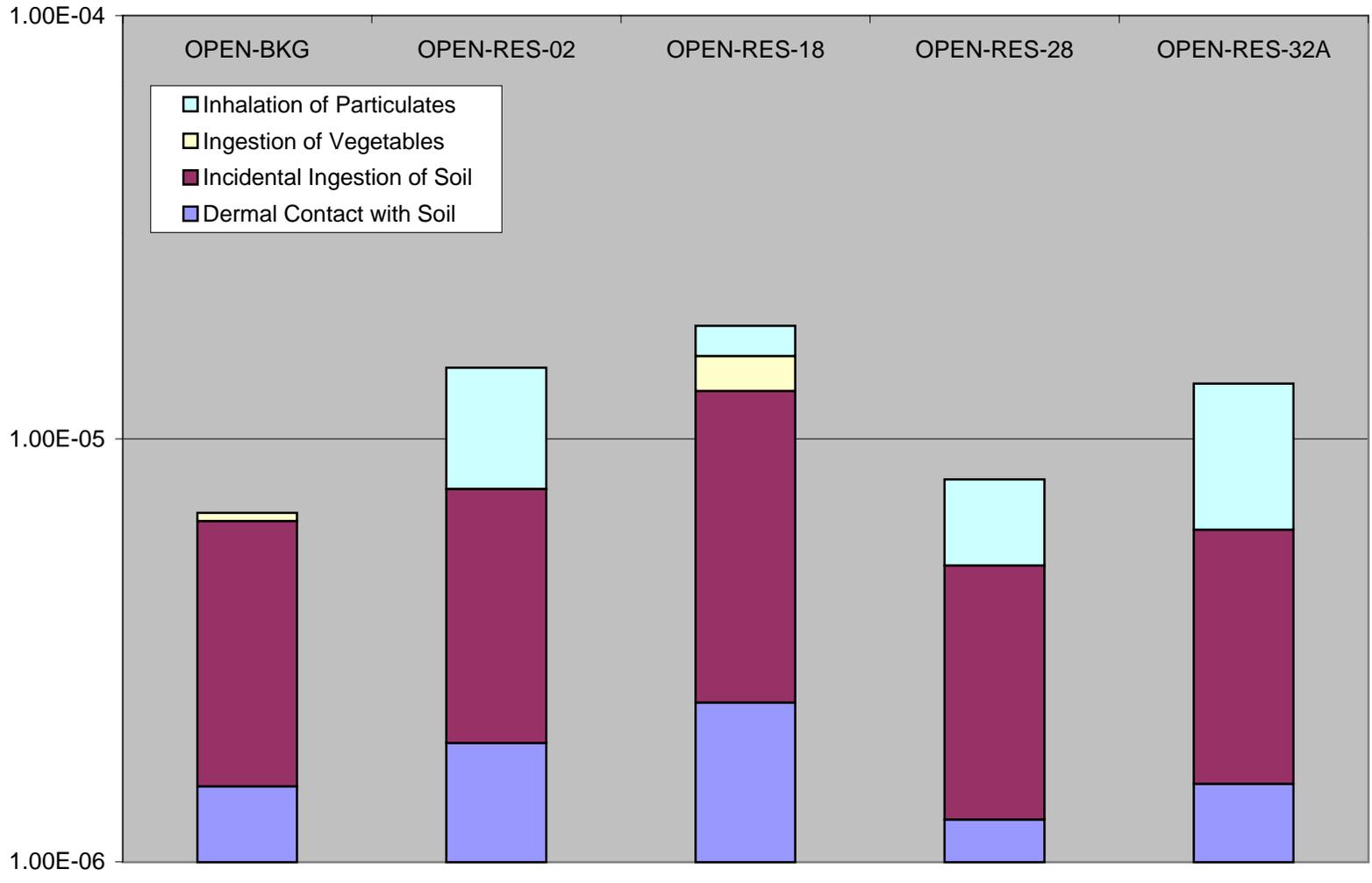
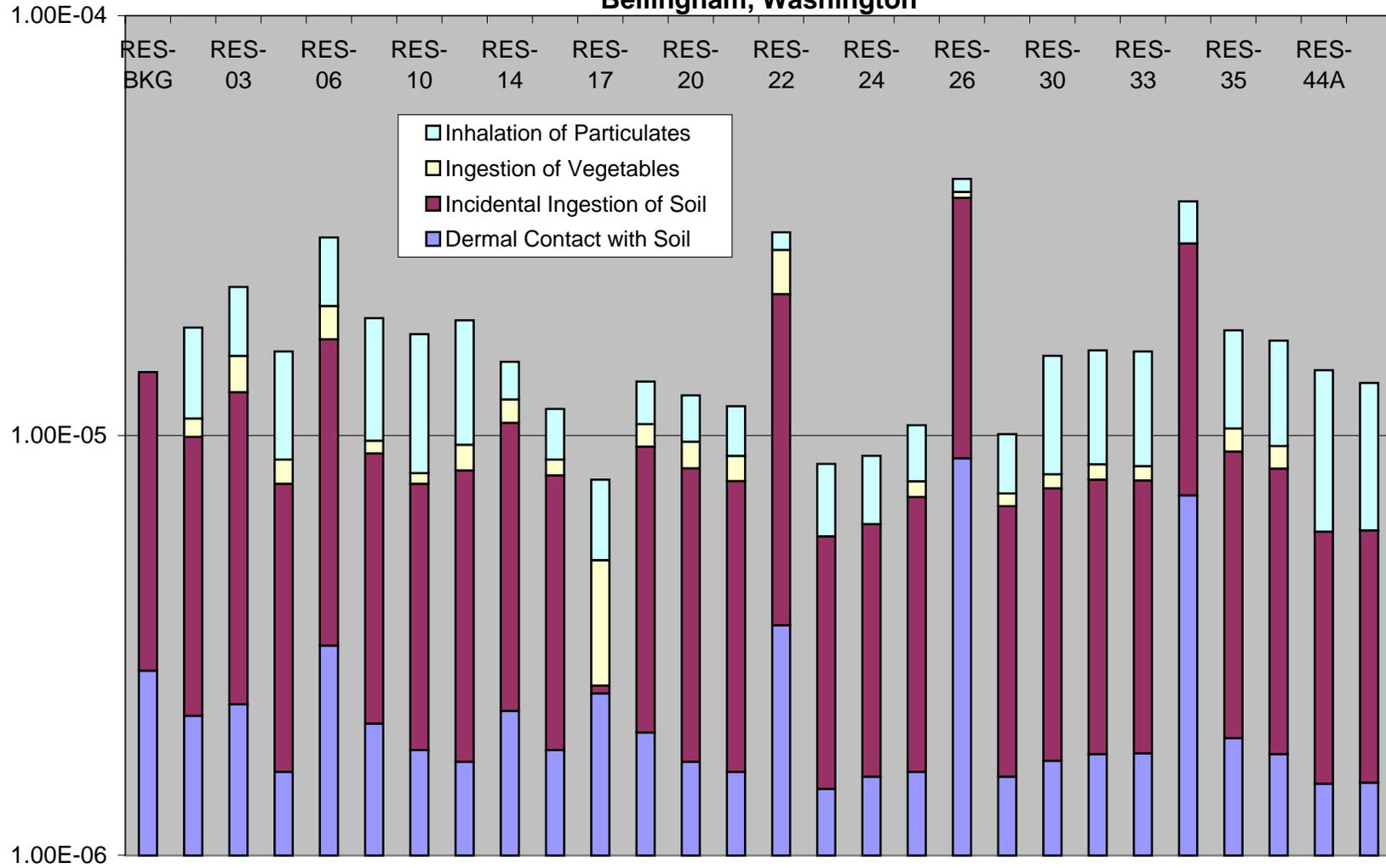


Figure 5-2
Excess Lifetime Cancer Risks for Residents
Biased Samples
The Oeser Company
Bellingham, Washington



6. SUMMARY AND CONCLUSIONS

The Oeser Company is an active wood-treating facility located in Bellingham, Washington, that has used organic treating solutions of creosote and PCP to preserve utility poles and pilings. The primary objective of this baseline HHRA was to evaluate potential adverse health effects attributable to site-related contaminants in the absence of remedial action. Contaminants from wood-treating wastes (PAHs [most compounds that make up creosote], PCP, and dioxins/furans [contaminants found in PCP treating solutions]) were the primary COPCs in surface and subsurface soil, groundwater, air, surface water, and sediment.

Current and future exposure scenarios were evaluated for on-site workers, on- and off-site residents, and off-site recreational visitors. Exposure to COPCs derived from facility surface soil was evaluated for the current on-site worker. For the current off-site residents, exposure to COPCs in off-site surface soil and air was evaluated. Exposure to COPCs derived from off-site surface soil, Little Squalicum Creek surface water and sediment, and air was evaluated for the current recreational visitor. For the future exposure scenario, exposure to COPCs derived from on-site surface and subsurface soil and groundwater was evaluated for on-site workers and on-site residents. For the future off-site resident, exposure to COPCs derived from soil was evaluated. Exposure to COPCs derived from surface and subsurface soil and Little Squalicum Creek surface water and sediment was evaluated for the future recreational visitor. The potential excess lifetime cancer risks and potential noncarcinogenic HIs for the RME case are summarized below.

6.1 CURRENT EXPOSURE SCENARIO

For the current exposure scenario, potential excess lifetime cancer risks and potential noncarcinogenic HIs were determined for the on-site worker, off-site resident, and off-site recreational visitor.

The potential RME excess lifetime cancer risks for the on-facility worker (1E-03 to 5E-04) associated with exposure to currently exposed surface soils exceeded EPA levels of concern. Dioxins/furans were the main contributors to the risks. Noncancer HIs were below the EPA's

acceptable level of 1. Most of the site is capped with either gravel or asphalt; therefore, exposure to surface soil under current conditions is limited to a few uncapped areas (Figure 6-1).

For the off-facility residents, potential excess lifetime cancer risks associated with exposure to surface soil were less than $1E-04$ for all but one location (OPEN-RES-53, estimated cancer risk of $2E-04$). However, this location is not a current residence; rather, it is an industrial property east of the site. Several locations that were noted as “residential” currently are undeveloped or are developed for commercial uses. These include Residence 42A ($8E-05$), Residence 43 ($3E-05$), Residence 50 ($7E-05$), Residence 53 ($2E-04$), and Residence 54 ($7E-05$). The COPCs contributing most to risk estimates were B(a)P equivalents and dioxin TEQ. The biased residential background sample and the open residential background sample were below EPA levels of concern.

For the off-site recreational visitor, potential excess lifetime cancer risks associated with exposure to surface soil were within EPA’s range of acceptable risks. The only noncancer HI (0.5) associated with exposure to surface soil is less than the EPA acceptable level for the recreational visitor. This estimate is for potential exposures at the spoils piles and is primarily due to TPH.

The potential excess lifetime cancer risks and potential noncancer HIs associated with exposure to sediment in Little Squalicum Creek were less than EPA acceptable levels for the recreational visitor.

The potential excess lifetime cancer risks associated with dermal exposure of the recreational visitor to the surface water of Little Squalicum Creek is $5E-04$. The risk was attributed mainly to dioxins/furans, but B(a)P and PCP also contributed to risk. The risk associated with the background surface water location was $1E-04$; however, this risk is based on one-half DLs for nondetected compounds. Potential noncancer effects associated with exposure to surface water were less than EPA acceptable levels.

As described in section 5.4.2 of this document, the assessment of risks and hazards from dermal contact via water to very lipophilic molecules, such as TCDD, B(a)P and PCP, is highly uncertain. Their dermal permeability coefficients are outside the effective predictive domain, and therefore the estimations of doses received from dermal contact are considered to be less than reliable, and probably leads to significant overestimates of risks and hazards. In addition, estimation of EPCs in surface water is inherently uncertain, since the concentrations of COPCs in the creek are unlikely to be constant over time. Finally, the frequency and duration that the recreational visitor actually comes into contact with the creek water is probably highly variable. The values used to estimate frequencies and durations of exposures to the creek water in this risk assessment were based on best professional judgment and were intended to be conservative.

The potential excess lifetime cancer risks associated with inhalation of COPCs in air were within the EPA's acceptable range. Penta-, hexa-, and hepta-chlorinated dioxin congeners and benzene were detected at AS29, which had the highest risks ($3E-05$) for the off-site resident, but at similar concentrations as those detected at the background location. PCP was not detected at the background sampling location. Therefore, the estimated excess lifetime cancer risks at AS29 probably are attributable to operations of The Oeser Company. Noncancer HIs exceeded the EPA's acceptable level of 1 at sampling locations AS25 and AS29. These locations had HIs of 3 and 5, respectively, slightly above the background location HI of 2. The main COPC contributing to the elevated HI in AS25 was 1,2,4-trimethylbenzene. Increased concentrations of 1,2,4-trimethylbenzene; 2-methylnaphthalene; PCP; and dibenzofurans were the main contributors to the increased HIs at sampling location AS29. 1,2,4-Trimethylbenzene; 1,3,5-trimethylbenzene; and benzene were COPCs at the background sampling location that contributed to the elevated HI of 2. The increased HI associated with compounds detected at AS29 probably is due to facility operations. Sampling stations AS29 and AS25 were located at The Oeser Company's northeast fence line, which is located directly downwind of the facility. In addition, air concentration data derived from the air monitoring stations may not represent steady-state concentrations. These concentrations can vary greatly depending on local atmospheric conditions such as wind speed, wind direction, and precipitation. Facility operations also may greatly influence contaminant concentrations. The increased potential excess lifetime cancer risks and HIs attributed to detected air concentrations may be overestimated or underestimated, depending on how close these values are to the actual average long-term (i.e., 30-year) air concentrations to which the off-site residents potentially would be exposed.

Potential excess lifetime cancer risks and HIs were within acceptable levels for air exposures for the recreational visitor.

The total cancer risk across all COPCs for the Tilbury Cement Company groundwater wells exceeded the EPA criteria for the current worker scenario. Dermal exposure to groundwater while showering contributed the greatest risk at TC-5 ($4E-04$) and TC-6 ($2E-4$). However, no COPCs were detected in these wells; the estimated excess lifetime cancer risks for on-facility worker exposure to groundwater is based solely on the use of one-half DLs for nondetected compounds. Consequently, actual risks to on-facility workers may be even less. No noncarcinogenic COPCs were identified.

6.2 FUTURE EXPOSURE SCENARIO

For the future exposure scenario, potential excess lifetime cancer risks and potential noncarcinogenic HIs were determined for the on-site worker, on-site resident, and off-site recreational visitor.

The potential excess lifetime cancer risks associated with surface soils exceeded EPA criteria for the on-facility resident (2E-03 to 7E-03) and the on-facility worker (6E-04 to 2E-03). The risks were attributed primarily to detected dioxins/furans. Noncancer HIs were below the EPA's acceptable level of 1. For this exposure scenario, it was assumed that all soil caps were removed; therefore, all surface soil samples were evaluated (Tables 2-2a through 2-2d).

The potential excess lifetime cancer risks for the future on-site resident associated with exposure to subsurface soil exceeded EPA criteria for every subarea and multiple depth intervals (Table 6-1). The upper depth intervals greatly exceeded EPA acceptable levels, with decreasing risks at lower depth intervals; however, the risks attributed to the subsurface soil of the East and West Treatment areas and the North Treatment Area exceeded EPA acceptable levels at every depth interval. In most cases, cPAHs and/or dioxins/furans were the main chemicals contributing to the risk, but PCP and TPH also were detected throughout the subsurface soil. HIs for all subarea subsurface soils for the future on-site resident generally increased with depth, with the highest HIs found in the 6- to 12-foot interval for all areas except the East and West Treatment Areas and the North Treatment Area (Table 6-2). HIs for all subareas exceeded 1 within this depth interval, except the Wood Storage Area. HIs for the East and West Treatment areas and the North Treatment Area exceeded 1 in all subsurface soil intervals. The increased HIs were attributed to naphthalene and 2-methylnaphthalene.

Similar to the on-site future resident, the potential excess lifetime cancer risks for the on-site future worker exceeded the EPA's acceptable risk range throughout subsurface depth intervals (Table 6-3). The HIs for the on-site future worker generally increased with depth for each subarea, with the highest HIs across all areas found in the 6- to 12-foot interval, with the exception of the East and West Treatment Areas and North Treatment Area (Table 6-4). All subareas exceeded 1 within this depth interval, except the North Pole Yard and the Wood Storage Area. HIs for the East and West Treatment areas and the North Treatment Area exceeded 1 in all subsurface soil intervals.

The potential excess lifetime cancer risks for the potential future on-site resident exceeded EPA acceptable levels for all deep water groundwater wells and the background well. The COPCs that have contributed to risks for each well are the 2,3,7,8-TCDD TEQ and B(a)P equivalents. However, the concentrations of individual dioxin/furan congeners and cPAHs did not exceed their respective screening

toxicity values, and the calculation of the 2,3,7,8-TCDD TEQ and B(a)P equivalents is based largely on the use of one-half DLs for nondetected compounds. Given that the risk levels in the background well exceed EPA acceptable risk levels and that primary COPC concentrations were calculated based on one-half DLs, the risks associated with use of groundwater likely are overestimated. HIs for the on-facility resident were less than 1.

Potential excess lifetime cancer risks and HIs for the future on-site worker were below EPA criteria for exposure to groundwater. Excess lifetime cancer risks ranges from 6E-06 to 1E-05 for on-site wells, while the excess lifetime cancer risk for the background well is 8E-06. At least one dioxin congener was detected in each well, however, the majority of risk calculated for groundwater exposure is due to use of one-half of the detection limits for dioxin congeners and PAHs.

Table 6-1

Excess Lifetime Cancer Risks Associated with Facility Soil
 Residential Exposure Scenario
 Reasonable Maximum Exposure
 The Oeser Company
 Bellingham, Washington

Depth (feet bgs)	Facility Subarea					
	ETA/WTA	NPY	NTA	SPY	TPA	WSA
Surface	NA	7.E-03	6.E-03	4.E-03	3.E-03	2.E-03
0 to 6	3.E-03	9.E-04	3.E-03	3.E-05	2.E-04	2.E-04
6 to 12	1.E-03	2E-05	4.E-04	1.E-03	2.E-05	2.E-05
12 to 18	3.E-03	1.E-06	3.E-04	1.E-06	NQ	3.E-06

Key:

- ETA = East Treatment Area.
- NA = Not applicable (Surface soil samples were not collected.).
- NPY = North Pole Area.
- NQ = Not quantified. There were no contaminants of potential concern.
- NTA = North Treatment Area.
- SPY = South Pole Area.
- TPA = Treated Pole Area.
- WSA = Wood Storage Area.
- WTA = West Treatment Area.

Table 6-2

Hazard Indices Associated with Facility Soil
 Residential Exposure Scenario
 Reasonable Maximum Exposure
 The Oeser Company
 Bellingham, Washington

Depth (feet bgs)	Facility Subarea					
	ETA/WTA	NPY	NTA	SPY	TPA	WSA
Surface	NA	0.08	0.05	0.01	0.02	0.007
0 to 6	72	1	5	0.006	0.7	0.3
6 to 12	17	1	11	16	7	1
12 to 18	49	NQ	21	NQ	NQ	NQ

Key:

- ETA = East Treatment Area.
- NA = Not applicable (Surface soil samples were not collected.).
- NPY = North Pole Area.
- NQ = Not quantified. There were no contaminants of potential concern.
- NTA = North Treatment Area.
- SPY = South Pole Area.
- TPA = Treated Pole Area.
- WSA = Wood Storage Area.
- WTA = West Treatment Area.

Table 6-3

Excess Lifetime Cancer Risks Associated with Facility Soil
 Worker Exposure Scenario
 Reasonable Maximum Exposure
 The Oeser Company
 Bellingham, Washington

Depth (feet bgs)	Facility Subarea					
	ETA/WTA	NPY	NTA	SPY	TPA	WSA
Surface	NA	2.E-03	2.E-03	1.E-03	9.E-04	6.E-04
0 to 6	1.E-03	4.E-04	2.E-03	1.E-05	7.E-05	9.E-05
6 to 12	5.E-04	7E-06	1.E-03	7.E-04	9.E-06	8.E-06
12 to 18	1.E-03	6.E-07	2.E-04	5.E-07	NQ	1.E-06

Key:

- ETA = East Treatment Area.
- NA = Not applicable (Surface soil samples were not collected.).
- NPY = North Pole Area.
- NQ = Not quantified. There were no contaminants of potential concern.
- NTA = North Treatment Area.
- SPY = South Pole Area.
- TPA = Treated Pole Area.
- WSA = Wood Storage Area.
- WTA = West Treatment Area.

Table 6-4

Hazard Indices Associated with Facility Soil
 Worker Exposure Scenario
 Reasonable Maximum Exposure
 The Oeser Company
 Bellingham, Washington

Depth (feet bgs)	Facility Subarea					
	ETA/WTA	NPY	NTA	SPY	TPA	WSA
Surface	NA	0.01	0.009	0.002	0.004	0.001
0 to 6	11	0.2	0.6	0.001	0.1	0.04
6 to 12	3	0.2	2	3	1	0.2
12 to 18	8	NQ	3	NQ	NQ	NQ

Key:

- ETA = East Treatment Area.
- NA = Not applicable (Surface soil samples were not collected.).
- NPY = North Pole Area.
- NQ = Not quantified. There were no contaminants of potential concern.
- NTA = North Treatment Area.
- SPY = South Pole Area.
- TPA = Treated Pole Area.
- WSA = Wood Storage Area.
- WTA = West Treatment Area.

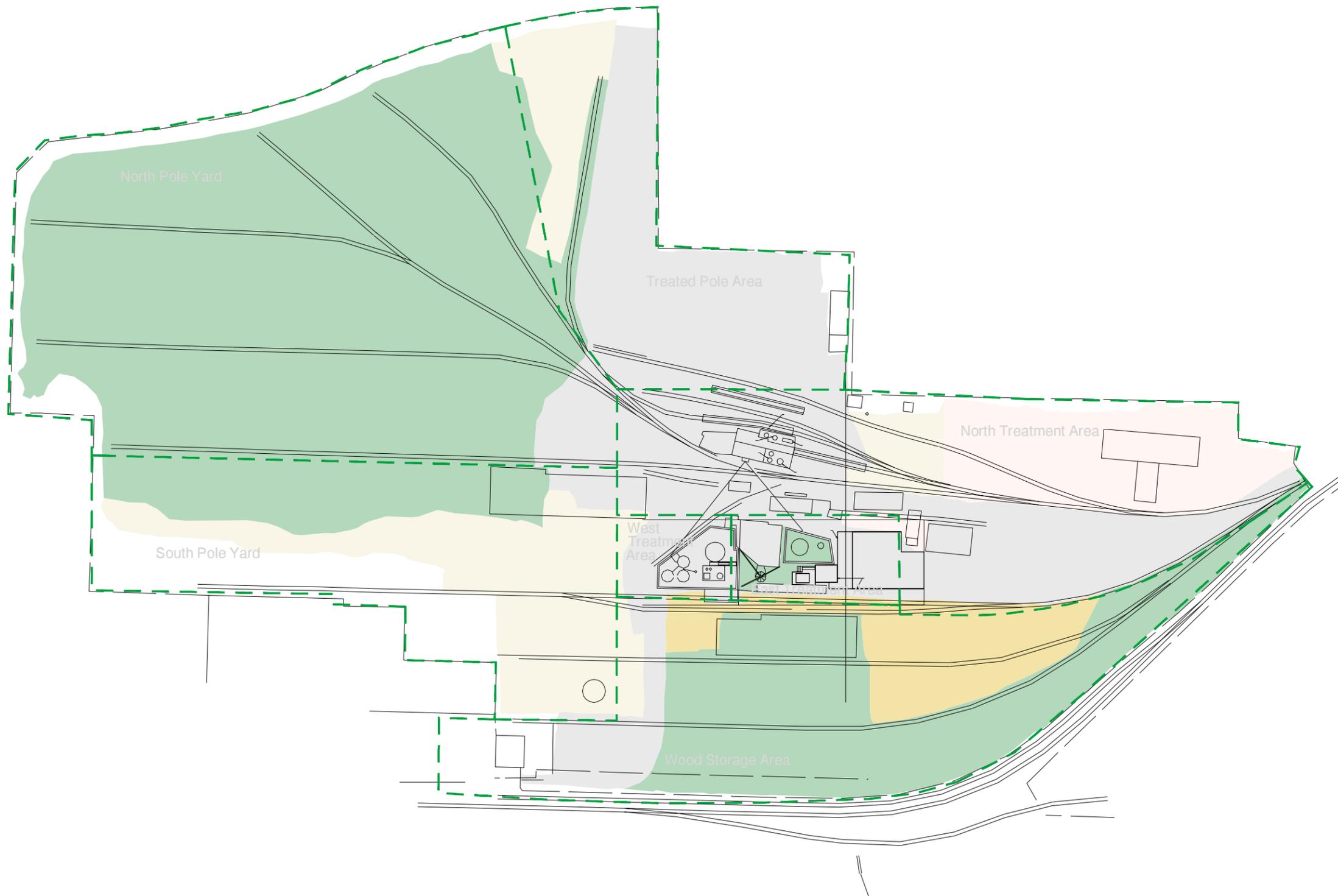
Figure 6-1

THE OESER COMPANY SUPERFUND SITE

Bellingham, Washington

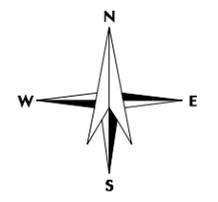
Remedial Investigation

Facility Surface Cover Map
as of July 2000



Legend

- Vegetation
- Asphalt
- Exposed Soil
- Sawdust
- Gravel Cap



MAP SOURCE

Oeser Company Site Map
Larry Steele & Associates
Survey Date: 12/3/1997



ecology and environment, inc.
International Specialists in the Environment
Seattle, Washington

/data1/oeser/rfls/flg6-1.aml

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APPENDIX A
TECHNICAL APPROACH FOR RISK ASSESSMENT TABLES

TABLE 1
 SELECTION OF EXPOSURE PATHWAYS
 THE OESER COMPANY SUPERFUND SITE, BELLINGHAM, WA

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future	Surface Soil	Surface Soil	Facility surface soil	Workers	Adult	Dermal Absorption	On-facility	Quant	Facility is operational and workers have access to unpaved, potentially contaminated soils. EPA removal activities included soil removal and capping of some contaminated surface soils. Lower levels of contamination remain in other areas of the site; therefore, this exposure pathway will be assessed.
						Ingestion	On-facility	Quant	Facility is operational and workers have access to unpaved, potentially contaminated soils. EPA removal activities included soil removal and capping of some contaminated surface soils. Lower levels of contamination remain in other areas of the site; therefore, this exposure pathway will be assessed.
Current/Future	Surface Soil	Surface Soil	Facility surface soil	Trespasser/Visitor	Child/Adult	Dermal Absorption	On-facility	None	Facility is not fully secured from site visitors/trespassers, because access is still possible through the road and railroad tracks. However, this pathway will not be assessed because exposures will be minimal compared to worker exposures.
						Ingestion	On-facility	None	Facility is not fully secured from site visitors/trespassers, because access is still possible through the road and railroad tracks. However, this pathway will not be assessed because exposures will be minimal compared to worker exposures.
Current/Future	Surface Soil	Surface Soil	Surface soil near Little Squalicum Creek	Recreational User	Adolescents	Dermal Absorption	Off-facility	Quant	Contaminated soils have been detected at the location of seeps and suspected spoils piles on the banks of Little Squalicum Creek. Evaluation of an adolescent recreational user should be representative of other receptors (i.e., adults and children).
						Ingestion	Off-facility	Quant	Contaminated soils have been detected at the location of seeps and suspected spoils piles on the banks of Little Squalicum Creek. Evaluation of an adolescent recreational user should be representative of other receptors (i.e., adults and children).
Current/Future	Surface Soil	Plant Tissue	Blackberries growing along Little Squalicum Creek	Recreational User	Adolescents	Ingestion	Off-facility	Qual	Blackberries growing to the south may be impacted by contaminants that have migrated off-facility. This pathway will be assessed if the evaluation of residential vegetable consumption predicts a potential unacceptable risk. Exposures from this exposure pathway would be lower than through residential vegetable consumption.
Current/Future	Surface Soil	Surface Soil	Nearby residences	Resident	Child/Adult	Dermal Absorption Ingestion	Off-facility	Quant	Evaluations of chemicals in on-facility and off-facility air suggest the potential for migration of contaminants to off-facility soil.
							Off-facility	Quant	Evaluations of chemicals in on-facility and off-facility air suggest the potential for migration of contaminants to off-facility soil.
Current/Future	Surface Soil	Plant Tissue	Homegrown produce from nearby residences	Resident	Child/Adult	Ingestion	Off-facility	Quant	Homegrown produce at nearby residences may potentially be impacted by contaminants that have migrated off-facility. This pathway will be assessed through a quantitative screening in the preliminary off-facility risk evaluation and may be carried into the baseline risk assessment.

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Current/Future	Subsurface Soil	Subsurface Soil	Facility subsurface soil	Workers	Adult	Dermal Absorption	On-facility	None	Potential utility maintenance or construction workers may be exposed to contaminants in subsurface soils. This pathway will not be evaluated because exposures would be minimal compared to future excavation scenarios, where excavation and redevelopment results in transport of subsurface soil to surface soil.
						Ingestion	On-facility	None	Potential utility maintenance or construction workers may be exposed to contaminants in subsurface soils. This pathway will not be evaluated because exposures would be minimal compared to future excavation scenarios.
Current/Future	Surface Water	Surface Water	Little Squalicum Creek	Recreational User	Adolescents	Dermal Absorption	Off-facility	Quant	Contaminants from the facility may potentially be migrating to the creek. Contaminants have been detected in surface water from Little Squalicum Creek. Evaluation of an adolescent recreational user should be representative of other receptors (i.e., adults and children).
						Ingestion	Off-facility	None	Creek conditions make chronic incidental ingestion of creek water unlikely.
Current/Future	Sediment	Sediment	Little Squalicum Creek	Recreational User	Adolescents	Dermal Absorption	Off-facility	Quant	Contaminants from the facility may potentially be migrating to the creek. Contaminants have been detected in sediment from Little Squalicum Creek. Evaluation of an adolescent recreational user should be representative of other receptors (i.e., adults and children).
						Ingestion	Off-facility	None	Creek conditions make chronic incidental ingestion of creek sediment unlikely.
Current/Future	Surface Water	Animal Tissue	Little Squalicum Creek	Fisher	Child/Adult	Ingestion	Off-facility	None	No information has been found to indicate that Little Squalicum Creek currently, or in the past, has supported a fish/shellfish population.
Current/Future	Sediment	Animal Tissue	Little Squalicum Creek	Fisher	Child/Adult	Ingestion	Off-facility	None	No information has been found to indicate that Little Squalicum Creek currently, or in the past, has supported a fish/shellfish population.
Current/Future	Groundwater	Shallow Groundwater	Tap water from on-facility shallow saturated zones	Workers	Adult	Ingestion	On-facility	None	Contaminants have been detected in shallow saturated zones; however, no seeps have been observed on-facility. Use of water from the shallow saturated zones is very unlikely and will not be evaluated; however, shallow groundwater may be impacting Little Squalicum Creek and/or deep groundwater. These pathways will be quantified and are identified below.
						Dermal Absorption	On-facility	None	Contaminants have been detected in shallow saturated zones; however, no seeps have been observed on-facility. Use of water from the shallow saturated zones is very unlikely and will not be evaluated; however, shallow groundwater may be impacting Little Squalicum Creek and/or deep groundwater. These pathways will be quantified and are identified below.

TABLE 1
SELECTION OF EXPOSURE PATHWAYS
THE OESER COMPANY SUPERFUND SITE, BELLINGHAM, WA

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future	Groundwater	Shallow Groundwater	Tap water from off-facility shallow saturated zones	Resident	Child/Adult	Ingestion	Off-facility	None	Water from the shallow saturated zone is not used for any purposes on- or off facility and will not be evaluated; however, shallow groundwater may be impacting Little Squalicum Creek and/or deep groundwater. These pathways will be quantified and are identified below.
						Dermal Absorption	Off-facility	None	Water from the shallow saturated zone is not used for any purposes on- or off facility and will not be evaluated; however, shallow groundwater may be impacting Little Squalicum Creek and/or deep groundwater. These pathways will be quantified and are identified below.
						Inhalation	Off-facility	None	Water from the shallow saturated zone is not used for any purposes on- or off facility and will not be evaluated; however, shallow groundwater may be impacting Little Squalicum Creek and/or deep groundwater. These pathways will be quantified and are identified below.
Current/Future	Groundwater	Shallow or deep groundwater	Little Squalicum Creek	Recreational User	Adolescents	Ingestion	Off-facility	None	Chronic contact with seepage water is unlikely. Contact with associated soils and sediments is much more likely and is identified in the previous pathways.
						Dermal Absorption	Off-facility	None	Chronic contact with seepage water is unlikely. Contact with associated soils and sediments is much more likely and is identified in the previous pathways.
Current	Groundwater	Deep groundwater	Deep aquifer - tap water	Worker	Adult	Ingestion	On-facility	None	Groundwater is not currently used for any purposes on-facility.
Current	Groundwater	Deep groundwater	Deep aquifer - tap water	Resident	Child/Adult	Ingestion	Off-facility	None	Contaminants have been detected in the deep aquifer beneath the facility; however contaminants have not been detected in wells off-facility. No known wells in the area serve as potable water supplies.
						Dermal Absorption	Off-facility	None	Contaminants have been detected in the deep aquifer beneath the facility; however contaminants have not been detected in wells off-facility. No known wells in the area serve as potable water supplies.
Current/Future	Air	Particulates and vapors	Breathing zone air on-facility	Worker	Adult	Inhalation	On-facility	Quant	Contaminants have been detected in on-facility air. On-facility capping has reduced but not eliminated this pathway.
Current/Future	Air	Particulates and vapors	Breathing zone air off-facility	Resident	Child/Adult	Inhalation	Off-facility	Quant	Contaminants have been detected in off-facility air. On-facility capping has reduced but not eliminated this pathway.
Current/Future	Air	Particulates and vapors	Breathing zone air off-facility	Recreational User	Adolescents	Inhalation	Off-facility	Quant	Contaminants have been detected in off-facility air. On-facility capping has reduced but not eliminated this pathway. Evaluation of an adolescent recreational user should be representative of other receptors (i.e., adults and children).
Current/Future	Air	Particulates and vapors	Breathing zone air on-facility	Trespasser/Visitor	Child/Adult	Inhalation	On-facility	None	Facility is not fully secured from site visitors/trespassers, because access is still possible through the road and railroad tracks. However, this pathway will not be assessed because exposures will be minimal compared to worker exposures.
Future	Surface Soil	Surface Soil	Facility surface soil	Resident	Child/Adult	Dermal Absorption	On-facility	Quant	The main portion of the facility is currently zoned heavy impact industrial, but the northeast portion of the site is zoned residential and residences are located adjacent to the site. Future land uses for all areas of the site are uncertain.
						Ingestion	On-facility	Quant	The main portion of the facility is currently zoned heavy impact industrial, but the northeast portion of the site is zoned residential and residences are located adjacent to the site. Future land uses for all areas of the site are uncertain.
Future	Subsurface Soil	Subsurface Soil	Facility subsurface soil	Workers	Adult	Dermal Absorption	On-facility	Quant	Future excavation/development could result in subsurface soils being exposed at the surface where they would be available for direct contact exposures.
						Ingestion	On-facility	Quant	Future excavation/development could result in subsurface soils being exposed at the surface where they would be available for direct contact exposures.

TABLE 1
 SELECTION OF EXPOSURE PATHWAYS
 THE OESER COMPANY SUPERFUND SITE, BELLINGHAM, WA

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future	Subsurface Soil	Subsurface Soil	Facility subsurface soil	Trespasser/Visitor	Child/Adult	Dermal Absorption	On-facility	None	Facility is not fully secured from site visitors/trespassers, because access is still possible through the road and railroad tracks. However, this pathway will not be assessed because exposures will be minimal compared to worker exposures.
						Ingestion	On-facility	None	Facility is not fully secured from site visitors/trespassers, because access is still possible through the road and railroad tracks. However, this pathway will not be assessed because exposures will be minimal compared to worker exposures.
Future	Subsurface Soil	Subsurface Soil	Facility subsurface soil	Resident	Child/Adult	Dermal Absorption Ingestion	On-facility	Quant	Subsurface soils may be exposed if future residential development occurs.
Future	Groundwater	Deep groundwater	Deep aquifer - tap water	Worker	Adult	Ingestion	On-facility	Quant	On-facility/Tilbury Cement Co groundwater could be used as potable water in the future.
						Dermal Absorption	On-facility	Quant	On-facility/Tilbury Cement Co groundwater could be used as potable water in the future.
Future	Groundwater	Deep groundwater	Deep aquifer - tap water	Resident	Child/Adult	Ingestion	On-facility	Quant	Water from the deep aquifer could be used as a residential water supply if the facility is developed in the future.
						Dermal Absorption	On-facility	Quant	Water from the deep aquifer could be used as a residential water supply if the facility is developed in the future.
						Inhalation	On-facility	Quant	Water from the deep aquifer could be used as a residential water supply if the facility is developed in the future.
Future	Air	Particulates and vapors	Breathing zone air on-facility	Resident	Child/Adult	Inhalation	On-facility	Quant	If the facility is developed for residential use in the future, residents could potentially be exposed to airborne surface and subsurface soil contaminants through volatilization or through blown dust.

TABLE 4.1
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Tap
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Ingestion	CW	Concentration in Groundwater	µg/L	TBD	--	TBD	--	Chronic Daily Intake (CDI) (mg/kg-day) $CW \times IR-W \times EF \times ED \times CF1 \times 1/BW \times 1/AT$
	IR-W	Ingestion Rate of Water	L/day	2	EPA 1989c	1.4	EPA 1993	
	EF	Exposure Frequency	days/year	350	EPA 1991b	234	EPA 1993	
	ED	Exposure Duration	years	24	EPA 1991b	9	EPA 1993	
	CF1	Conversion Factor 1	mg/µg	0.001	--	0.001	--	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	8,760	EPA 1989c	3,285	EPA 1993	
Dermal	CW	Concentration in Groundwater	µg/L	TBD	--	TBD	--	$CDI \text{ (mg/kg-day)} =$ if $ED < t^*$, then $DA_e = 2FA \times K \times C_w \times [(6\tau \times ED)/\pi]^{1/2} \times 0.001 \times 0.001$ if $ED > t^*$, then $DA_e = FA \times K \times C_w \times [ED/(1 + B) + 2\tau[(1 + 3B + B^2)/(1 + B)^2]] \times 0.001 \times 0.001$ $DAD = DA_e \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$
	SA	Skin surface area exposed	cm ²	18,000	EPA 2000	18,000	EPA 2000	
	K	Permeability constant	cm/hour	Chemical-specific	EPA RAGs E	Chemical-specific	EPA RAGs E	
	EF	Exposure Frequency	days/year	350	EPA 1991b	350	EPA 1993	
	ED	Exposure Duration	years	24	EPA 1991b	9	EPA 1993	
	ET	Exposure time	hours/day	0.25	EPA 1998e	0.17	EPA 1998e	
	DA _e	Absorbed Dose per Event	mg/cm ² -event	TBD	EPA RAGs E	TBD	EPA RAGs E	
	FA	Fraction Absorbed	-	Chemical-specific	EPA RAGs E	Chemical-specific	EPA RAGs E	
	tau	Lag Time per Event	hr/event	Chemical-specific	EPA RAGs E	Chemical-specific	EPA RAGs E	
	t*	Time to reach steady-state	hr	Chemical-specific	EPA RAGs E	Chemical-specific	EPA RAGs E	
	B	Ratio of permeability coefficient	-	Chemical-specific	EPA RAGs E	Chemical-specific	EPA RAGs E	
	DAD	Dermally Absorbed Dose	mg/kg-day	TBD	EPA RAGs E	TBD	EPA RAGs E	
	CF1	Conversion Factor 1	mg/µg	0.001	--	0.001	--	
	CF2	Conversion Factor 2	L/cm ³	0.001	--	0.001	--	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	8,760	EPA 1989c	3,285	EPA 1993	

TABLE 4.2
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Tap
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Ingestion	CW	Concentration in Groundwater	µg/L	TBD	--	--	--	Chronic Daily Intake (CDI) (mg/kg-day) $CW \times IR-W \times EF \times ED \times CF1 \times 1/BW \times 1/AT$
	IR-W	Ingestion Rate of Water	L/day	1	Cal/EPA 1994	--	--	
	EF	Exposure Frequency	days/year	350	EPA 1991b	--	--	
	ED	Exposure Duration	years	6	EPA 1991b	--	--	
	CF1	Conversion Factor 1	mg/µg	0.001	--	--	--	
	BW	Body weight	kg	15	EPA 1989c	--	--	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	--	--	
	AT-N	Averaging Time (Non-Cancer)	days	2,190	EPA 1989c	--	--	
Dermal	CW	Concentration in Groundwater	µg/L	TBD	--	--	--	CDI (mg/kg-day) = if $ED < t^*$, then $DA_e = 2FA \times K \times C_w \times [(6\tau \times ED)/\pi]^{1/2} \times 0.001 \times 0.001$ if $ED > t^*$, then $DA_e = FA \times K \times C_w \times [ED/(1 + B) + 2\tau[(1 + 3B + B^2)/(1 + B)^2] \times 0.001 \times 0.001$ $DAD = DA_e \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$
	SA	Skin surface area exposed	cm ²	6,500	EPA 2000	--	--	
	K	Permeability constant	cm/hour	Chemical-specific	EPA RAGs E	--	--	
	EF	Exposure Frequency	days/year	350	EPA 1991b	--	--	
	ED	Exposure Duration	years	6	EPA 1991b	--	--	
	ET	Exposure Time	hours/day	0.25	EPA 1998e	--	--	
	DA _e	Absorbed Dose per Event	mg/cm ² -event	TBD	EPA RAGs E	--	--	
	FA	Fraction Absorbed	-	Chemical-specific	EPA RAGs E	--	--	
	tau	Lag Time per Event	hr/event	Chemical-specific	EPA RAGs E	--	--	
	t*	Time to reach steady-state	hr	Chemical-specific	EPA RAGs E	--	--	
	B	Ratio of permeability coefficient	-	Chemical-specific	EPA RAGs E	--	--	
	DAD	Dermally Absorbed Dose	mg/kg-day	TBD	EPA RAGs E	--	--	
	CF1	Conversion Factor 1	mg/µg	0.001	--	--	--	
	CF2	Conversion Factor 2	L/cm ³	0.001	--	--	--	
	BW	Body weight	kg	15	EPA 1989c	--	--	
AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	--	--		
AT-N	Averaging Time (Non-Cancer)	days	2,190	EPA 1989c	--	--		

TABLE 4.3
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Future
Medium: Indoor Air
Exposure Medium: Groundwater
Exposure Point: Volatiles in Tap Water
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Inhalation	CA	Chemical concentration in air	mg/m ³	TBD	--	TBD	--	$CDI (mg/kg-day) = CA \times IR-A \times EF \times ED \times 1/BW \times 1/AT$
	CF1	Conversion Factor 1	mg/ug	0.001				
	K	Volatility Factor	L/m3	0.5	EPA 1991b			
	IR-A	Inhalation Rate of Air	m3/day	20	EPA 1991b	20	EPA 1991b	
	EF	Exposure Frequency	days/year	350	EPA 1991b	234	EPA 1993	
	ED	Exposure Duration	years	24	EPA 1991b	9	EPA 1993	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	8,760	EPA 1989c	3,285	EPA 1993	

TABLE 4.4
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Future
Medium: Indoor Air
Exposure Medium: Groundwater
Exposure Point: Volatiles in Tap Water
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Inhalation	CA	Chemical concentration in air	mg/m ³	TBD	--	--	--	$CDI (mg/kg-day) = CA \times IR-A \times EF \times ED \times 1/BW \times 1/AT$
	CF1	Conversion Factor 1	mg/ug	0.001				
	K	Volatility Factor	L/m3	0.5	EPA 1991b			
	IR-A	Inhalation Rate of Air	m3/day	10	EPA 1989c	--	--	
	EF	Exposure Frequency	days/year	350	EPA 1991b	--	--	
	ED	Exposure Duration	years	6	EPA 1991b	--	--	
	BW	Body weight	kg	15	EPA 1989c	--	--	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	--	--	
	AT-N	Averaging Time (Non-Cancer)	days	2,190	EPA 1989c	--	--	

TABLE 4.5
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Current
Medium: Soil
Exposure Medium: Soil
Exposure Point: Residential Soil
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Ingestion	CS	Concentration in Soil	mg/kg	TBD	--	TBD	--	Chronic Daily Intake (CDI) (mg/kg-day) CS x IR-S x EF x ED x CF1 x 1/BW x 1/AT
	IR-S	Ingestion Rate of Soil	mg/day	100	EPA 1991b	50	EPA 1993	
	EF	Exposure Frequency	days/year	350	EPA 1991b	234	EPA 1993	
	ED	Exposure Duration	years	24	EPA 1991b	9	EPA 1993	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	0.000001	--	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	8,760	EPA 1989c	3,285	EPA 1993	
Dermal	CS	Concentration in Soil	mg/kg	TBD	--	TBD	--	DAD (Dermal Absorbed Dose) (mg/kg-day) = CS x CF1 x AF x ABS x SA x EV x EF x ED x 1/BW x 1/AT
	SA	Skin surface area exposed	cm ² /day	2,500	EPA 2000	2,500	EPA 2000	
	AF	Adherence factor	mg/cm ²	0.1	EPA 2000	0.1	EPA 2000	
	ABS	Absorption factor	--	Chemical-specific	EPA 2000	Chemical-specific	EPA 2000	
	EV	Event Frequency	events/day	1	EPA RAGs E	1	EPA RAGs E	
	EF	Exposure Frequency	days/year	350	EPA 1991b	234	EPA 1993	
	ED	Exposure Duration	years	24	EPA 1991b	9	EPA 1993	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	0.000001	--	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	8,760	EPA 1989c	3,285	EPA 1993	

TABLE 4.6
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Current
Medium: Soil
Exposure Medium: Soil
Exposure Point: Residential Soil
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Ingestion	CS	Concentration in Soil	mg/kg	TBD	--	--	--	Chronic Daily Intake (CDI) (mg/kg-day) CS x IR-S x EF x ED x CF1 x 1/BW x 1/AT
	IR-S	Ingestion Rate of Soil	mg/day	200	EPA 1991b	--	--	
	EF	Exposure Frequency	days/year	350	EPA 1991b	--	--	
	ED	Exposure Duration	years	6	EPA 1991b	--	--	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	--	--	
	BW	Body weight	kg	15	EPA 1989c	--	--	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	--	--	
	AT-N	Averaging Time (Non-Cancer)	days	2,190	EPA 1989c	--	--	
Dermal	CS	Concentration in Soil	mg/kg	TBD	--	--	--	DAD (Dermal Absorbed Dose) (mg/kg-day) = CS x CF1 x AF x ABS x SA x EV x EF x ED x 1/BW x 1/AT
	SA	Skin surface area exposed	cm ² /day	2,200	EPA 2000	--	--	
	AF	Adherence factor	mg/cm ²	0.2	EPA 2000	--	--	
	ABS	Absorption factor	--	Chemical-specific	EPA 2000	--	--	
	EV	Event Frequency	events/day	1	EPA RAGs E	--	--	
	EF	Exposure Frequency	days/year	350	EPA 1991b	--	--	
	ED	Exposure Duration	years	6	EPA 1991b	--	--	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	--	--	
	BW	Body weight	kg	15	EPA 1989c	--	--	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	--	--	
	AT-N	Averaging Time (Non-Cancer)	days	2,190	EPA 1989c	--	--	

TABLE 4.7
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Current Medium: Outdoor Air Exposure Medium: Outdoor Air Exposure Point: Outdoor Air Receptor Population: Resident Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Inhalation	CA	Chemical concentration in air	mg/m ³	TBD	--	TBD	--	CDI (mg/kg-day) = CA x IR-A x EF x ED x 1/BW x 1/AT
	IR-A	Inhalation Rate of Air	m ³ /day	20	EPA 1991b	20	EPA 1991b	
	EF	Exposure Frequency	days/year	350	EPA 1991b	234	EPA 1993	
	ED	Exposure Duration	years	24	EPA 1991b	9	EPA 1993	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	8,760	EPA 1989c	3,285	EPA 1993	

TABLE 4.8
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Current
Medium: Outdoor Air
Exposure Medium: Outdoor Air
Exposure Point: Outdoor Air
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Inhalation	CA	Chemical concentration in air	mg/m ³	TBD	--	--	--	CDI (mg/kg-day) = CA x IR-A x EF x ED x 1/BW x 1/AT
	IR-A	Inhalation Rate of Air	m3/day	10	EPA 1989c	--	--	
	EF	Exposure Frequency	days/year	350	EPA 1991b	--	--	
	ED	Exposure Duration	years	6	EPA 1991b	--	--	
	BW	Body weight	kg	15	EPA 1989c	--	--	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	--	--	
	AT-N	Averaging Time (Non-Cancer)	days	2,190	EPA 1989c	--	--	

TABLE 4.9
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Soil
Exposure Point: Residential soil
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Ingestion	CS	Concentration in Soil	mg/kg	TBD	--	TBD	--	Chronic Daily Intake (CDI) (mg/kg-day) $CS \times IR-S \times EF \times ED \times CF1 \times 1/BW \times 1/AT$
	IR-S	Ingestion Rate of Soil	mg/day	100	EPA 1991b	50	EPA 1993	
	EF	Exposure Frequency	days/year	350	EPA 1991b	234	EPA 1993	
	ED	Exposure Duration	years	24	EPA 1991b	9	EPA 1993	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	0.000001	--	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
Dermal	CS	Concentration in Soil	mg/kg	TBD	--	TBD	--	DAD (Dermal Absorbed Dose) (mg/kg-day) = $CS \times CF1 \times AF \times ABS \times SA \times EV \times EF \times ED \times 1/BW \times 1/AT$
	SA	Skin surface area exposed	cm ² /day	2,500	EPA 2000	2,500	EPA 2000	
	AF	Adherence factor	mg/cm ²	0.1	EPA 2000	0.1	EPA 2000	
	ABS	Absorption factor	--	Chemical-specific	EPA 2000	Chemical-specific	EPA 2000	
	EV	Event Frequency	events/day	1	EPA RAGs E	1	EPA RAGs E	
	EF	Exposure Frequency	days/year	350	EPA 1991b	234	EPA 1993	
	ED	Exposure Duration	years	24	EPA 1991b	9	EPA 1993	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	0.000001	--	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
Inhalation	CS	Chemical concentration in soil	mg/kg	TBD	--	TBD	--	CDI (mg/kg-day) = $CS \times [1/VF + 1/PEF] \times IR-A \times EF \times ED \times 1/BW \times 1/AT$
	PEF	Particulate emission factor	m ³ /kg	2.1 x 10 ⁹	EPA 1996b	2.1 x 10 ⁹	EPA 1996b	
	VF	Volatilization factor	m ³ /kg	Chemical-specific	(1)	Chemical-specific	(1)	
	IR-A	Inhalation Rate of Air	m ³ /day	20	EPA 1991b	20	EPA 1991b	
	EF	Exposure Frequency	days/year	350	EPA 1991b	234	EPA 1993	
	ED	Exposure Duration	years	24	EPA 1991b	9	EPA 1993	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	8,760	EPA 1989c	3,285	EPA 1993	

(1) Chemical-specific volatilization factors are presented in EPA 1998a.

TABLE 4.10
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Future Medium: Soil Exposure Medium: Soil Exposure Point: Residential Soil Receptor Population: Resident Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Ingestion	CS	Concentration in Soil	mg/kg	TBD	--	--	--	Chronic Daily Intake (CDI) (mg/kg-day) $CS \times IR-S \times EF \times ED \times CF1 \times 1/BW \times 1/AT$
	IR-S	Ingestion Rate of Soil	mg/day	200	EPA 1991b	--	--	
	EF	Exposure Frequency	days/year	350	EPA 1991b	--	--	
	ED	Exposure Duration	years	6	EPA 1991b	--	--	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	--	--	
	BW	Body weight	kg	15	EPA 1989c	--	--	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	--	--	
	AT-N	Averaging Time (Non-Cancer)	days	2,190	EPA 1989c	--	--	
Dermal	CS	Concentration in Soil	mg/kg	TBD	--	--	--	DAD (Dermal Absorbed Dose) (mg/kg-day) = $CS \times CF1 \times AF \times ABS \times SA \times EV \times EF \times ED \times 1/BW \times 1/AT$
	SA	Skin surface area exposed	cm ² /day	2,200	EPA 2000	--	--	
	AF	Adherence factor	mg/cm ²	0.2	EPA 2000	--	--	
	ABS	Absorption factor	--	Chemical-specific	EPA 2000	--	--	
	EV	Event Frequency	events/day	1	EPA RAGs E	--	--	
	EF	Exposure Frequency	days/year	350	EPA 1991b	--	--	
	ED	Exposure Duration	years	6	EPA 1991b	--	--	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	--	--	
	BW	Body weight	kg	15	EPA 1989c	--	--	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	--	--	
AT-N	Averaging Time (Non-Cancer)	days	2,190	EPA 1989c	--	--		
Inhalation	CS	Chemical concentration in soil	mg/kg	TBD	--	--	--	CDI (mg/kg-day) = $CS \times [1/VF + 1/PEF] \times IR-A \times EF \times ED \times 1/BW \times 1/AT$
	PEF	Particulate emission factor	m ³ /kg	2.1 x 10 ⁹	EPA 1996b	--	--	
	VF	Volatilization factor	m ³ /kg	Chemical-specific	(1)	--	--	
	IR-A	Inhalation Rate of Air	m ³ /day	10	EPA 1989c	--	--	
	EF	Exposure Frequency	days/year	350	EPA 1991b	--	--	
	ED	Exposure Duration	years	6	EPA 1991b	--	--	
	BW	Body weight	kg	15	EPA 1989c	--	--	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	--	--	
	AT-N	Averaging Time (Non-Cancer)	days	2,190	EPA 1989c	--	--	

(1) Chemical-specific volatilization factors are presented in EPA 1998a.

TABLE 4.12
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Current
 Medium: Outdoor Air
 Exposure Medium: Outdoor Air
 Exposure Point: Outdoor Air
 Receptor Population: Recreational Visitor
 Receptor Age: Adolescent (8-18)

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Inhalation	CA	Chemical concentration in air	mg/m ³	TBD	--	TBD	--	CDI (mg/kg-day) = CA x IR-A x EF x ED x FI-A x 1/BW x 1/AT
	IR-A	Inhalation Rate of Air	m ³ /day	20	EPA 1991b	20	EPA 1991b	
	EF	Exposure Frequency	days/year	104	BPJ (1)	26	BPJ (1)	
	ED	Exposure Duration	years	11	BPJ (2)	9	EPA 1993	
	FI-A	Fraction air inhaled	Unitless	0.25	BPJ (3)	0.125	BPJ (3)	
	BW	Body weight	kg	49	EFH 1997	49	EFH 1997	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	4,015	EPA 1989c	3,285	EPA 1993	

- (1) Recreational users were assumed to visit the site 2 days per week for the entire year for the RME scenario; for the average scenario, recreational users were assumed to visit the site for 1 day per week for 6 months out of each year.
- (2) An exposure duration of 11 years was selected to represent the entire duration of the age group for the RME scenario.
- (3) Recreational users were assumed to visit the site for 4 out of 16 waking hours for the RME scenario; for the average scenario, recreational users were assumed to visit the

TABLE 4.13
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Future Medium: Soil Exposure Medium: Soil Exposure Point: Soil Receptor Population: Recreational User Receptor Age: Adolescent (8-18 years old)
--

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Ingestion	CS	Concentration in Soil	mg/kg	TBD	--	TBD	--	Chronic Daily Intake (CDI) (mg/kg-day) $CS \times IR-S \times EF \times ED \times CF1 \times FC-S \times 1/BW \times 1/AT$
	IR-S	Ingestion Rate of Soil	mg/day	100	EPA 1991b	50	EPA 1993	
	EF	Exposure Frequency	days/year	104	BPJ (1)	26	BPJ (1)	
	ED	Exposure Duration	years	11	(2)	9	EPA 1993	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	0.000001	--	
	FC-S	Fraction soil contacted	Unitless	0.25	BPJ (3)	0.125	BPJ (3)	
	BW	Body weight	kg	49	EFH 1997	49	EFH 1997	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	4,015	EPA 1989c	3,285	EPA 1993	
Dermal	CS	Concentration in Soil	mg/kg	TBD	--	TBD	--	DAD (Dermal Absorbed Dose) (mg/kg-day) = $CS \times CF1 \times AF \times ABS \times SA \times EV \times EF \times ED \times FC-S \times 1/BW \times 1/AT$
	SA	Skin surface area exposed	cm ² /day	2,500	EPA 2000	2,500	EPA 2000	
	AF	Adherence factor	mg/cm ²	0.1	EPA 2000	0.1	EPA 2000	
	ABS	Absorption factor	--	Chemical-specific	EPA 2000	Chemical-specific	EPA 2000	
	EV	Event Frequency	events/day	1	EPA RAGs E	1	EPA RAGs E	
	EF	Exposure Frequency	days/year	104	EPA 1991b	26	EPA 1993	
	ED	Exposure Duration	years	11	EPA 1991b	9	EPA 1993	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	0.000001	--	
	FC-S	Fraction soil contacted	Unitless	0.25	BPJ (3)	0.125	BPJ (3)	
	BW	Body weight	kg	49	EPA 1989c	49	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
AT-N	Averaging Time (Non-Cancer)	days	4,015	EPA 1989c	3,285	EPA 1993		
Inhalation	CS	Chemical concentration in soil	mg/kg	TBD	--	TBD	--	CDI (mg/kg-day) = $CS \times [1/VF + 1/PEF] \times IR-A \times EF \times ED \times FI-A \times 1/BW \times 1/AT$
	PEF	Particulate emission factor	m ³ /kg	2.1 x 10 ⁹	EPA 1996b	2.1 x 10 ⁹	EPA 1996b	
	VF	Volatilization factor	m ³ /kg	Chemical-specific	(4)	Chemical-specific	(4)	
	IR-A	Inhalation Rate of Air	m ³ /day	20	EPA 1991b	20	EPA 1991b	
	EF	Exposure Frequency	days/year	104	BPJ (1)	26	BPJ (1)	
	ED	Exposure Duration	years	11	EPA 1991b	9	EPA 1993	
	FI-A	Fraction air inhaled	Unitless	0.25	BPJ (3)	0.125	BPJ (3)	
	BW	Body weight	kg	49	EFH 1997	49	EFH 1997	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	4,015	EPA 1989c	3,285	EPA 1993	

- (1) Recreational users were assumed to visit the site 2 days per week for the entire year for the RME scenario; for the average scenario, recreational users were assumed to visit the site 2 days per week for the entire year.
- (2) An exposure duration of 11 years was selected to represent the entire duration of the age group for the RME scenario.
- (3) Recreational users were assumed to visit the site for 4 out of 16 waking hours for the RME scenario; for the average scenario, recreational users were assumed to visit the site for 2 hours per day.
- (4) Chemical-specific volatilization factors are presented in EPA 1998a.

TABLE 4.14
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Current/Future
Medium: Surface Water
Exposure Medium: Surface Water
Exposure Point: Little Squalicum Creek
Receptor Population: Recreational Visitor
Receptor Age: Adolescent (8-18 years old)

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Dermal	CW	Concentration in Surface Water	µg/L	TBD	--	TBD	--	$CDI \text{ (mg/kg-day)} =$ if $ED < t^*$, then $DA_e = 2FA \times K \times C_w \times [(6\tau \times ED)/\pi]^{1/2} \times 0.001 \times 0.001$ if $ED > t^*$, then $DA_e = FA \times K \times C_w \times [ED/(1 + B) + 2\tau[(1 + 3B + B^2)/(1 + B)^2] \times 0.001 \times 0.001$ $DAD = DA_e \times EV \times ED \times EF \times SA \times 1/BW \times 1/AT$
	SA	Skin surface area exposed	cm ²	2,400	EFH 1997 (1)	2,000	EFH 1997 (1)	
	K	Permeability constant	cm/hour	Chemical-specific	EPA RAGs E	Chemical-specific	EPA RAGs E	
	DA _e	Absorbed Dose per Event	ng/cm ² -event	TBD	EPA RAGs E	TBD	EPA RAGs E	
	FA	Fraction Absorbed	-	Chemical-specific	EPA RAGs E	Chemical-specific	EPA RAGs E	
	tau	Lag Time per Event	hr/event	Chemical-specific	EPA RAGs E	Chemical-specific	EPA RAGs E	
	t*	Time to reach steady-state	hr	Chemical-specific	EPA RAGs E	Chemical-specific	EPA RAGs E	
	B	Ratio of permeability coefficient	-	Chemical-specific	EPA RAGs E	Chemical-specific	EPA RAGs E	
	DAD	Dermally Absorbed Dose	mg/kg-day	TBD	EPA RAGs E	TBD	EPA RAGs E	
	CF1	Conversion Factor 1	mg/µg	0.001	--	0.001	--	
	CF2	Conversion Factor 2	L/cm ³	0.001	--	0.001	--	
	EF	Exposure Frequency	days/year	104	BPJ (3)	26	BPJ (3)	
	ED	Exposure Duration	years	11	BPJ (4)	9	EPA 1993	
	ET	Exposure Time	hours/day	4	BPJ (5)	2	BPJ (5)	
	CF1	Conversion Factor 1	mg/µg	0.001	--	0.001	--	
	CF2	Conversion Factor 2	L/cm ³	0.001	--	0.001	--	
	BW	Body weight	kg	49	EFH 1997	49	EFH 1997	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
AT-N	Averaging Time (Non-Cancer)	days	4,015	EPA 1989c	3,285	EPA 1993		

- (1) The skin surface area for this age group was estimated from the skin surface area reported for boys age 12-13 because they represent the middle of the age group. The percentage of total body surface area was determined for the head and neck (15%), face (15%), and hands (15%). This percentage of skin surface area exposed was multiplied by the 90th percentile and 50th percentile whole body skin surface area for 12-13 year olds for RME and average scenarios, respectively.
- (2) Permeability constants for chemicals are provided in EPA RAGs E..
- (3) Recreational users were assumed to visit the site 2 days per week for the entire year for the RME scenario; for the average scenario, recreational users were assumed to visit the site for 1 day per week for 6 months of the year.
- (4) An exposure duration of 11 years was selected to represent the entire duration of the age group for the RME scenario.
- (5) Recreational users were assumed to visit the site for 4 out of 16 waking hours for the RME scenario; for the average scenario, recreational users were assumed to visit the site for 2 out of 16 waking hours.

TABLE 4.15
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Current/Future
 Medium: Sediment
 Exposure Medium: Sediment
 Exposure Point:
 Receptor Population: Recreational User
 Receptor Age: Adolescent (8-18 years old)

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Dermal	CS	Concentration in Soil	mg/kg	TBD	--	TBD	--	DAD (Dermal Absorbed Dose) (mg/kg-day) = CS x CF1 x AF x ABS x SA x EV x EF x ED x FC-S x 1/BW x 1/AT
	SA	Skin surface area exposed	cm ² /day	2,400	EFH 1997 (1)	2,400	EFH 1997 (1)	
	AF	Adherence factor	mg/cm ²	0.1	EPA 2000	0.1	EPA 2000	
	ABS	Absorption factor	--	Chemical-specific	EPA 2000	Chemical-specific	EPA 2000	
	EV	Event Frequency	events/day	1	EPA RAGs E	1	EPA RAGs E	
	EF	Exposure Frequency	days/year	104	EPA 1991b (2)	26	EPA 1993 (2)	
	ED	Exposure Duration	years	11	EPA 1991b (3)	9	EPA 1993	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	0.000001	--	
	FC-S	Fraction soil contacted	Unitless	0.25	BPJ (4)	0.125	BPJ (4)	
	BW	Body weight	kg	49	EPA 1989c	49	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	4,015	EPA 1989c	3,285	EPA 1993	

(1) The skin surface area for this age group was estimated from the skin surface area reported for boys age 12-13 because they represent the middle of the age group. The percentage of total body surface area was determined for half legs (15%). This percentage of skin surface area exposed was multiplied by the 90th percentile and 50th percentile whole body skin surface area for 12-13 year olds for RME and average scenarios, respectively.

(2) Recreational users were assumed to visit the site 2 days per week for the entire year for the RME scenario; for the average scenario, recreational users were assumed to visit the site for 1 day per week for 6 months out of each year.

(3) An exposure duration of 11 years was selected to represent the entire duration of the age group for the RME scenario.

(4) Recreational users were assumed to wade in Little Squalicum Creek for 4 out of 16 waking hours for the RME scenario; for the average scenario, recreational users were assumed wade in Little Squalicum Creek for 2 out of 16 waking hours.

TABLE 4.16
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Soil
Exposure Point: On-Facility Soil
Receptor Population: Worker
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Ingestion	CS	Concentration in Soil	mg/kg	TBD	--	TBD	--	Chronic Daily Intake (CDI) (mg/kg-day) $CS \times IR-S \times EF \times ED \times CF1 \times 1/BW \times 1/AT$
	IR-S	Ingestion Rate of Soil	mg/day	50	EPA 1991b	50	EPA 1991b	
	EF	Exposure Frequency	days/year	250	EPA 1991b	219	EPA 1993e	
	ED	Exposure Duration	years	25	EPA 1991b	6	EPA 1997	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	0.000001	--	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	9,125	EPA 1989c	2,190	EPA 1989c	
Dermal	CS	Concentration in Soil	mg/kg	TBD	--	TBD	--	DAD (Dermal Absorbed Dose) (mg/kg-day) = $CS \times CF1 \times AF \times ABS \times SA \times EV \times EF \times ED \times FC-S \times 1/BW \times 1/AT$
	SA	Skin surface area exposed	cm ² /day	2,500	EPA 2000	2,500	EPA 2000	
	AF	Adherence factor	mg/cm ²	0.2	EPA 2000	0.1	EPA 2000	
	ABS	Absorption factor	--	Chemical-specific	EPA 2000	Chemical-specific	EPA 2000	
	EV	Event Frequency	events/day	1	EPA RAGs E	1	EPA RAGs E	
	EF	Exposure Frequency	days/year	250	EPA 1991b	219	EPA 1993	
	ED	Exposure Duration	years	25	EPA 1991b	6	EPA 1993	
	FI-S	Fraction of soil contacted	Unitless	0.5	--	0.5	--	
	CF1	Conversion Factor 1	kg/mg	0.000001	--	0.000001	--	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	9,125	EPA 1989c	2,190	EPA 1993	
Inhalation	CS	Chemical concentration in soil	mg/kg	TBD	--	TBD	--	CDI (mg/kg-day) = $CS \times [1/VF + 1/PEF] \times IR-A \times EF \times ED \times FI-A \times 1/BW \times 1/AT$
	PEF	Particulate emission factor	m ³ /kg	2.1 x 10 ⁹	EPA 1996b	2.1 x 10 ⁹	EPA 1996b	
	VF	Volatilization factor	m ³ /kg	Chemical-specific	(1)	Chemical-specific	(1)	
	IR-A	Inhalation Rate of Air	m ³ /day	20	EPA 1991b	20	EPA 1991b	
	EF	Exposure Frequency	days/year	250	EPA 1991b	219	EPA 1993	
	ED	Exposure Duration	years	25	EPA 1991b	6	EPA 1997	
	FI-A	Fraction air inhaled	Unitless	0.5	--	0.5	--	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	9,125	EPA 1989c	2,190	EPA 1989c	

(1) Chemical-specific volatilization factors are presented in EPA 1998a.

TABLE 4.17
VALUES USED FOR DAILY INTAKE CALCULATIONS
Oeser Company
Bellingham, Washington

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Tap
Receptor Population: Worker
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CT Value	CT Rationale/Reference	Intake Equation/Model Name
Ingestion	CW	Concentration in Groundwater	µg/L	TBD	--	TBD	--	Chronic Daily Intake (CDI) (mg/kg-day) CW x IR-W x EF x ED x FI-W x CF1 x 1/BW x 1/AT
	IR-W	Ingestion Rate of Water	L/day	2	EPA 1989c	1.4	EPA 1993	
	EF	Exposure Frequency	days/year	250	EPA 1991b	219	EPA 1993	
	ED	Exposure Duration	years	25	EPA 1991b	6	EPA 1997	
	FI-W	Fraction Water Ingested	Unitless	0.5	BPJ	0.25	BPJ	
	CF1	Conversion Factor 1	mg/µg	0.001	--	0.001	--	
	BW	Body weight	kg	70	EPA 1989c	70	EPA 1989c	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989c	25,550	EPA 1989c	
	AT-N	Averaging Time (Non-Cancer)	days	9,125	EPA 1989c	2,190	EPA 1989c	

TABLE 5.1
NON-CANCER TOXICITY DATA -- ORAL/DERMAL ROUTES
Oeser Company
Bellingham, Washington

Chemical of Potential Concern	Chronic/Subchronic	Oral RfD Value	Oral RfD Units	Oral-to-Dermal Adjustment Factor (1)	Adjusted Dermal RfD (2)	Units	Primary Target Organ	Combined Uncertainty/Modifying Factors	Sources of RfD: Target Organ	Dates of RfD: Target Organ (3) (MM/DD/YY)
Acenaphthene	Chronic	6.0E-02	mg/kg-day	89%	5.34E-02	mg/kg-day	Liver	3000	IRIS	10/10/00
Benzene	Chronic	1.0E-03	mg/kg-day	100%	1.00E-03	mg/kg-day	Bone Marrow	1000	NCEA	09/01/98
Benzidine	Chronic	3.0E-03	mg/kg-day	100%	3.00E-03	mg/kg-day	Brain, liver	1000	IRIS	10/10/00
Benzo(a)anthracene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzo(a)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzo(b)fluoranthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzo(j)fluoranthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzo(k)fluoranthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
sec-Butylbenzene	Chronic	1.0E-02	mg/kg-day	100%	1.00E-02	N/A	N/A	N/A	NCEA	N/A
Carbazole	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Chrysene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,e)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,h)acridine	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,h)anthracene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,h)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,i)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,j)acridine	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,l)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
7H-Dibenzo(c,g)carbazole	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzofuran	Chronic	4.0E-03	mg/kg-day	100%	4.00E-03	mg/kg-day	N/A	N/A	Withdrawn	N/A
7,12-Dimethylbenz(a)anthracene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Fluoranthene	Chronic	4.00E-02	mg/kg-day	89%	3.56E-02	mg/kg-day	Kidney, liver, blood	3000	IRIS	10/10/00
Fluorene	Chronic	4.00E-02	mg/kg-day	89%	3.56E-02	mg/kg-day	Blood	3000	IRIS	10/10/00
Indeno(1,2,3-cd)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
2-Methylnaphthalene	Chronic	2.0E-02	mg/kg-day	89%	1.78E-02	mg/kg-day	Whole body	3000	IRIS (4)	10/10/00
Naphthalene	Chronic	2.0E-02	mg/kg-day	89%	1.78E-02	mg/kg-day	Whole body	3000	IRIS	10/10/00
Pentachlorophenol	Chronic	3.0E-02	mg/kg-day	76%	2.28E-02	mg/kg-day	Liver, kidney	100	IRIS	10/10/00
Phenanthrene	Chronic	3.0E-01	mg/kg-day	89%	2.67E-01	mg/kg-day	None	3000	IRIS (4)	10/10/00
n-Propylbenzene	Chronic	1.0E-02	mg/kg-day	100%	1.00E-02	mg/kg-day	N/A	N/A	NCEA	N/A
Pyrene	Chronic	3.0E-02	mg/kg-day	89%	2.67E-02	mg/kg-day	Kidney	3000	IRIS	10/10/00
1,2,4-Trimethylbenzene	Chronic	5.0E-02	mg/kg-day	100%	5.00E-02	mg/kg-day	N/A	N/A	NCEA	N/A
1,3,5-Trimethylbenzene	Chronic	5.0E-02	mg/kg-day	100%	5.00E-02	mg/kg-day	N/A	N/A	NCEA	N/A
PCDDs/PCDFs	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

IRIS = Integrated Risk Information System.

HEAST= Health Effects Assessment Summary Tables.

RfD = Reference dose

PCDDs/PCDFs = Polychlorinated dibenzodioxins/furans

N/A = Not Applicable

NCEA= National Center Environmental Assessment

(1) For adjustment from administered to absorbed dose. Refer to RAGS, Part E.

(2) Equation used for derivation: Dermal RfD = Oral RfD x Oral-to-Dermal Adjustment Factor

(3) For IRIS values, the date IRIS was searched.

For HEAST values, the date of HEAST publication.

(4) Naphthalene RfD used as surrogate for 2-methylnaphthalene.

Anthracene RfD used as surrogate for phenanthrene.

TABLE 5.2
NON-CANCER TOXICITY DATA -- INHALATION ROUTE
Oeser Company
Bellingham, Washington

Chemical of Potential Concern	Chronic/ Subchronic	Value Inhalation RfC	Units	Adjusted Inhalation RfD (1)	Units	Primary Target Organ	Combined Uncertainty/Modifying Factors	Sources of RfC:RfD: Target Organ	Dates (2) (MM/DD/YY)
Acenaphthene	Chronic	N/A	mg/m ³	6.0E-02	mg/kg-day	N/A	N/A	Route extrapolation	N/A
Benzene	Chronic	9.0E-03	mg/m ³	2.6E-03	mg/kg-day	Bone Marrow	1000	NCEA	09/01/98
Benzidine	Chronic	N/A	N/A	3.0E-03	mg/kg-day	N/A	N/A	Route extrapolation	N/A
Benzo(a)anthracene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzo(a)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzo(b)fluoranthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzo(j)fluoranthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzo(k)fluoranthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
sec-Butylbenzene	Chronic	N/A	mg/m ³	1.0E-02	mg/kg-day	N/A	N/A	Route extrapolation	N/A
Carbazole	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Chrysene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,e)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,h)acridine	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,h)anthracene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,h)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,i)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,j)acridine	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzo(a,l)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
7H-Dibenzo(c,g)carbazole	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Dibenzofuran	Chronic	N/A	mg/m ³	4.0E-03	mg/kg-day	N/A	N/A	Route extrapolation	N/A
7,12-Dimethylbenz(a)anthracene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Fluoranthene	Chronic	N/A	mg/m ³	4.0E-02	mg/kg-day	N/A	N/A	Route extrapolation	N/A
Fluorene	Chronic	N/A	mg/m ³	4.0E-02	mg/kg-day	N/A	N/A	Route extrapolation	N/A
Indeno(1,2,3-cd)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
2-Methylnaphthalene	Chronic	3.0E-03	mg/m ³	8.6E-04	mg/kg-day	Respiratory tract	3000	IRIS (3)	10/10/00
Naphthalene	Chronic	3.0E-03	mg/m ³	8.6E-04	mg/kg-day	Respiratory	3000	IRIS	10/10/00
Pentachlorophenol	Chronic	N/A	mg/m ³	3.0E-02	mg/kg-day	N/A	N/A	Route extrapolation	N/A
Phenanthrene	Chronic	N/A	N/A	3.0E-01	mg/kg-day	N/A	N/A	Route extrapolation (3)	10/10/00
n-Propylbenzene	Chronic	N/A	mg/m ³	1.0E-02	mg/kg-day	N/A	N/A	Route extrapolation	N/A
Pyrene	Chronic	N/A	mg/m ³	3.0E-02	mg/kg-day	N/A	N/A	Route extrapolation	N/A
1,2,4-Trimethylbenzene	N/A	N/A	N/A	1.7E-03	mg/kg-day	N/A	N/A	NCEA	N/A
1,3,5-Trimethylbenzene	N/A	N/A	N/A	1.7E-03	mg/kg-day	N/A	N/A	NCEA	N/A
PCDDs/PCDFs	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

IRIS = Integrated Risk Information System
HEAST= Health Effects Assessment Summary Tables
RfC = Reference concentration
RfD= Reference dose
PCDDs/PCDFs = Polychlorinated dibenzodioxins/furans
N/A = Not Applicable
NCEA= National Center Environmental Assessment

- Equation used for derivation: $\text{Inhalation RfD} = \text{RfC} \times (20 \text{ m}^3/\text{day}) / 70 \text{ kg}$.
- For IRIS values, the date IRIS was searched.
For HEAST values, the date of HEAST publication.
For NCEA values, the date of the issue paper.
- Naphthalene RfD used as surrogate for 2-methylnaphthalene.
Anthracene RfD used as surrogate for phenanthrene.

TABLE 6.1
 CANCER TOXICITY DATA -- ORAL/DERMAL ROUTES
 Oeser Company
 Bellingham, Washington

Chemical of Potential Concern	Oral Cancer Slope Factor SFo	Oral-to-Dermal Adjustment Factor (1)	Adjusted Dermal Cancer Slope Factor (2) SFd	Units	Weight of Evidence Group	Source	Date (3) (MM/DD/YY)
Acenaphthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzene	5.5E-02	100%	5.50E-02	(mg/kg-day) ⁻¹	A	IRIS (4)	10/10/00
Benzidine	2.3E+02	100%	2.30E+02	(mg/kg-day) ⁻¹	A	IRIS	10/10/00
Benzo(a)anthracene	7.3E-01	89%	8.20E-01	(mg/kg-day) ⁻¹	B2	NCEA	07/01/93
Benzo(a)pyrene	7.3E+00	89%	8.20E+00	(mg/kg-day) ⁻¹	B2	IRIS	10/10/00
Benzo(b)fluoranthene	7.3E-01	89%	8.20E-01	(mg/kg-day) ⁻¹	B2	NCEA	07/01/93
Benzo(j)fluoranthene	7.3E-01	89%	8.20E-01	(mg/kg-day) ⁻¹	B2	California EPA	1996
Benzo(k)fluoranthene	7.3E-02	89%	8.20E-02	(mg/kg-day) ⁻¹	B2	NCEA	07/01/93
sec-Butylbenzene	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Carbazole	2.0E-02	100%	2.00E-02	(mg/kg-day) ⁻¹	B2	HEAST	07/01/97
Chrysene	7.3E-03	89%	8.20E-03	(mg/kg-day) ⁻¹	B2	NCEA	07/01/93
Dibenzo(a,e)pyrene	7.3E+00	89%	8.20E+00	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzo(a,h)acridine	7.3E-01	89%	8.20E-01	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzo(a,h)anthracene	7.3E+00	89%	8.20E+00	(mg/kg-day) ⁻¹	B2	NCEA	07/01/93
Dibenzo(a,h)pyrene	7.3E+01	89%	8.20E+01	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzo(a,i)pyrene	7.3E+01	89%	8.20E+01	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzo(a,j)acridine	7.3E-01	89%	8.20E-01	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzo(a,l)pyrene	7.3E+01	89%	8.20E+01	(mg/kg-day) ⁻¹	B2	California EPA	1996
7H-Dibenzo(c,g)carbazole	7.3E+00	89%	8.20E+00	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzofuran	N/A	N/A	N/A	N/A	D	IRIS	10/10/00
7,12-Dimethylbenz(a)anthracene	1.46E+02	89%	1.64E+02	(mg/kg-day) ⁻¹	B2	California EPA	1996
Fluoranthene	N/A	N/A	N/A	N/A	D	IRIS	10/10/00
Fluorene	N/A	N/A	N/A	N/A	D	IRIS	10/10/00
Indeno(1,2,3-cd)pyrene	7.3E-01	89%	8.20E-01	(mg/kg-day) ⁻¹	B2	NCEA	07/01/93
2-Methylnaphthalene	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Naphthalene	N/A	N/A	N/A	N/A	C	IRIS	10/10/00
Pentachlorophenol	1.2E-01	76%	1.58E-01	(mg/kg-day) ⁻¹	B2	IRIS	10/10/00
Phenanthrene	N/A	N/A	N/A	N/A	D	IRIS	10/10/00
n-Propylbenzene	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Pyrene	N/A	N/A	N/A	N/A	D	IRIS	10/10/00
1,2,4-Trimethylbenzene	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1,3,5-Trimethylbenzene	N/A	N/A	N/A	N/A	N/A	N/A	N/A
2,3,7,8-Tetrachlorodibenzodioxin	1.50E+05	50%	3.00E+05	(mg/kg-day) ⁻¹	B2	HEAST	07/01/97
2,3,7,8-Tetrachlorodibenzofuran	1.50E+04	50%	3.00E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,7,8-Pentachlorodibenzodioxin	1.50E+05	50%	3.00E+05	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,7,8-Pentachlorodibenzofuran	7.50E+03	50%	1.50E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
2,3,4,7,8-Pentachlorodibenzofuran	7.50E+04	50%	1.50E+05	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,4,7,8-Hexachlorodibenzodioxin	1.50E+04	50%	3.00E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,4,7,8-Hexachlorodibenzofuran	1.50E+04	50%	3.00E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,6,7,8-Hexachlorodibenzodioxin	1.50E+04	50%	3.00E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,6,7,8-Hexachlorodibenzofuran	1.50E+04	50%	3.00E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,7,8,9-Hexachlorodibenzodioxin	1.50E+04	50%	3.00E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,7,8,9-Hexachlorodibenzofuran	1.50E+04	50%	3.00E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
2,3,4,6,7,8-Hexachlorodibenzofuran	1.50E+04	50%	3.00E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,4,6,7,8-Heptachlorodibenzodioxin	1.50E+03	50%	3.00E+03	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,4,6,7,8-Heptachlorodibenzofuran	1.50E+03	50%	3.00E+03	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,4,7,8,9-Heptachlorodibenzofuran	1.50E+03	50%	3.00E+03	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
Octachlorodibenzodioxin	1.50E+01	50%	3.00E+01	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
Octachlorodibenzofuran	1.50E+01	50%	3.00E+01	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998

IRIS = Integrated Risk Information System
 HEAST= Health Effects Assessment Summary Tables
 NCEA= National Center Environmental Assessment
 N/A = Not Applicable

EPA Group:
 A - Human carcinogen
 B1 - Probable human carcinogen - indicates that limited human data are available
 B2 - Probable human carcinogen - indicates sufficient evidence in animals inadequate or no evidence in humans
 C - Possible human carcinogen
 D - Not classifiable as a human carcinogen
 E - Evidence of noncarcinogenicity

- (1) For adjustment from administered to absorbed dose. Refer to RAGS, Part E.
- (2) Equation used for derivation : SFd = SFo / (oral-to-dermal absorption factor)
- (3) For IRIS values, the date IRIS was searched.
 For HEAST values, the date of HEAST publication.
 For NCEA values, the date of the issue paper.
- (4) SFo for benzene is upper end of range given in IRIS.

TABLE 6.2
 CANCER TOXICITY DATA -- INHALATION ROUTE
 Oeser Company
 Bellingham, Washington

Chemical of Potential Concern	Unit Risk	Units	Adjustment (1)	Inhalation Cancer Slope Factor	Units	Weight of Evidence Group	Source	Date (2) (MM/DD/YY)
Acenaphthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzene	7.8E-06	(ug/m ³) ⁻¹	3,500	2.73E-02	(mg/kg-day) ⁻¹	A	IRIS (3)	10/10/00
Benzo(a)anthracene	6.7E-02	(ug/m ³) ⁻¹	3,500	2.35E+02	(mg/kg-day) ⁻¹	A	IRIS	10/10/00
Benzo(a)pyrene	N/A	N/A	N/A	3.10E-01	(mg/kg-day) ⁻¹	B2	NCEA	N/A
Benzo(b)fluoranthene	N/A	N/A	N/A	3.10E+00	(mg/kg-day) ⁻¹	B2	NCEA	N/A
Benzo(j)fluoranthene	N/A	N/A	N/A	3.10E-01	(mg/kg-day) ⁻¹	B2	California EPA	1996
Benzo(k)fluoranthene	N/A	N/A	N/A	3.10E-02	(mg/kg-day) ⁻¹	B2	NCEA	N/A
sec-Butylbenzene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Carbazole	N/A	N/A	N/A	2.0E-02	(mg/kg-day) ⁻¹	B2	Route extrapolation	N/A
Chrysene	N/A	N/A	N/A	3.10E-03	(mg/kg-day) ⁻¹	B2	NCEA	N/A
Dibenzo(a,e)pyrene	N/A	N/A	N/A	3.1E+00	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzo(a,h)acridine	N/A	N/A	N/A	3.1E-01	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzo(a,h)anthracene	N/A	N/A	N/A	3.10E+00	(mg/kg-day) ⁻¹	B2	NCEA	N/A
Dibenzo(a,h)pyrene	N/A	N/A	N/A	3.1E+01	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzo(a,i)pyrene	N/A	N/A	N/A	3.1E+01	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzo(a,j)acridine	N/A	N/A	N/A	3.1E-01	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzo(a,l)pyrene	N/A	N/A	N/A	3.1E+01	(mg/kg-day) ⁻¹	B2	California EPA	1996
7H-Dibenzo(c,g)carbazole	N/A	N/A	N/A	3.1E+00	(mg/kg-day) ⁻¹	B2	California EPA	1996
Dibenzofuran	N/A	N/A	N/A	N/A	N/A	D	IRIS	10/10/00
7,12-Dimethylbenz(a)anthracene	N/A	N/A	N/A	6.2E+01	(mg/kg-day) ⁻¹	B2	California EPA	1996
Fluoranthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Fluorene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Indeno(1,2,3-cd)pyrene	N/A	N/A	N/A	3.10E-01	(mg/kg-day) ⁻¹	B2	NCEA	N/A
2-Methylnaphthalene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Naphthalene	N/A	N/A	N/A	N/A	N/A	C	IRIS	10/10/00
Pentachlorophenol	N/A	N/A	N/A	1.2E-01	(mg/kg-day) ⁻¹	B2	Route extrapolation	N/A
Phenanthrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
n-Propylbenzene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1,2,4-Trimethylbenzene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
1,3,5-Trimethylbenzene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
2,3,7,8-Tetrachlorodibenzodioxin	N/A	N/A	N/A	1.50E+05	(mg/kg-day) ⁻¹	B2	HEAST	0701/97
2,3,7,8-Tetrachlorodibenzofuran	N/A	N/A	N/A	1.50E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,7,8-Pentachlorodibenzodioxin	N/A	N/A	N/A	1.50E+05	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,7,8-Pentachlorodibenzofuran	N/A	N/A	N/A	7.50E+03	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
2,3,4,7,8-Pentachlorodibenzofuran	N/A	N/A	N/A	7.50E+04	(mg/kg-day) ⁻¹	B3	Van der Berg et al.	1998
1,2,3,4,7,8-Hexachlorodibenzodioxin	N/A	N/A	N/A	1.50E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,4,7,8-Hexachlorodibenzofuran	N/A	N/A	N/A	1.50E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,6,7,8-Hexachlorodibenzodioxin	N/A	N/A	N/A	1.50E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,6,7,8-Hexachlorodibenzofuran	N/A	N/A	N/A	1.50E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,7,8,9-Hexachlorodibenzodioxin	N/A	N/A	N/A	1.50E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,7,8,9-Hexachlorodibenzofuran	N/A	N/A	N/A	1.50E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
2,3,4,6,7,8-Hexachlorodibenzofuran	N/A	N/A	N/A	1.50E+04	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,4,6,7,8-Heptachlorodibenzodioxin	N/A	N/A	N/A	1.50E+03	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,4,6,7,8-Heptachlorodibenzofuran	N/A	N/A	N/A	1.50E+03	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
1,2,3,4,7,8,9-Heptachlorodibenzodioxin	N/A	N/A	N/A	1.50E+03	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
Octachlorodibenzodioxin	N/A	N/A	N/A	1.50E+01	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998
Octachlorodibenzofuran	N/A	N/A	N/A	1.50E+01	(mg/kg-day) ⁻¹	B2	Van der Berg et al.	1998

IRIS = Integrated Risk Information System
 HEAST= Health Effects Assessment Summary Tables
 N/A = Not Applicable

EPA Group:
 A - Human carcinogen
 B1 - Probable human carcinogen - indicates that limited human data are available
 B2 - Probable human carcinogen - indicates sufficient evidence in animals and inadequate or no evidence in humans
 C - Possible human carcinogen
 D - Not classifiable as a human carcinogen
 E - Evidence of noncarcinogenicity

(1) Adjustment Factor applied to Unit Risk to calculate Inhalation Slope Factor
 70 kg x 1/20 m³/day x 1000 ug/mg
 (2) For IRIS values, the date IRIS was searched.
 For HEAST values, the date of HEAST publication.
 (3) Unit risk for benzene is upper end of range given in IRIS.
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APPENDIX B
TOXICOLOGICAL PROFILES

Benzene

Benzene is present in the environment from both natural sources (including volcanos, natural constituents of crude oil, forest fires, plant volatiles) and anthropogenic sources (including the use of gasoline, spills, and industrial effluents). Today, most benzene is produced from petroleum sources. Benzene has a long history of industrial use, most notably as a solvent and as a starting material for the synthesis of other chemicals (Hazardous Substance Data Bank [HSDB] 2000).

Benzene enters the atmosphere primarily from fugitive emissions and exhaust connected with its use in gasoline. Another important source is emissions associated with its production and use as an industrial intermediate. In addition, there are discharges into water from industrial effluents and losses during spills. If benzene is released to soil, it will be subject to rapid volatilization near the surface and that which does not evaporate will be highly to very highly mobile in the soil and may leach to groundwater (HSDB 2000).

Inhalation is the major route of exposure to benzene, although both ingestion and dermal contact also are important (United States Environmental Protection Agency [EPA] 1998a). Most of what is known about the human health effects of benzene exposure, including immunotoxicity and hematotoxicity, is based on studies of workers, who were usually exposed for long periods to high concentrations of benzene (EPA 1998b). Benzene has been shown to produce neurotoxic effects in test animals and humans after short-term exposure to high concentrations; however, long-term neurotoxicity exposure studies are lacking (EPA 1998a). There is some evidence of reproductive and developmental effects due to benzene exposure from human epidemiological studies, but data are not conclusive to link low exposure concentrations to effects (EPA 1998a).

Hematotoxicity and immunotoxicity have been consistently reported to be the most critical noncancer effects both in limited studies in humans and experimental animals. The most recent provisional oral reference dose (RfD) of 1×10^{-3} mg/kg-day and inhalation reference concentration (RfC) of 9×10^{-3} mg/m³ (which converts to a RfD of 2.6×10^{-3} mg/kg-day), were derived from an occupational subchronic inhalation study that identified immunotoxicity as the critical effect. The LOAEL from the study was adjusted downward by an uncertainty factor of 1,000 to account for use of a LOAEL rather than a NOAEL, human variability and protection of sensitive subpopulations, extrapolation from a subchronic study to chronic exposure, and data base uncertainties. The oral RfD reflects an additional adjustment for greater oral absorption relative to absorption from the inhalation route. The oral RfD is supported by a similar estimate obtained from a co-principal mouse subchronic drinking water study. Confidence in the oral and inhalation RfDs is medium. Although the principal studies were well conducted, confidence is reduced by uncertainties mentioned above (EPA 1998a).

Benzene is classified as a Group A human carcinogen for all routes of exposure based upon convincing human evidence as well as supporting evidence from animal studies (Integrated Risk Information System [IRIS] 2000). Significantly increased risks of leukemia, chiefly acute myelogenous leukemia, have been reported in benzene-exposed workers in the chemical industry, shoemaking and oil refineries (HSDB 2000). In animals, it has been shown that exposure to benzene increases the risk of cancer in multiple organ systems, including the hematopoietic system, oral and nasal cavities, liver, forestomach, preputial gland, lung, ovary, and mammary

gland (HSDB 2000). The oral and inhalation slope factors (SFs) designated for benzene are 2.9×10^{-2} and 2.91×10^{-2} (milligrams per kilogram per day [mg/kg-day]¹), respectively, and are based on the statistically significant increased incidence of leukemia in individuals with 5 or more years of occupational inhalation exposure to benzene (IRIS 2000).

References

Hazardous Substance Data Bank (HSDB), December 4, 2000, *Benzene*, <http://www.tomescps.com/DATA/HS/HS35.HTM?Top=Yes>

Integrated Risk Information System (IRIS), December 4, 2000, United States Environmental Protection Agency, <http://www.epa.gov/iris/subst/0276.htm>

United States Environmental Protection Agency (EPA), 1998a, *(Draft) Toxicological Review of Benzene (Noncancer Effects)*, National Center for Environmental Assessment (NCEA), Office of Research and Development, EPA, Washington D.C., NCEA-S-0455.

United States Environmental Protection Agency (EPA), 1998b, *Carcinogenic Effects of Benzene: An Update*, National Center for Environmental Assessment, Office of Research and Development, EPA, Washington D.C., EPA/600/P-97/001F.

Fuels (EPH, VPH)

Petroleum products are derived from crude oil. Total petroleum hydrocarbons (TPHs) are measured as volatile petroleum hydrocarbons (VPHs) and extractable petroleum hydrocarbons (EPHs). VPHs consist primarily of the components of gasoline; EPHs consist primarily of the components of diesel. Petroleum hydrocarbons can also be divided into two major fractions based on their structures: (1) aliphatic hydrocarbons, including alkanes and cycloalkanes, and (2) aromatic hydrocarbons, including benzene and polynuclear aromatic hydrocarbons. The molecular size, or chain length, of the petroleum hydrocarbon is expressed using the number of carbons in the chain. For example, EC5 would be designated for a petroleum hydrocarbon containing five carbons in its structure.

Toxic effects from diesel and gasoline can occur from exposure through inhalation, ingestion, and dermal contact. Noncarcinogenic toxic effects and assigned toxicity values of the components of gasoline and diesel are discussed separately for each chemical class including aromatics and alkenes (EC5-EC36), and alkanes (EC5-EC36). TPH fractions are not considered carcinogens; therefore, individual carcinogens, such as benzene, must be calculated separately (Washington Department of Ecology [WDOE] 1999).

Exposure to alkanes causes neurotoxic effects and dermal irritation; neurotoxic effects decrease and dermal irritation increase with increasing chain length (Massachusetts Department of Environmental Protection [MDEP] 1994). The WDOE (1999) designated an oral reference dose (RfD) for alkanes (EC5-EC8) based on the oral RfD for n-hexane (5.7 milligrams per kilogram per day [mg/kg-day]). The n-hexane RfD was extrapolated from the reference concentration (RfC; 5.7 mg/kg-day) designated by the EPA based on epithelial lesions in the nasal cavity in mice following subchronic inhalation exposure and neurotoxicity based on an epidemiological inhalation study. The WDOE (1999) designated an oral RfD for alkanes (>EC8-EC16; 0.03 mg/kg-day) based on "mixture of alkanes" for oral exposure. The WDOE based an oral RfD for higher chain alkanes (>EC16 - EC36; 2 mg/kg-day) based on the oral RfD for mineral oil. Studies of human subjects who had prolonged or excessive exposure to white mineral oil displayed minor structural and functional changes in the cells of the liver, lung, spleen and mesenteric lymph nodes (MDEP 1994).

Exposure to alkenes and aromatics causes dermal irritation and effects to the central nervous system, liver, kidneys, cardiac, and renal system effects (MDEP 1994). The WDOE designated an oral RfD for alkenes and aromatics (collectively designated as aromatics by WDOE) of chain length EC8-EC16 based on the oral RfD for biphenyl (0.05 mg/kg-day). The biphenyl oral RfD was designated by the EPA based on kidney damage effects in rats following chronic oral exposure (IRIS 2000). The WDOE designated an oral RfD for alkenes (>EC16-EC36; 0.03 mg/kg-day) based an oral RfD for pyrene. The pyrene RfD was designated by the EPA based on kidney effects (renal tubular pathology, decreased kidney weights) in mice following subchronic oral exposure (IRIS 2000).

References

Massachusetts Department of Environmental Protection (MDEP), 1994, *Interim Final Petroleum Report: Development of Health-Based Alternative to the Total Petroleum Hydrocarbon (TPH)*

Parameter, MDEP, Bureau of Waste Site Cleanup, Massachusetts.

Integrated Risk Information System (IRIS), December 4, 2000, *Pyrene, Biphenyl, and n-Hexane*, <http://www.epa.gov/iris/>.

Washington Department of Ecology (WDOE), 1999, *Calculation of TPH Human Health Direct Contact Cleanup Levels Using Default Compositions*, WDOE, Cleanup Program, Olympia, Washington.

Noncarcinogenic Polynuclear Aromatic Hydrocarbons

This toxicological profile for noncarcinogenic polynuclear aromatic hydrocarbons (PAHs) includes discussion of environmental sources and toxicological effects of naphthalene and 2-methylnaphthalene. Environmental sources of naphthalene and 2-methylnaphthalene include the distillation and fractionation of either petroleum or coal tar and the manufacture of phthalate plasticizers, resins, dyes, and insect repellents (EPA 1998).

The routes of human exposure to naphthalene and 2-methylnaphthalene include ingestion, inhalation, and dermal contact. Naphthalene is expected to be absorbed through the gastrointestinal tract, the respiratory tract, and the skin (EPA 1998). Toxic effects from exposure to naphthalene include hemolytic anemia, cataracts, and respiratory toxicity towards the respiratory tract (EPA 1998).

There are no studies for oral or dermal exposure of humans or animals to methylnaphthalenes (Agency for Toxic Substances and Disease Registry [ASTDR] 1994). There is limited chronic oral dose-response data for naphthalene in humans or animals (EPA 1998). Data from studies of mice exposed acutely to injections of naphthalene, or 1- or 2-methylnaphthalene, or chronically to 1- or 2-methylnaphthalene in the diet provide suggestive evidence that chronic oral exposure to naphthalene at low doses may produce lung injury (Integrated Risk Information System [IRIS] 2000). However, deriving an oral reference dose (RfD) for naphthalene was judged to be too uncertain, based on metabolic differences between methylnaphthalenes and naphthalene and the absence of lung injury in subchronic oral studies in rats (IRIS 2000). An oral RfD of 2×10^{-2} milligrams per kilogram per day (mg/kg-day) has been developed for naphthalene based on decreased mean terminal body weight in male rats following oral exposure (IRIS 2000). Confidence in the oral RfD is low based on the lack of adequate chronic oral data, the lack of dose-response data for naphthalene-induced hemolytic anemia, and the lack of two-generation reproductive toxicity studies (IRIS 2000). IRIS (2000) does not list an oral RfD specific for methylnaphthalenes; therefore, the RfD for naphthalene was used to assess 2-methylnaphthalene.

Toxic effects from the inhalation route of human exposure to naphthalene include hemolytic anemia and cataracts, though adequate exposure-response data are not available for these effects in humans or animals (EPA 1998). An inhalation reference concentration (RfC) of 3×10^{-3} milligrams per cubic meter (mg/m^3) has been developed for naphthalene based on nasal effects; including hyperplasia and metaplasia in respiratory and olfactory epithelium, respectively; in mice following chronic inhalation exposure (IRIS 2000). Confidence in the inhalation RfC is medium based on the absence of human or primate toxicity data and the uncertainty that the RfC is protective for hemolytic anemia and cataracts effects to humans (IRIS 2000). IRIS (2000) does not list an inhalation RfC specific for methylnaphthalenes; therefore, the RfC for naphthalene was used to assess 2-methylnaphthalene.

Data for humans are inadequate to evaluate a plausible association between naphthalene and cancer (EPA 1998); naphthalene is classified as a possible human carcinogen, Group C (IRIS 2000). The EPA has not designated oral slope factors or inhalation risk estimates for naphthalene or methylnaphthalene. Evidence of possible carcinogenicity in humans exposed to naphthalene and methylnaphthalenes include observations of benign respiratory tumors and one carcinoma in female mice exposed to only naphthalene by inhalation and increase in respiratory tumors associated with exposure to 1-methylnaphthalene (IRIS 2000).

References

Agency for Toxic Substances and Disease Registry (ASTDR), 1994, *(Draft) Toxicological Profile for Naphthalene*, ASTDR, United States Department of Health and Human Services, Public Health Service, Atlanta, Georgia.

Integrated Risk Information System (IRIS), December 5, 2000, United States Environmental Protection Agency, <http://www.epa.gov/iris/subst/0436.htm>

United States Environmental Protection Agency (EPA), August 1998, *Toxicological Review of Naphthalene (CAS No. 91-20-3), In Support of Summary Information on the Integrated Risk Information System (IRIS)*, EPA, Washington D.C.

Pentachlorophenol (PCP), a synthetic substance, is principally used as an industrial wood preservative (Hazardous Substance Data Bank [HSDB] 2000). PCP is present in the environment as the result of emissions of factories, hazardous waste sites, and other anthropogenic sources (HSDB 2000).

Human are generally exposed to technical-grade pentachlorophenol, which usually contains such toxic impurities as polychlorinated dibenzo-p-dioxins and dibenzofurans (Agency for Toxic Substances and Disease Registry [ATSDR] 2000). Some of the effects observed in humans (or the severity and dose-response characteristics of the effects) may be partially related to the presence of impurities (ASTDR 2000). In general, the most significant routes of human exposure to PCP are from occupational exposure by inhalation of contaminated workplace air and by dermal contact. Additionally, humans can be exposed to PCP by ingestion of contaminated drinking water, food, or soil (ASTDR 2000).

There is limited data available on the inhalation toxicity of PCP in humans and animals (ASTDR 2000 and Integrated Risk Information System [IRIS] 2000). There is limited information on the adverse effects in human from oral exposure to PCP; however, oral exposure to PCP has been shown to affect the liver, kidney, central nervous system, endocrine system, immune system, and reproductive system in animals (ASTDR 2000 and IRIS 2000). Dermal exposure by humans to PCP has been shown cause adverse effects to the liver, kidneys, skin, blood, lungs, and gastrointestinal tract; however, the relationship between dose and effects has not been quantified to date (ASTDR 2000).

Brief human exposures to high levels of PCP may result in adverse effects to organ systems, including the liver, skin, blood, lungs, central nervous system, and gastrointestinal tract. Such poisoning may also result in death. Long-term exposures to lower levels of PCP can cause damage to the liver, blood, and central nervous system (ASTDR 2000).

An oral reference dose (RfD) of 3×10^{-2} milligrams per kilogram per day (mg/kg-day) has been developed for PCP based on liver and kidney pathology observed in rats following oral exposure (IRIS 2000). PCP is classified as a probable human carcinogen, group B2, based on animal studies and lack of supporting human data (IRIS 2000). An oral slope factor of 1.2×10^{-1} (mg/kg-day)⁻¹ has been developed for PCP based on the increased incidence of liver tumors, pheochromocytomas, and hemangiosarcomas in female mice following oral exposure (IRIS 2000).

References

Agency for Toxic Substances and Disease Registry (ATSDR), February 2000, *Toxicological Profile for Pentachlorophenol (Draft)*, ATSDR, United States Department of Health and Human Services, Public Health Services, Atlanta, Georgia.

Hazardous Substance Data Bank (HSDB), December 4, 2000, *Pentachlorophenol*, <http://www.tomescps.com/DATA/HS/HS894.HTM?Top=Yes>

Integrated Risk Information System (IRIS), December 4, 2000, United States Environmental Protection Agency, <http://www.epa.gov/iris/subst/0086.htm>

Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans (Dioxins/Furans)

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are two classes of chemicals, also known as dioxins and furans, that are structurally related, tricyclic, almost planar, aromatic organic compounds that exhibit similar physical, chemical, and, to some extent, biological properties. There are 75 PCDDs (known as congeners [members of the same chemical family]) and 135 PCDFs (congeners) differentiated by the number and location of chlorine atoms that are present in each congener. Most studies of dioxins/furans focus on the most toxic member of this family of chemicals, 2,3,7,8-tetrachlorodibenzodioxin (TCDD), which is commonly (and inaccurately) called dioxin.

Dioxins are produced by natural processes, although at much lower levels than are produced by man. Historically, commercial or municipal waste incineration, manufacture, and use of certain herbicides and chlorine bleaching of pulp and paper resulted in the major releases of dioxins to air and water. Currently, the uncontrolled burning of residential waste and accidental fires at landfills are thought to be among the largest sources of dioxins to the environment (EPA 2000).

Though 2,3,7,8-TCDD is susceptible to photodecomposition, it is generally resistant to other degradation processes. Dioxins and furans may persist in the environment for a long time. These chemicals have extremely low vapor pressures, extremely low water solubilities, and a strong tendency to adsorb to soil or sediment particles. Crops grown in contaminated soil may take up 2,3,7,8-TCDD in their roots. 2,3,7,8-TCDD bioconcentrates in some aquatic organisms and may bioaccumulate through the food chain.

Workers involved in the production or use of chlorinated pesticides can be exposed to 2,3,7,8-TCDD, as can workers at municipal and industrial incinerators and hazardous waste sites. The general public can be exposed to dioxins and furans by direct contact with contaminated soil or by consuming contaminated fish, meat, milk, or root vegetables. For populations living near waste incinerators, inhalation of small particles of contaminated fly ash, could be a major source of exposure. Exposures from drinking water are probably negligible.

In humans, overexposure to 2,3,7,8-TCDD has caused chloracne, a severe skin lesion which can be very disfiguring and which often lasts for years after exposure. There is limited evidence to suggest the 2,3,7,8-TCDD causes liver damage, loss of appetite and weight loss and digestive disorders in humans. Animal studies have shown many different adverse effects from 2,3,7,8-TCDD exposure. The severity and type of adverse effect varies with species. Animal studies have demonstrated severe liver damage, severe weight loss followed by death, toxicity to the immune system, spontaneous abortions, and malformations in offspring whose mothers were exposed to the chemical during pregnancy. In addition, 2,3,7,8-TCDD has been demonstrated to cause cancer in rats and mice. EPA has classified 2,3,7,8-TCDD as a Group B2 probable human carcinogen. Oral and inhalation slope factors have been derived, both equal to 1.5×10^5 (milligrams per kilogram per day [mg/kg-day])⁻¹, based on an increased incidence of respiratory system and liver cancers observed in rats who were exposed to 2,3,7,8-TCDD in their diet.

References:

United States Environmental Protection Agency (EPA), 2000, *Questions and Answers about Dioxins*, EPA, Office of Research and Development, National Center for Environmental Assessment, Interagency Working Group on Dioxin, Washington, D.C.

United States Environmental Protection Agency (EPA), 1989, *Interim procedures for estimating risks associated with exposure to mixtures of chlorinated dibenzo-p-dioxins and -dibenzofurans (CDDs and CDFs) and 1989 update*, U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, D.C.; EPA/625/3-89/016.

Vanden Berg, M. et al., 1998, *Toxic Equivalency Factors (TEFs) for PCBs, PCDDs, PCDFs, for Humans and Wildlife*, Environmental Health perspectives, Volume 106, Number 12. Pp. 775-792.

Table 1.	
2,3,7,8-TCDD EQUIVALENCY FACTORS	
CONGENER	TOXIC EQUIVALENCY FACTOR
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	1
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.0001
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.0001

Key:

TCDD =	Tetrachlorodibenzo-p-dioxin
PeCDD =	Pentachlorodibenzo-p-dioxin
HxCDD =	Hexachlorodibenzo-p-dioxin
HpCDD =	Heptachlorodibenzo-p-dioxin
OCDD =	Octachlorodibenzo-p-dioxin
TCDF =	Tetrachlorodibenzofuran
PeCDF =	Pentachlorodibenzofuran
HxCDF =	Hexachlorodibenzofuran
HpCDF =	Heptachlorodibenzofuran
OCDF =	Octachlorodibenzofuran

Source: Environmental Health Perspectives, Volume 106, Number 12, December 1998, pages 1-36.

Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs contain only carbon and hydrogen and consist of two or more fused benzene rings in linear, angular, or cluster arrangements. PAHs are formed during the incomplete burning of fossil fuel, garbage, or any organic matter. PAHs produced by burning may be carried into the air on dust particles and distributed into water and soil. In general, PAHs do not evaporate easily and do not dissolve in water.

Exposure to PAHs may occur by inhaling airborne particles, drinking water, or accidentally ingesting soil or dust containing PAHs. In addition, smoking tobacco or eating charcoal-broiled food are common routes of exposure to PAHs.

Some PAHs are known carcinogens, and potential health effects caused by PAHs are usually discussed in terms of an individual PAH compound's carcinogenic or non-carcinogenic effects. Little attention has been paid to noncarcinogenic effects of PAHs. Rapidly growing tissues, such as the intestinal lining, bone marrow, lymphoid organs, blood cells, and testes seem to be especially susceptible targets to non-carcinogenic effects. Concentrations of 150 mg/kg or more administered to laboratory animals have been shown to inhibit body growth. Neither an oral or inhalation RfD have been developed (Integrated Risk Information System [IRIS] 2000).

Exposure to benzo(a)pyrene (B(a)P) and other carcinogenic PAHs, including benzo(b)fluoranthene, chrysene, benzo(k)fluoranthene, dibenzofuran, and indeno(1,2,3-cd)pyrene, can cause cancer at the point of exposure. Sufficient animal carcinogenicity data exists for EPA to classify these PAH compounds, as class B2, probable human carcinogens (IRIS 2000). B(a)P is used as the surrogate for evaluation of the toxicity of all of the Class B2 carcinogenic PAHs, because only B(a)P has been assigned an oral slope factor by EPA (EPA 2000).

Animals exposed to high levels of B(a)P in air develop lung tumors; when exposed via the dietary route they develop stomach tumors; and when B(a)P is painted on skin, animals develop skin tumors. Benzo(b)fluoranthene produced tumors in mice after lung implantation, intraperitoneal or subcutaneous injection, and skin painting. Benzo(k)fluoranthene produced tumors after lung implantation in mice and when administered with a promoting agent in skin-painting studies. Chrysene produced carcinomas and malignant lymphoma in mice after intraperitoneal injection and skin carcinomas in mice following dermal exposure. Indeno(1,2,3-cd)pyrene produced tumors in mice following lung implants, subcutaneous injection and dermal exposure (IRIS 2000).

The oral slope factor of $7.3 \text{ (mg/kg-day)}^{-1}$ is based on a geometric mean of four slope factors obtained by differing modeling procedures. The slope factors calculated ranged from 4.5 to $11.7 \text{ (mg/kg-day)}^{-1}$. The slope factors were calculated from two different studies in two species of outbred rodents. The first study found an increased incidence of forestomach tumors in male and female CRW-Swiss mice given B(a)P in their diets. The second study found an increased incidence of tumors of the forestomach, esophagus and larynx in Sprague-Dawley rats fed B(a)P. These studies have several commonalities including the mode of administration, tumor site, tumor types and the presumed mechanisms of action. The data are considered to be less than optimal, but acceptable, and the use of the geometric mean of four slope factors is preferred because it makes use of more of the available data (IRIS 2000).

An inhalation unit risk or slope factor for B(a)P of $3.1 \text{ (mg/kg-day)}^{-1}$ (obtained from the Health Environmental Affects Summary Table [HEAST] as referenced by EPA [2000]) was used for the purposes of this risk evaluation.

References

Integrated Risk Information System (IRIS), December 4, 2000; *Benzo(a)pyrene, Benzo(b)fluoranthene, Chrysene, Benzo(k)fluoranthene, Dibenzofuran, and Indeno(1,2,3-cd)pyrene*; United States Environmental Protection Agency, <http://www.epa.gov/iris/subst/>.

United States Environmental Protection Agency (EPA), Region 9, December 4, 2000, *Preliminary Remediation Goals (PRGs)*, EPA, Region 9, <http://www.epa.gov/region09/waste/sfund/prg/index.htm>

1,2,4-Trimethylbenzene

Production of 1,2,4-trimethylbenzene (1,2,4-TMB) occurs during petroleum refining, and it is used in a number of industrial applications including the production of trimellitic anhydrides, pharmaceuticals, and dyes (EPA 1994). 1,2,4-TMB (also called *Pseudocumene*) is released directly to the environment as a component of gasoline and as an emission from gasoline-powered vehicles, municipal waste-treatment plants, and coal-fired power stations (HSDB 2000).

Absorption of 1,2,4-TMB occurs from oral, inhalation, and dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption. 1,2,4-TMB is lipophilic and may accumulate in fat and fatty tissues (EPA 1994). Acute exposure to 1,2,4-TMB is irritating to the skin, eyes, and mucous membranes and can cause central nervous system depression and thrombocytopenia (HAZARTEXT 2000).

Chronic effects from exposure to 1,2,4-TMB may include nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations to blood clotting. Hematological effects may have been due to trace amounts of benzene, based on a study of health effects to painters who worked with a solvent containing 50% 1,2,4-TMB and 30% 1,3,5-TMB. Toxic effects in rats from chronic inhalation exposure to a TMB isometric mixture included decreased weight gain, lymphopenia, and neutrophilia. There is no information regarding the carcinogenicity of 1,2,4-TMB (EPA 1994).

The EPA has designated a provisional oral reference dose (RfD) of 0.05 milligram per kilogram per day (mg/kg-day) for 1,2,4-TMB based on observed effects of decreased body weight gain; increased liver and kidney weights; and increased serum phosphorous levels in rats from chronic oral exposure to 1,3,5-TMB. The EPA used 1,3,5-TMB as a surrogate to derive the 1,2,4-TMB RfD, based on an assumption that 1,2,4- and 1,3,5-TMB are similarly metabolized (as supported by urinary excretion data) and have similar toxicological endpoints (potency and target organs). Confidence in the principal study is high, and confidence in the database is low. Consequently, the overall confidence in the provisional RfD for 1,2,4-TMB is low (EPA 2000a).

The EPA has designated a provisional reference concentration (RfC) of 0.006 milligram per cubic meter (mg/m³) for 1,2,4-TMB, based on observed significant increases in central nervous system toxicity (vertigo, headaches, and drowsiness) and insignificant increases in the incidences of respiratory effects (bronchitis) and hematological effects (hyperchromic anemia and blood clotting alterations) in a subchronic occupational inhalation exposure to Fleet-X DV 99, a solvent containing 1,2,4-TMB (>50%); 1,3,5-TMB (>30%); and other aromatic organics. Confidence in the principal study is low, and confidence in the database is low (based on lacking adequate data for individual TMB isomers and developmental and reproductive toxicity studies). Consequently, confidence in the provisional RfC for 1,2,4-TMB is low (EPA 2000b).

References

HAZARTEXT, December 4, 2000, *Pseudocumene*,
<http://www.tomescps.com/DATA/DT/DT1110-3.htm>

Hazardous Substance Data Bank (HSDB), December 4, 2000, 1,2,4-TMB,
<http://www.tomescps.com/DATA/HS/HS5293.HTM?Top=Yes>

United States Environmental Protection Agency (EPA), December 11, 2000a, *Risk Assessment Issue Paper for: Derivation of a Provisional RfD for 1,2,4-Trimethylbenzene (CASRN 95-63-6) and 1,3,5-Trimethylbenzene (CASRN 108-67-8)*, EPA, National Center for Environmental Assessment, Superfund Technical Support Center, Cincinnati, Ohio.

United States Environmental Protection Agency (EPA), December 11, 2000b, *Risk Assessment Issue Paper for: Derivation of a Provisional RfC for 1,2,4-Trimethylbenzene (CASRN 95-63-6) and 1,3,5-Trimethylbenzene (CASRN 108-67-8)*, EPA, National Center for Environmental Assessment, Superfund Technical Support Center, Cincinnati, Ohio.

United States Environmental Protection Agency (EPA), 1994, *Chemical Summary for 1,2,4-Trimethylbenzene*, EPA, Office of Pollution Prevention and Toxics, Washington D.C.; EPA 749-F-94-022a.

APPENDIX C
WORKER EXPOSURE TO ON-FACILITY AIR

This appendix provides estimates of exposure to on-facility air and associated risks for current facility workers based on actual on-facility air sampling data. Although on-facility air releases during on-going facility operations are under the jurisdiction of the Northwest Air Pollution Authority (NWAPA), EPA analyzed air samples in attempt to identify potential sources of contaminants in air. Because these air sampling data were readily available, EPA directed E & E to calculate onsite risks to workers from inhalation of on-facility air. These risks are presented separately from the worker risks presented in Section 5 because the latter accounts for air exposure due to releases from contaminated soil, which is not the purview of NWAPA.

For this assessment, maximum concentrations at each air sampling station shown in Figure C-1 were screened against EPA Region 9 PRGs (EPA 2000d) to determine COPCs. A summary of exposure estimates and associated risks for each air station are provided in Tables C-1 and C-2. This section is not intended to be a stand alone risk assessment; it is added for informational purposes in addition to the objectives of the overall risk assessment.

Exposure Assessment

The on-facility air stations used for this assessment, AS-21, AS-22, AS-23, AS-25, and AS-29, are shown in Figure C-1. Three sets of air samples were collected during July 1999, with another three taken in September/October 1999. Air sampling data collected during facility operation are provided in the PSCSR (E & E 2000a). AS-21, AS-22, and AS-23 were placed in areas likely to yield the highest COPC concentrations, with locations adjacent to, and downwind of, the retort, evaporator, and tank farm/dip tank, respectively. At AS-21, instantaneous grab VOC samples (30 second sampling duration) were collected from the retort as soon as the door was opened and treated poles were removed. The short-term release as the door opened led to high VOC results for the retort sampling location. Time-integrated samples were collected over a 24-hour period at the evaporator (AS-22) and tank farm/dip tank (AS-23) to characterize the continuous emissions during operation at these locations. AS-25 and AS-29 were placed on the facility fenceline to characterize emissions downwind from treated poles stored in the North Pole Yard.

Maximum COPC concentrations from each air station were used as EPCs to assess exposure to current facility workers. Maximum concentrations were used because there was insufficient data to reliably calculate a 95% UCL. CDIs and LADIs were calculated using the methods described previously in Section 3.3.2 to determine potential exposure to noncarcinogenic and carcinogenic COPCs. CDIs and LADIs are expressed in mg/kg-day and are provided in Tables C-1 and C-2. Worker exposure factors used for evaluating inhalation exposure for this assessment can be found in TARA Table 4.16.

While data from these air stations represent emissions from on-facility sources, they are not likely to be representative of typical, long-term exposure to employees. Therefore, these data result in what is likely a conservative estimate of worker risks and hazards due to exposure to on-facility air. In addition, use of the maximum detected air concentration from a limited number of sampling events to represent chronic exposure is an uncertainty, likely to be conservative.

Toxicity Assessment

As in the HHRA, toxicity data for COPCs were used to determine the potential for adverse health effects with exposure. Toxicity values for carcinogenic and noncarcinogenic COPCs were obtained from IRIS, HEAST, and NCEA, which are described in Section 4. Toxicity values used for this assessment are included in Tables C-1 and C-2. For dioxin and furan congeners, toxicity was assessed using TEFs.

Risk Characterization

Risks from potential exposure to carcinogenic COPCs at each air station were calculated using the methods described in Section 5.2.1. The potential for adverse effects from exposure to noncarcinogenic COPCs at each air station were determined using the methods described in Section 5.2.2.

Excess lifetime cancer risks associated with inhalation of on-facility air at the sampled locations are shown in Table C-1, and hazards for noncarcinogenic effects are shown in Table C-2. The tables list individual COPCs present at each sample location. They are also summed for each sample location. Summed excess cancer risk estimates ranged from 6E-6 to 3E-3; hazard indices ranged from 0.4 to 3,000.

Table C-1				
Excess Lifetime Cancer Risks Associated With Inhalation of On-Facility Air by Current Workers				
The Oeser Company				
Bellingham, Washington				
COPC	Location	SF _i (mg/kg-d) ⁻¹	LADI (mg/kg-d)	Risk
Benzene	AS-21	2.70E-02	1.19E-02	3.2E-04
Pentachlorophenol	AS-21	1.20E-01	2.08E-04	2.5E-05
2,3,7,8-TCDD	AS-21	1.50E+05	3.52E-12	5.3E-07
1,2,3,7,8-PeCDD	AS-21	1.50E+05	6.06E-12	9.1E-07
1,2,3,6,7,8-HxCDD	AS-21	1.50E+05	3.35E-12	5.0E-07
1,2,3,7,8,9-HxCDD	AS-21	1.50E+05	3.17E-12	4.8E-07
1,2,3,4,6,7,8-HpCDD	AS-21	1.50E+05	9.71E-12	1.5E-06
OCDD	AS-21	1.50E+05	1.01E-12	1.5E-07
2,3,4,7,8-PeCDF	AS-21	1.50E+05	1.76E-12	2.6E-07
1,2,3,4,7,8-HxCDF	AS-21	1.50E+05	2.12E-12	3.2E-07
2,3,4,6,7,8-HxCDF	AS-21	1.50E+05	1.94E-12	2.9E-07
1,2,3,4,6,7,8-HpCDF	AS-21	1.50E+05	7.90E-12	1.2E-06
Summary Excess Cancer Risk =				4.E-04
Benzene	AS-22	2.70E-02	2.10E-04	5.7E-06
Pentachlorophenol	AS-22	1.20E-01	7.27E-04	8.7E-05
2,3,7,8-TCDD	AS-22	1.50E+05	1.12E-11	1.7E-06
1,2,3,7,8-PeCDD	AS-22	1.50E+05	5.91E-11	8.9E-06
1,2,3,6,7,8-HxCDD	AS-22	1.50E+05	6.75E-11	1.0E-05
1,2,3,7,8,9-HxCDD	AS-22	1.50E+05	3.38E-11	5.1E-06
1,2,3,4,6,7,8-HpCDD	AS-22	1.50E+05	1.86E-10	2.8E-05
OCDD	AS-22	1.50E+05	1.25E-11	1.9E-06
2,3,4,7,8-PeCDF	AS-22	1.50E+05	1.44E-11	2.2E-06
1,2,3,4,7,8-HxCDF	AS-22	1.50E+05	1.27E-11	1.9E-06
2,3,4,6,7,8-HxCDF	AS-22	1.50E+05	7.97E-12	1.2E-06
Summary Excess Cancer Risk =				2.E-04
Benzene	AS-23	2.70E-02	1.47E-04	4.0E-06
Pentachlorophenol	AS-23	1.20E-01	1.22E-05	1.5E-06
1,2,3,7,8-PeCDD	AS-23	1.50E+05	4.56E-12	6.8E-07
Summary Excess Cancer Risk =				6.E-06
Benzene	AS-25	2.70E-02	2.24E-04	6.0E-06
Pentachlorophenol	AS-25	1.20E-01	1.38E-05	1.7E-06
1,2,3,7,8-PeCDD	AS-25	1.50E+05	4.12E-12	6.2E-07
1,2,3,4,6,7,8-HpCDD	AS-25	1.50E+05	2.46E-11	3.7E-06
Summary Excess Cancer Risk =				1.E-05
Benzene	AS-29	2.70E-02	6.99E-05	1.9E-06
Pentachlorophenol	AS-29	1.20E-01	2.38E-02	2.9E-03
1,2,3,7,8-PeCDD	AS-29	1.50E+05	8.39E-12	1.3E-06
1,2,3,6,7,8-HxCDD	AS-29	1.50E+05	3.59E-12	5.4E-07
1,2,3,4,6,7,8-HpCDD	AS-29	1.50E+05	7.55E-12	1.1E-06
Summary Excess Cancer Risk =				3.E-03

Key:

COPC = Chemical of potential concern.
LADI = Lifetime average daily intake.
SF_i = Slope factor.

Table C-2				
Hazard Indices Associated With Inhalation of On-Facility Air by Current Workers				
The Oeser Company				
Bellingham, Washington				
COPC	Location	RfDi (mg/kg-d)	CDI (mg/kg-d)	HQ
Benzene	AS-21	1.70E-03	3.33E-02	2.0E+01
n-Butylbenzene	AS-21	1.00E-02	5.48E-02	5.5E+00
sec-Butylbenzene	AS-21	1.00E-02	1.90E+00	1.9E+02
tert-Butylbenzene	AS-21	1.00E-02	3.52E-01	3.5E+01
Ethylbenzene	AS-21	2.90E-01	5.87E-01	2.0E+00
Pentachlorophenol	AS-21	3.00E-02	5.83E-04	1.9E-02
n-Propylbenzene	AS-21	1.00E-02	1.29E+00	1.3E+02
iso-Propylbenzene	AS-21	1.10E-01	9.59E-01	8.7E+00
Toluene	AS-21	1.10E-01	6.65E-01	6.0E+00
1,2,4-Trimethylbenzene	AS-21	1.70E-03	2.94E+00	1.7E+03
1,3,5-Trimethylbenzene	AS-21	1.70E-03	7.44E-01	4.4E+02
Xylenes	AS-21	2.00E-01	6.07E-01	3.0E+00
Hazard Index =				3.E+03
Benzene	AS-22	1.70E-03	5.87E-04	3.5E-01
Pentachlorophenol	AS-22	3.00E-02	2.04E-03	6.8E-02
Hazard Index =				4.E-01
Benzene	AS-23	1.70E-03	4.11E-04	2.4E-01
Pentachlorophenol	AS-23	3.00E-02	3.41E-05	1.1E-03
Hazard Index =				2.E-01
Benzene	AS-25	1.70E-03	6.26E-04	3.7E-01
Pentachlorophenol	AS-25	3.00E-02	3.86E-05	1.3E-03
1,2,4-Trimethylbenzene	AS-25	1.70E-03	2.15E-03	1.3E+00
Hazard Index =				2.E+00
Benzene	AS-29	1.70E-03	1.96E-04	1.2E-01
Dibenzofuran	AS-29	4.00E-03	7.05E-03	1.8E+00
Pentachlorophenol	AS-29	3.00E-02	6.65E-02	2.2E+00
1,2,4-Trimethylbenzene	AS-29	1.70E-03	1.96E-03	1.2E+00
Hazard Index =				4.E+00

Key:

CDI = Chronic daily intake.
 HQ = Hazard quotient.
 RfDi = Reference dose.

Figure C-1

THE OESER COMPANY
SUPERFUND SITE

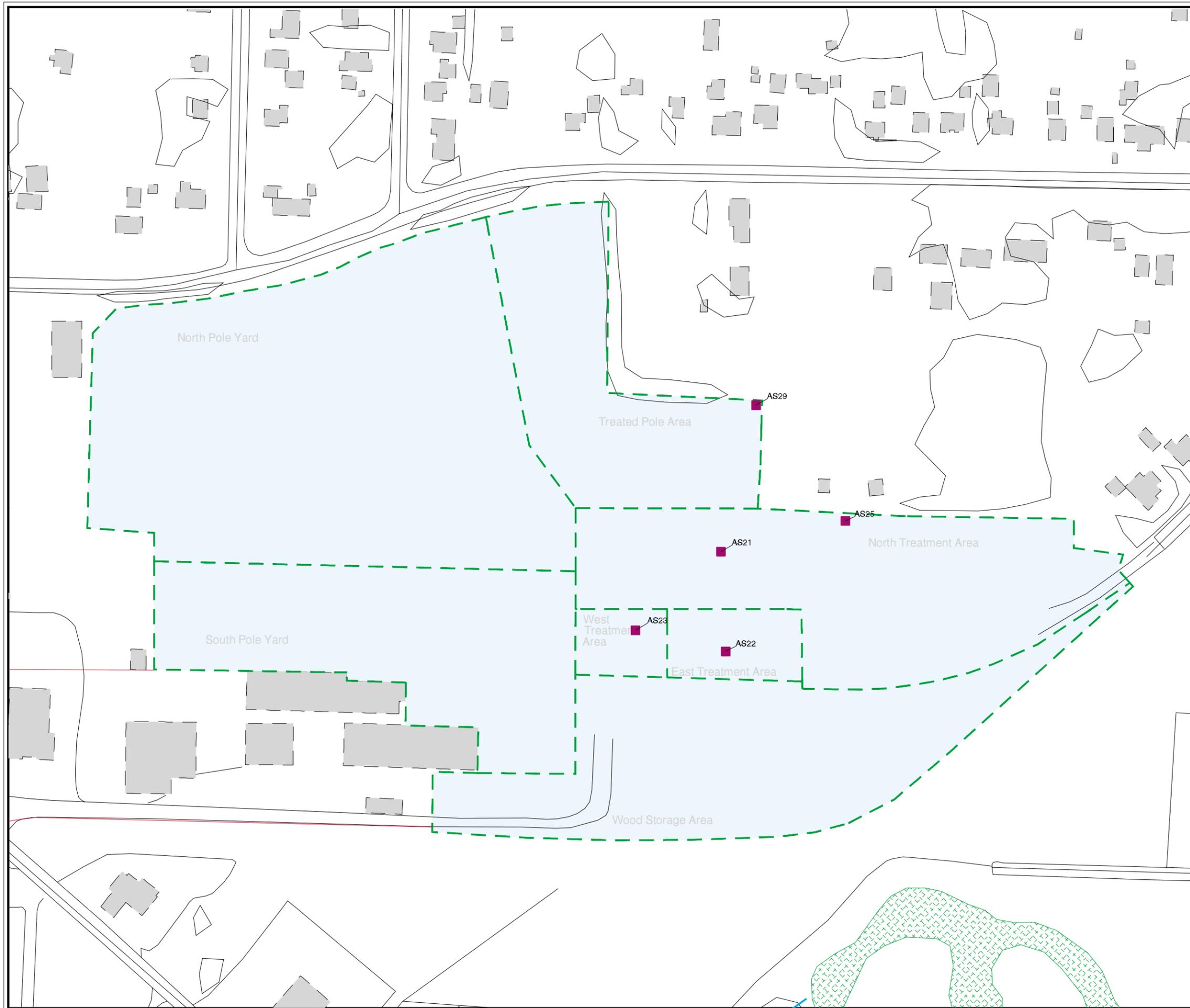
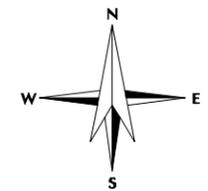
Bellingham, Washington

Human Health Risk Assessment

On-Facility Air Sampling Locations

LEGEND

■ Air Sampling Location



APPENDIX D
ASSESSMENT OF RISKS FOR PETROLEUM HYDROCARBONS

APPENDIX D

Methods for calculating risks from exposure to total petroleum hydrocarbon (TPH) mixtures under MTCA became effective on August 15, 2001. Therefore, risks due to exposure to TPH in soil were recalculated using Washington State Department of Ecology's "*Workbook for Calculating Cleanup Levels for Petroleum Contaminated Sites*," version MTCATPH10.

As an initial screening, all TPH concentration data collected under the remedial investigation were compared to MTCA Method A cleanup levels. Those sample locations with TPH concentrations exceeding Method A values were selected for further analysis. Tables D-1 and D-2 provide the results of the initial screening of TPH concentrations against Method A cleanup levels and specific sample locations, depths, and ground cover types where TPH concentrations exceeded the cleanup levels.

Fractionated data for those soil samples collected under the remedial investigation were used to calculate risks from exposure to TPH mixtures. The fractionated data for each soil sample were input in the Ecology workbook. Default values for soil porosity, volumetric water content, volumetric air content, soil bulk density, fraction organic carbon, and dilution factor were used. The default exposure scenarios provided in the workbook were changed to reflect the resident adult and child and worker exposure scenarios developed for the HHRA. The exposure factors used for these scenarios are provided in Tables 4.1 through 4.17. The workbook calculated the hazard index (HI) for exposure to noncarcinogenic fractions of TPH. [Risks associated with exposure to carcinogenic fractions of TPH (e.g., benzene, PAHs) were already assessed in the HHRA.] The HIs are based on incidental ingestion of TPH in soil as well as dermal contact with TPH in soil. Table D-3 provides HIs for exposure to TPH for each area, as calculated by Ecology's workbook. These values were added to the ingestion risks provided in Tables 5-1 through 5-9 for the appropriate scenarios.

No exceedances of TPH were found in facility surface soil. From 0 to 6 feet below ground surface (bgs), however, samples collected from the North Pole Yard (NPY) and North Treatment Area (NTA) contained TPH at concentrations exceeding Method A cleanup levels and resulted in a HI greater than the MTCA benchmark level of 1 for the future resident (HI = 4). Risks to future workers were below the MTCA benchmark level of 1.

At 6 to 12 feet bgs, samples from the NPY, NTA, and South Pole Yard (SPY) contained concentrations of TPH exceeding MTCA Method A cleanup levels. Hazards associated with exposure to the future resident in the SPY and NTA exceeded the MTCA benchmark of 1 (HI = 3, each location).

At 12 to 18 feet bgs, only the NTA contained TPH at concentrations exceeding Method A cleanup levels and risks only slightly exceeded the regulatory benchmark (HI = 2).

Overall, estimates of exposure for future residents and workers to TPH in on-facility soils resulted in slight exceedances of the MTCA regulatory benchmark of 1. These risks are further discussed in the HHRA summary, Section 6.0.

Table D-1

**Screening of Total Petroleum Hydrocarbon Concentrations Against Method A Cleanup Levels
The Oeser Company
Bellingham, WA**

Sample Area	Depth	EPC (mg/kg)		Method A Cleanup Level (mg/kg)	
		VPH	EPH	VPH	EPH
Wood Storage Area Cap	surface	65.9	NP	100	2,000
Wood Storage Area	6-12	179.5	NP	100	2,000
North Treatment Area	surface	65.9	NP	100	2,000
North Treatment Area	0-6	295.8	5,224.3	100	2,000
North Treatment Area	6-12	1,164.4	4,725.8	100	2,000
North Treatment Area	12-18	NP	20,000	100	2,000
North Pole Yard	0-6	193.5	2,267.7	100	2,000
North Pole Yard	6-12	481	NP	100	2,000
South Pole Yard	6-12	1,684.5	6,535	100	2,000

Key:

EPC = Exposure Point Concentration

EPH = Extractable Petroleum Hydrocarbons

mg/kg = milligram per kilogram

NP = Not present

Note: Bold indicates exceedance of MTCA Method A cleanup level.

Table D-2

**Sample Locations with Exceedances of MTCA Method A Cleanup Levels
for Total Petroleum Hydrocarbons and Ground Cover Type
The Oeser Company
Bellingham, WA**

Wood Storage Area		North Treatment Area		North Pole Yard		South Pole Yard	
B-Q20	exposed soil	MW-36-S	asphalt	B-B13	vegetation	B-O7	gravel cap
		B-M20	asphalt	B-J2	vegetation		
		MW-31-S	sawdust				

Table D-3 Hazard Indices Associated with Exposure to TPH Mixtures The Oeser Company Bellingham, WA		
	HI	
Sample Location	Future Resident	Future Worker
Samples from 0 to 6 feet bgs		
North Pole Yard	9.9E-01	1.0E-01
North Treatment Area	4.0E+00	4.0E-01
Samples from 6 to 12 feet bgs		
North Pole Yard	8.4E-01	1.0E-01
South Pole Yard	3.0E+00	4.0E-01
Wood Storage Area	3.6E-01	5.0E-02
North Treatment Area	3.0E+00	4.0E-01
Samples from 12 to 18 feet bgs		
North Treatment Area	1.6E+00	1.8E-01

Key:

bgs = below ground surface

ELCR = excess lifetime cancer risk

HI = hazard index

TPH = total petroleum hydrocarbons

Note: Values shown in bold indicate risks greater than 1E-05 or hazard index greater than 1