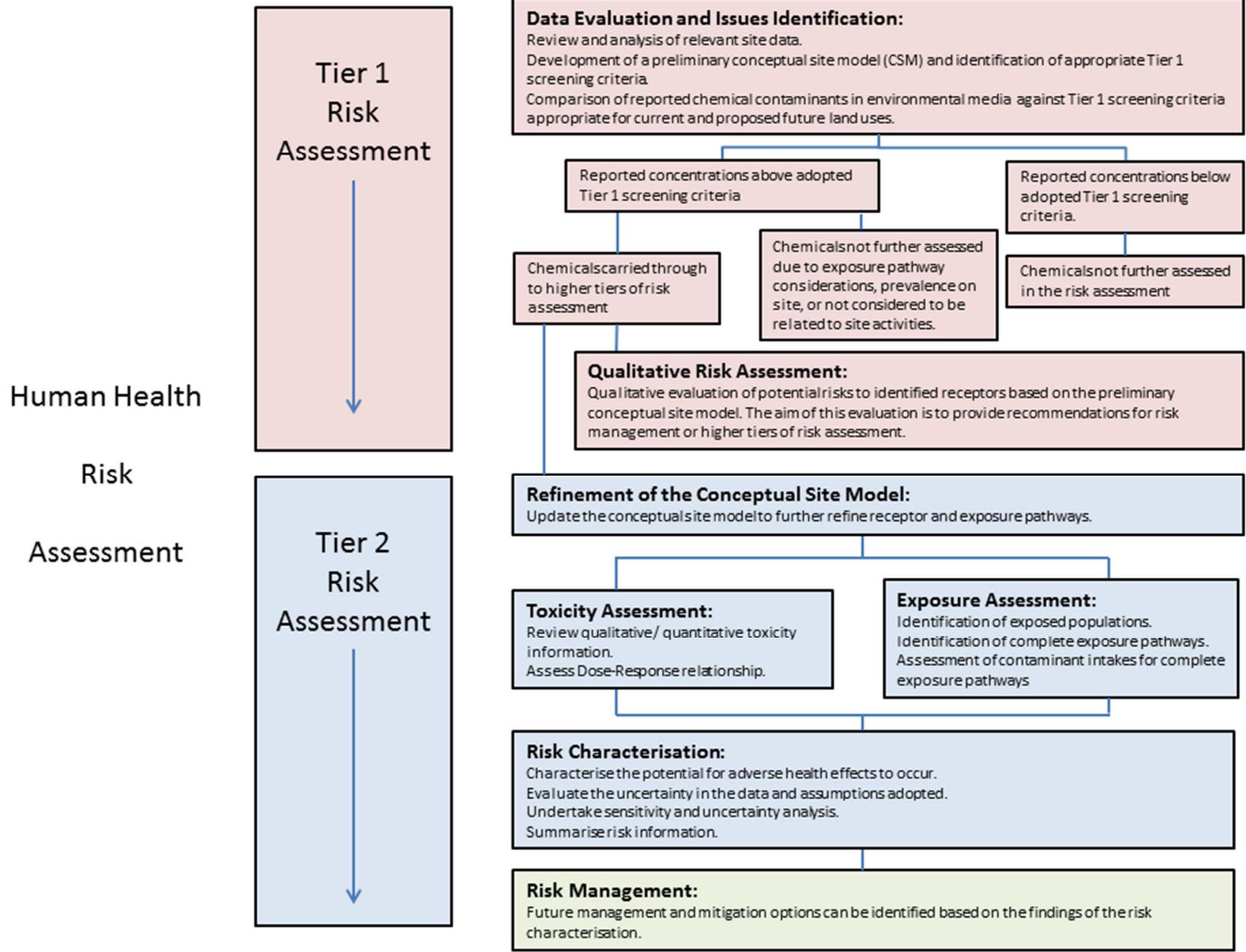


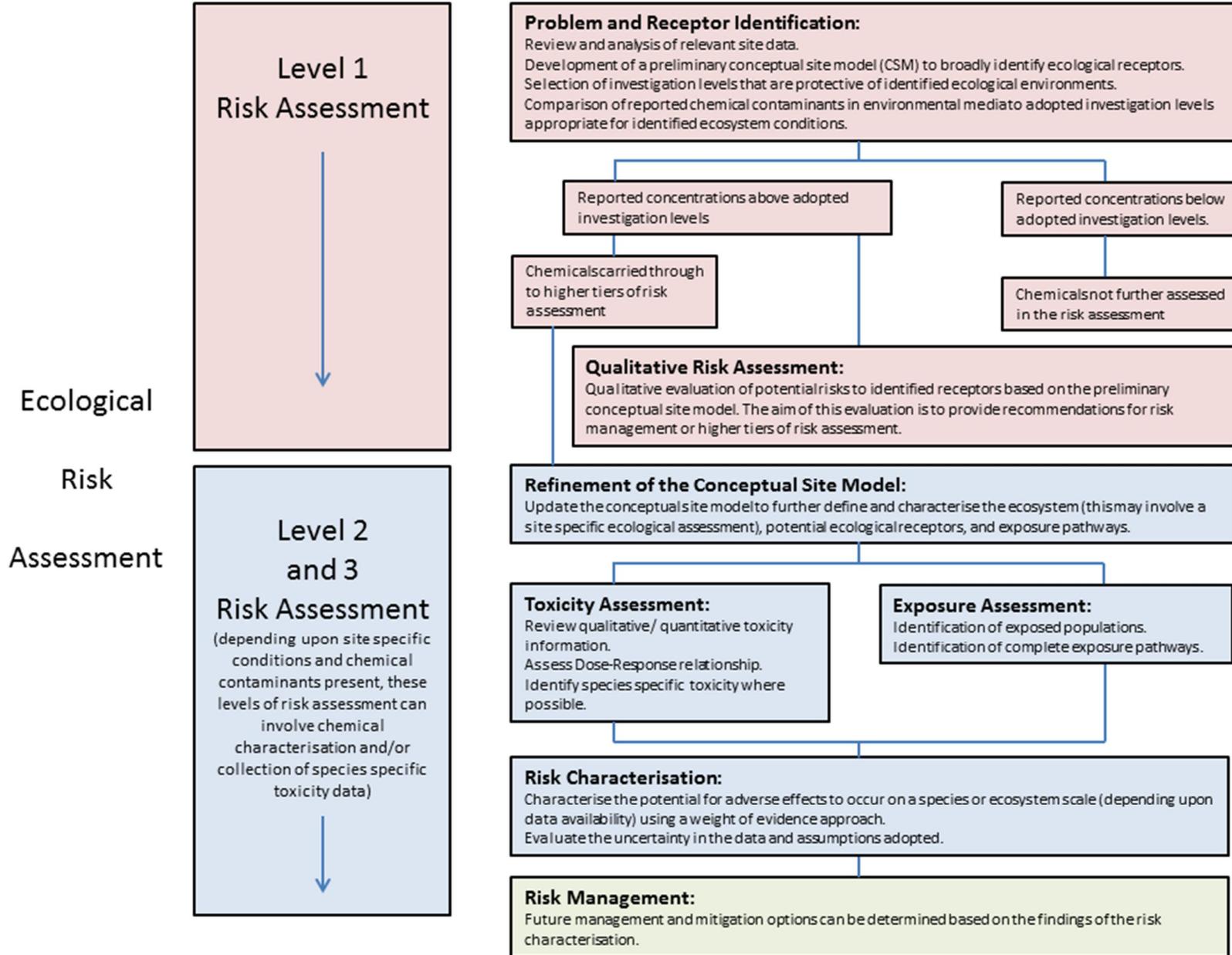
Appendix C

The Risk Assessment Process

The Risk Assessment Process



The Risk Assessment Process



Appendix D

RSL Calculator – Recreational User

Appendix D

**Site-specific
 Recreator Equation Inputs for Soil**

Variable	Value
TR (target cancer risk) unitless	0.00001
SA _{recsc} (skin surface area - child) cm ² /day	2366.7
SA _{recsa} (skin surface area - adult) cm ² /day	7170.8
SA ₀₋₂ (skin surface area - mutagenic) cm ² /day	1700
SA ₂₋₆ (skin surface area - mutagenic) cm ² /day	2700
SA ₆₋₁₆ (skin surface area - mutagenic) cm ² /day	6150
SA ₁₆₋₃₀ (skin surface area - mutagenic) cm ² /day	7900
SA _{recsa} (skin surface area - adult) cm ² /day	7170.8
THQ (target hazard quotient) unitless	1
LT (lifetime - recreator) year	70
IFS _{rec-adj} (age-adjusted soil ingestion factor) mg/kg	5824.012
DFS _{rec-adj} (age-adjusted soil dermal factor) mg/kg	558960.307
IFSM _{rec-adj} (mutagenic age-adjusted soil ingestion factor) mg/kg	30506.667
DFSM _{rec-adj} (mutagenic age-adjusted soil dermal factor) mg/kg	1546966.324
EF ₀₋₂ (exposure frequency) day/year	104
EF ₂₋₆ (exposure frequency) day/year	104
EF ₆₋₁₆ (exposure frequency) day/year	104
EF ₁₆₋₃₀ (exposure frequency) day/year	104
EF _{recsc} (exposure frequency - child) day/year	104
EF _{recsa} (exposure frequency - adult) day/year	104
EF _{recsa} (exposure frequency - adult) day/year	104
EF _{recs} (exposure frequency - recreator) day/year	104
IRS ₀₋₂ (soil intake rate) mg/day	100
IRS ₂₋₆ (soil intake rate) mg/day	100
IRS ₆₋₁₆ (soil intake rate) mg/day	100
IRS ₁₆₋₃₀ (soil intake rate) mg/day	60
IRS _{recsc} (soil intake rate - child) mg/day	100
IRS _{recsa} (soil intake rate - adult) mg/day	76.7
IRS _{recsa} (soil intake rate - adult) mg/day	76.7
ED ₀₋₂ (exposure duration) year	2
ED ₂₋₆ (exposure duration) year	4
ED ₆₋₁₆ (exposure duration) year	10
ED ₁₆₋₃₀ (exposure duration) year	14
ED _{recsc} (exposure duration - child) year	6
ED _{recsa} (exposure duration - adult) year	24
ED _{recsa} (exposure duration - adult) year	24
ED _{recs} (exposure duration - recreator) year	30
ET ₀₋₂ (exposure time) hr/day	2
ET ₂₋₆ (exposure time) hr/day	2
ET ₆₋₁₆ (exposure time) hr/day	2
ET ₁₆₋₃₀ (exposure time) hr/day	2
ET _{recsc} (exposure time - child) hr/day	2
ET _{recsa} (exposure time - adult) hr/day	2
ET _{recsa} (exposure time - adult) hr/day	2
ET _{recs} (exposure time - recreator) hr/day	2
BW ₀₋₂ (body weight) kg	10.5
BW ₂₋₆ (body weight) kg	25
BW ₆₋₁₆ (body weight) kg	70
BW ₁₆₋₃₀ (body weight) kg	70
BW _{recsc} (body weight - child) kg	20.2
BW _{recsa} (body weight - adult) kg	70
BW _{recsa} (body weight - adult) kg	70
AF ₀₋₂ (skin adherence factor) mg/cm ²	1.7
AF ₂₋₆ (skin adherence factor) mg/cm ²	1.7
AF ₆₋₁₆ (skin adherence factor) mg/cm ²	1.7
AF ₁₆₋₃₀ (skin adherence factor) mg/cm ²	1.7
AF _{recsc} (skin adherence factor - child) mg/cm ²	1.7
AF _{recsa} (skin adherence factor - adult) mg/cm ²	1.7
AF _{recsa} (skin adherence factor - adult) mg/cm ²	1.7
City (Climate Zone) PEF Selection	Default
A _s (acres) PEF Selection	0.5
Q/C _{wp} (g/m ² -s per kg/m ³) PEF Selection	93.77
PEF (particulate emission factor) m ³ /kg	1359344438
A (PEF Dispersion Constant)	16.2302
B (PEF Dispersion Constant)	18.7762
C (PEF Dispersion Constant)	216.108
V (fraction of vegetative cover) unitless	0.5
U _m (mean annual wind speed) m/s	4.69
U _t (equivalent threshold value)	11.32
F(x) (function dependant on U _m /U _t) unitless	0.194
City (Climate Zone) VF Selection	Default
A _s (acres) VF Selection	0.5
Q/C _{wp} (g/m ² -s per kg/m ³) VF Selection	68.18
foc (fraction organic carbon in soil) g/g	0.006
ρ _b (dry soil bulk density) g/cm ³	1.5
ρ _s (soil particle density) g/cm ³	2.65
θ _w (water-filled soil porosity) L _{water} /L _{soil}	0.15
T (exposure interval) s	950000000
A (VF Dispersion Constant)	11.911
B (VF Dispersion Constant)	18.4385
C (VF Dispersion Constant)	209.7845

Output generated 17JAN2013:00:10:46

Appendix D

**Site-specific
 Recreator Risk-Based Screening Levels (RSL) for Soil**
 ca=Cancer, nc=Noncancer, ca* (Where nc SL < 100 x ca SL),
 ca** (Where nc SL < 10 x ca SL),
 max=SL exceeds ceiling limit (see User's Guide), sat=SL exceeds csat

Chemical	CAS Number	SF (mg/kg-day)	SFO Ref	Inhalation Unit Risk (ug/m ³) ⁻¹	IUR Ref	Chronic RfD (mg/kg-day)	RfD Ref	Chronic RfC (mg/m ³)	RfC Ref	GIAB S	GIAB ABS	Volatilization Factor (m ³ /kg)	Soil Saturation Concentration (mg/kg)	Calculation Emission Factor (m ³ /kg)	Ingestion SL (mg/kg)	Dermal SL (mg/kg)	Inhalation SL (mg/kg)	Carcinogenic SL (mg/kg)	Ingestion SL (Child) (mg/kg)	Dermal SL (Child) (mg/kg)	Inhalation SL (Child) (mg/kg)	Noncarcinogenic SL (Child) (mg/kg)	Ingestion SL (Adult) (mg/kg)	Dermal SL (Adult) (mg/kg)	Inhalation SL (Adult) (mg/kg)	Noncarcinogenic SL (Adult) (mg/kg)	Screening Level (mg/kg)
Acetone	67-64-1	-	-	-	-	9.00E-01	I	-	-	-	-	1.47E+04	1.14E+05	1.36E+09	8.12E+01	-	1.20E+02	4.84E+01	2.84E+04	-	1.91E+07	6.17E+05	2.88E+06	-	1.91E+07	2.51E+06	6.17E+05 sat
Acetophenone	98-86-2	-	-	-	-	1.00E-01	I	-	-	-	-	6.43E+04	2.52E+03	1.36E+09	-	-	-	-	7.09E+04	-	-	7.09E+04	3.20E+05	-	-	3.20E+05	7.09E+04 sat
Acrylonitrile	107-13-1	5.40E-01	I	6.80E-05	I	4.00E-02	A	2.00E-03	I	1	-	8.27E+03	1.13E+04	1.36E+09	2.58E+00	2.69E-01	2.73E+05	2.44E-01	2.13E+01	5.29E+00	6.97E+02	6.80E+02	1.28E+05	-	6.97E+02	6.93E+02	4.84E+01 ca**
Aldrin	309-00-2	1.70E+01	I	4.90E-03	I	3.00E-05	I	-	-	-	0.1	-	-	1.36E+09	-	-	-	-	-	-	-	9.61E+01	6.05E+00	-	6.97E+02	5.69E+00	2.44E-01 ca**
Allyl Chloride	107-05-1	2.10E-02	C	6.00E-06	C	-	-	1.00E-03	I	1	-	1.70E+03	1.42E+03	1.36E+09	2.09E+03	-	2.79E+02	2.46E+02	-	-	7.18E+01	7.18E+01	-	-	7.18E+01	7.18E+01	7.18E+01 ca**
Aminobiphenyl, 4-	92-67-1	2.10E+01	C	6.00E-03	C	-	-	-	-	-	0.1	-	-	1.36E+09	2.09E+00	2.18E-01	2.23E+05	1.97E-01	-	-	-	-	-	-	-	-	1.97E-01 ca**
Aniline	62-53-3	5.70E-03	I	1.60E-06	C	7.00E-03	P	1.00E-03	I	1	0.1	-	-	1.36E+09	7.70E+03	8.02E+02	8.35E+08	7.26E+02	4.96E+03	1.23E+03	5.72E+07	9.88E+02	2.24E+04	1.41E+03	5.72E+07	1.33E+03	7.26E+02 ca**
Arsenic, Inorganic	7440-38-2	1.50E+00	I	4.30E-03	I	3.00E-04	I	1.50E-05	C	1	0.03	-	-	1.36E+09	2.92E+01	1.02E-01	3.11E+05	7.54E+00	2.13E+02	1.76E+02	8.59E+05	9.64E+01	9.61E+02	2.02E+02	8.59E+05	1.67E+02	7.54E+00 ca**
Benzene	71-43-2	5.50E-02	I	7.80E-06	I	4.00E-03	I	3.00E-02	I	1	-	3.81E+03	1.82E+03	1.36E+09	7.98E+02	-	4.80E+02	2.99E+02	2.84E+03	-	4.81E+03	1.78E+03	1.28E+04	-	4.81E+03	3.50E+03	2.99E+02 ca**
Bis(2-chloroethoxy)methane	111-91-1	-	-	-	-	3.00E-03	P	-	-	1	0.1	-	-	1.36E+09	-	-	-	-	2.13E+03	5.29E+02	-	4.23E+02	9.61E+03	6.05E+02	-	5.69E+02	4.23E+02 ca**
Bis(2-ethylhexyl)phthalate	117-81-7	1.40E-02	I	2.40E-06	C	2.00E-02	I	-	-	1	0.1	-	-	1.36E+09	3.13E+03	3.26E+02	5.57E+08	2.96E+02	1.42E+04	3.52E+03	-	2.82E+03	6.41E+04	4.03E+03	-	3.79E+03	2.96E+02 ca**
Bromobenzene	108-86-1	-	-	-	-	8.00E-03	I	6.00E-02	I	1	-	9.01E+03	6.79E+02	1.36E+09	-	-	-	-	5.67E+03	-	2.28E+04	4.54E+03	2.56E+04	-	2.28E+04	1.21E+04	4.54E+03 sat
Bromochloromethane	74-97-5	-	-	-	-	4.00E-02	X	4.00E-02	X	1	-	3.86E+03	4.04E+03	1.36E+09	-	-	-	-	2.13E+03	5.29E+02	-	4.23E+02	9.61E+03	6.05E+02	-	5.69E+02	4.23E+02 ca**
Bromodichloromethane	75-27-4	6.20E-02	I	3.70E-05	C	2.00E-02	I	-	-	1	-	4.27E+03	9.31E+02	1.36E+09	7.08E+02	-	1.13E+02	9.77E+01	1.42E+04	-	6.50E+03	6.50E+03	-	6.50E+03	6.50E+03	6.50E+03 sat	
Bromoform	75-25-2	7.90E-03	I	1.10E-06	I	2.00E-02	I	-	-	1	0.1	-	-	1.36E+09	5.55E+03	5.79E+02	1.21E+09	5.24E+02	1.42E+04	3.52E+03	-	2.82E+03	6.41E+04	4.03E+03	-	3.79E+03	5.24E+02 ca**
Bromomethane	74-83-9	-	-	-	-	1.40E-03	I	5.00E-03	I	1	-	1.50E+03	3.59E+03	1.36E+09	-	-	-	-	9.93E+02	-	3.17E+02	2.40E+02	4.48E+03	-	3.17E+02	2.40E+02 ca**	
Butyl Benzyl Phthalate	85-68-7	1.90E-03	P	-	-	2.00E-01	I	-	-	1	0.1	-	-	1.36E+09	2.31E+04	2.41E+03	-	2.18E+03	1.42E+05	3.52E+04	-	2.82E+04	6.41E+05	4.03E+04	-	3.79E+04	2.18E+03 ca**
Butylbenzene, n-	104-51-8	-	-	-	-	5.00E-02	P	-	-	1	-	8.77E+03	1.08E+02	1.36E+09	-	-	-	-	3.54E+04	-	-	3.54E+04	1.60E+05	-	-	1.60E+05	3.54E+04 sat
Cadmium (Diet)	7440-43-9	-	-	1.80E-03	I	1.00E-03	I	2.00E-05	C	0.025	0.001	-	-	1.36E+09	-	-	7.42E+05	7.42E+05	7.09E+02	4.41E+02	1.14E+06	2.72E+02	3.20E+03	5.04E+02	1.14E+06	4.35E+02	2.72E+02 max
Carbon Disulfide	75-15-0	-	-	-	-	1.00E-01	I	7.00E-01	I	1	-	1.26E+03	7.38E+02	1.36E+09	-	-	-	-	7.09E+04	-	3.70E+04	2.43E+04	3.20E+05	-	3.70E+04	3.32E+04	2.43E+04 sat
Carbon Tetrachloride	56-23-5	7.00E-02	I	6.00E-06	I	4.00E-03	I	1.00E-01	I	1	-	1.61E+03	4.58E+02	1.36E+09	6.27E+02	-	2.63E+02	1.85E+02	2.84E+03	-	6.77E+03	2.00E+03	1.28E+04	-	6.77E+03	1.85E+02 ca**	
Chlordane	12789-03-6	3.50E-01	I	1.00E-04	I	5.00E-04	I	7.00E-04	I	1	0.04	-	-	1.36E+09	1.25E+02	3.26E+01	1.34E+07	2.59E+01	3.54E+02	2.20E+02	4.01E+07	1.36E+02	1.60E+03	2.52E+02	4.01E+07	2.18E+02	2.59E+01 ca**
Chlorobenzene	108-90-7	-	-	-	-	2.00E-02	I	5.00E-02	P	1	-	6.94E+03	7.61E+02	1.36E+09	-	-	-	-	1.42E+04	-	1.46E+04	7.20E+03	6.41E+04	-	1.46E+04	1.19E+04	7.20E+03 sat
Chloroform	67-66-3	3.10E-02	C	2.30E-05	I	1.00E-02	I	9.77E-02	A	1	-	2.83E+03	2.54E+03	1.36E+09	1.42E+03	-	1.21E+02	1.11E+02	7.09E+03	-	1.16E+04	4.40E+03	3.20E+04	-	1.16E+04	8.53E+03	1.11E+02 ca**
Chloromethane	74-87-3	-	-	-	-	9.00E-02	I	9.00E-02	I	1	-	1.27E+03	1.32E+03	1.36E+09	-	-	-	-	5.67E+04	-	4.80E+03	4.80E+03	-	4.80E+03	4.80E+03	4.80E+03 sat	
Chloronaphthalene, Beta- (2-Chlorophenol, 2-	91-58-7	-	-	-	-	8.00E-02	I	-	-	1	-	8.60E+04	-	1.36E+09	-	-	-	-	5.67E+04	-	-	5.67E+04	2.56E+05	-	-	2.56E+05	5.67E+04 ca**
Chlorophenol, 2-	95-57-8	-	-	-	-	5.00E-03	I	-	-	1	-	1.34E+05	2.19E+04	1.36E+09	-	-	-	-	3.54E+03	-	-	3.54E+03	1.60E+04	-	-	1.60E+04	3.54E+03 ca**
Chlorotoluene, o-	95-49-8	-	-	-	-	2.00E-02	I	-	-	1	-	8.74E+03	9.07E+02	1.36E+09	-	-	-	-	1.42E+04	-	-	1.42E+04	6.41E+04	-	-	6.41E+04	1.42E+04 sat
Chlorotoluene, p-	106-43-4	-	-	-	-	2.00E-02	X	-	-	1	-	7.85E+03	2.53E+02	1.36E+09	-	-	-	-	1.42E+04	-	-	1.42E+04	6.41E+04	-	-	6.41E+04	1.42E+04 sat
Chlorpyrifos	2921-88-2	-	-	-	-	1.00E-03	A	-	-	1	0.1	-	-	1.36E+09	-	-	-	-	7.09E+02	1.76E+02	-	1.41E+02	3.20E+03	2.02E+02	-	1.90E+02	1.41E+02 ca**
Copper	7440-50-8	-	-	-	-	4.00E-02	H	-	-	1	-	-	-	1.36E+09	-	-	-	-	2.84E+04	-	-	2.84E+04	1.28E+05	-	-	1.28E+05	2.84E+04 ca**
Cresol, m-	108-39-4	-	-	-	-	5.00E-02	I	6.00E-01	C	1	0.1	-	-	1.36E+09	-	-	-	-	3.54E+04	8.81E+03	3.43E+10	7.06E+03	1.60E+05	1.01E+04	3.43E+10	9.48E+03	7.06E+03 ca**
Cresol, p-chloro-m-Cumene	59-50-7	-	-	-	-	1.00E-01	A	-	-	1	0.1	-	-	1.36E+09	-	-	-	-	7.09E+04	1.76E+04	-	1.41E+04	3.20E+05	2.02E+04	-	1.90E+04	1.41E+04 ca**
DDD	72-54-8	2.40E-01	I	6.90E-05	C	1.00E-01	I	4.00E-01	I	1	0.1	-	6.68E+03	2.68E+02	1.36E+09	1.83E+02	1.90E+01	1.94E+07	7.09E+04	-	1.13E+05	4.35E+04	3.20E+05	-	1.13E+05	8.33E+04	4.35E+04 sat
DDE, p,p'	72-55-9	3.40E-01	I	9.70E-05	C	-	-	-	-	1	0.1	-	-	1.36E+09	1.29E+02	1.34E+01	1.38E+07	1.22E+01	7.09E+04	-	-	-	3.20E+05	-	-	1.72E+01 ca**	
DDT	50-29-3	3.40E-01	I	9.70E-05	I	5.00E-04	I	-	-	1	0.03	-	-	1.36E+09	1.29E+02	4.48E+01	1.38E+07	3.33E+01	3.54E+02	2.94E+02	-	1.61E+02	1.60E+03	3.36E+02	-	2.78E+02	3.33E+01 ca**
Diazinon	333-41-5	-	-	-	-	7.00E-04	A	-	-	1	0.1	-	-	1.36E+09	1.29E+02	4.48E+01	1.38E+07	3.33E+01	4.96E+02	1.23E+02	-	9.88E+01	2.24E+03	1.41E+02	-	1.33E+02	9.88E+01 ca**
Dibromo-3-chloropropane, 1,2-	96-12-8	8.00E-01	P	6.00E-03	P	2.00E-04	P	2.00E-04	I	1	-	3.44E+04	9.79E+02	1.36E+09	1.05E+01	-	2.23E+00	1.84E+00	1.42E+02	-	2.90E+02	9.52E+01	6.41E+02	-	2.90E+02	2.00E+02	1.84E+00 ca**
Dibromochloromethane	124-48-1	8.40E-02	I	2.70E-05	C	2.00E-02	I	-	-	1	0.1	8.55E+03	8.02E+02	1.36E+09	5.22E+02	5.44E+01	3.11E+02	4.25E+01	1.42E+04	3.52E+03	-	2.82E+03	6.41E+04	4.03E+03	-	3.79E+03	4.25E+01 ca**
Dibromoethane, 1,2-	106-93-4	2.00E+00	I	6.00E-04	I	9.00E-03	I	9.00E-03	I	1	-	9.30E+03	1.34E+03	1.36E+09	2.19E+01	-	1.52E+01	8.99E+00	6.38E+03	-	3.5						

Appendix D

**Site-specific
 Recreator Risk-Based Screening Levels (RSL) for Soil**
 ca=Cancer, nc=Noncancer, ca* (Where nc SL < 100 x ca SL),
 ca** (Where nc SL < 10 x ca SL),
 max=SL exceeds ceiling limit (see User's Guide), sat=SL exceeds csat

Chemical	CAS Number	SF (mg/kg-day)	SFO Ref	Inhalation Unit Risk (ug/m ³) ⁻¹	IUR Ref	Chronic RfD (mg/kg-day)	RfD Ref	Chronic RfC (mg/m ³)	RfC Ref	GIAB S	GIAB ABS	Volatilization Factor	Soil Saturation Concentration (mg/kg)	Calculation Emission Factor (m ³ /kg)	Ingestion SL (mg/kg) TR=1.0E-5	Dermal SL (mg/kg) TR=1.0E-5	Inhalation SL (mg/kg) TR=1.0E-5	Carcinogenic c SL (mg/kg) TR=1.0E-5	Ingestion SL (Child) (mg/kg) HQ=1	Dermal SL (Child) (mg/kg) HQ=1	Inhalation SL (Child) (mg/kg) HQ=1	Noncarcinogenic c SL (Child) (mg/kg) HI=1	Ingestion SL (Adult) (mg/kg) HQ=1	Dermal SL (Adult) (mg/kg) HQ=1	Inhalation SL (Adult) (mg/kg) HQ=1	Noncarcinogenic ic SL (Adult) (mg/kg) HI=1	Screening Level (mg/kg)
Methidathion	950-37-8	-	-	-	-	1.00E-03	I	-	-	1	0.1	-	-	1.36E+09	-	-	-	-	7.09E+02	1.76E+02	-	1.41E+02	3.20E+03	2.02E+02	-	1.90E+02	1.41E+02 ca**
Methoxychlor	72-43-5	-	-	-	-	5.00E-03	I	-	-	1	0.1	-	-	1.36E+09	-	-	-	-	3.54E+03	8.81E+02	-	7.06E+02	1.60E+04	1.01E+03	-	9.48E+02	7.06E+02 ca**
Methyl Ethyl Ketone (2-Butanone)	78-93-3	-	-	-	-	6.00E-01	I	5.00E+00	I	1	-	1.31E+04	2.84E+04	1.36E+09	-	-	-	-	4.25E+05	-	2.76E+06	3.69E+05	1.92E+06	-	2.76E+06	1.13E+06	3.69E+05 sat
Methyl Isobutyl Ketone (4-methyl-2-Pentanone)	108-10-1	-	-	-	-	8.00E-02	H	3.00E+00	I	1	-	1.14E+04	3.36E+03	1.36E+09	-	-	-	-	5.67E+04	-	1.44E+06	5.46E+04	2.56E+05	-	1.44E+06	2.17E+05	5.46E+04 sat
Methyl Parathion	298-00-0	-	-	-	-	2.50E-04	I	-	-	1	0.1	-	-	1.36E+09	-	-	-	-	1.77E+02	4.41E+01	-	3.53E+01	8.01E+02	5.04E+01	-	4.74E+01	3.53E+01 sat
Methyl tert-Butyl Ether (MTBE)	1634-04-4	1.80E-03	C	2.60E-07	C	-	-	3.00E+00	I	1	-	5.28E+03	8.87E+03	1.36E+09	2.44E+04	-	1.99E+04	1.10E+04	-	-	6.66E+05	6.66E+05	-	-	6.66E+05	6.66E+05	1.10E+04 sat
Methylcholanthrene, 3-Methyl	56-49-5	2.20E+01	C	6.30E-03	C	-	-	-	-	1	0.1	-	-	1.36E+09	3.81E-01	7.51E-02	8.37E+04	6.27E-02	-	-	-	-	-	-	-	-	6.27E-02 ca**
Methylene Chloride	75-09-2	2.00E-03	I	1.00E-08	I	6.00E-03	I	6.00E-01	I	1	-	2.36E+03	3.32E+03	1.36E+09	4.19E+03	-	9.15E+04	4.00E+03	4.25E+03	-	5.96E+04	3.97E+03	1.92E+04	-	5.96E+04	1.45E+04	3.97E+03 sat
Mercury (elemental)	7439-97-6	-	-	-	-	-	-	3.00E-04	I	1	-	3.24E+03	3.13E+00	1.36E+09	-	-	-	-	-	-	4.10E+02	4.10E+02	-	-	4.10E+02	4.10E+02 sat	
Naphthylamine, 2-	91-59-8	1.80E+00	C	0.00E+00	C	-	-	-	-	1	0.1	-	-	1.36E+09	2.44E+01	2.54E+00	-	-	-	-	-	-	-	-	-	-	2.30E+00 ca**
Nickel Soluble Salts	7440-02-0	-	-	2.60E-04	C	2.00E-02	I	9.00E-05	A	0.04	-	-	-	1.36E+09	-	-	5.14E+06	5.14E+06	1.42E+04	-	5.15E+06	1.41E+04	6.41E+04	-	5.15E+06	6.33E+04	1.41E+04 ca**
Nitroaniline, 2-	88-74-4	-	-	-	-	1.00E-02	X	5.00E-05	X	1	0.1	-	-	1.36E+09	-	-	-	-	7.09E+03	1.76E+03	2.86E+06	1.41E+03	3.20E+04	2.02E+03	2.86E+06	1.89E+03	1.41E+03 max
Nitrobenzene	98-95-3	-	-	4.00E-05	I	2.00E-03	I	9.00E-03	I	1	-	7.88E+04	3.05E+03	1.36E+09	-	-	1.94E+03	1.94E+03	1.42E+03	-	2.99E+04	1.35E+03	6.41E+03	-	2.99E+04	5.27E+03	1.35E+03 ca**
Nitropropane, 2-	79-46-9	-	-	2.70E-03	H	-	-	2.00E-02	I	1	-	1.41E+04	4.86E+03	1.36E+09	-	-	5.15E+00	5.15E+00	-	-	1.19E+04	1.19E+04	-	-	1.19E+04	1.19E+04 sat	
Nitroso-di-N-butylamine, N-Nitroso	924-16-3	5.40E+00	I	1.60E-03	I	-	-	-	-	1	-	2.12E+05	-	1.36E+09	8.12E+00	-	1.30E+02	7.65E+00	-	-	-	-	-	-	-	-	7.65E+00 ca**
Nitroso-di-N-propylamine, N-Nitroso	621-64-7	7.00E+00	I	2.00E-03	C	-	-	-	-	1	0.1	-	-	1.36E+09	6.27E+00	6.53E-01	6.68E+05	5.91E-01	-	-	-	-	-	-	-	-	5.91E-01 ca**
Nitrosopiperidine [N-]	100-75-4	9.40E+00	C	2.70E-03	C	-	-	-	-	1	0.1	-	-	1.36E+09	4.67E+00	4.86E-01	4.95E+05	4.40E-01	-	-	-	-	-	-	-	-	4.40E-01 ca**
Octyl Phthalate, di-N-Parathion	117-84-0	-	-	1.20E-02	P	-	-	-	-	1	0.1	-	-	1.36E+09	-	-	-	-	8.51E+03	2.11E+03	-	1.69E+03	3.84E+04	2.42E+03	-	2.28E+03	1.69E+03 sat
Parathion	56-38-2	-	-	6.00E-03	H	-	-	-	-	1	0.1	-	-	1.36E+09	-	-	-	-	4.25E+03	1.06E+03	-	8.47E+02	1.92E+04	1.21E+03	-	1.14E+03	8.47E+02 sat
Pentachlorobenzene	608-93-5	-	-	8.00E-04	I	-	-	-	-	1	0.1	-	-	1.36E+09	-	-	-	-	5.67E+02	1.41E+02	-	1.13E+02	2.56E+03	1.61E+02	-	1.52E+02	1.13E+02 sat
Pentachloronitrobenzene	82-68-8	2.60E-01	H	-	-	3.00E-03	I	-	-	1	0.1	-	-	1.36E+09	1.69E+02	1.76E+01	-	1.59E+01	2.13E+03	5.29E+02	-	4.23E+02	9.61E+03	6.05E+02	-	5.69E+02	1.59E+02 ca**
Pentachlorophenol	87-86-5	4.00E-01	I	5.10E-06	C	5.00E-03	I	-	-	1	0.25	-	-	1.36E+09	1.10E+02	4.57E+00	2.62E+08	4.39E+00	3.54E+03	3.52E+02	-	3.21E+02	1.60E+04	4.03E+02	-	3.93E+02	4.39E+00 ca**
Phenol	108-95-2	-	-	3.00E-01	I	2.00E-01	C	1	0.1	-	-	-	-	1.36E+09	-	-	-	-	2.13E+05	5.29E+04	1.14E+10	4.23E+04	9.61E+05	6.05E+04	1.14E+10	5.69E+04	4.23E+04 ca**
Phorate	298-02-2	-	-	2.00E-04	H	-	-	-	-	1	0.1	-	-	1.36E+09	-	-	-	-	1.42E+02	3.52E+01	-	2.82E+01	6.41E+02	4.03E+01	-	3.79E+01	2.82E+01 ca**
Propyl benzene	103-65-1	-	-	1.00E-01	X	1.00E+00	X	1	0.1	1	0.1	7.53E+03	2.64E+02	1.36E+09	-	-	-	-	7.09E+04	1.76E+04	3.17E+05	1.35E+04	3.20E+05	2.02E+04	3.17E+05	1.79E+04	1.35E+04 sat
Aroclor 1016	12674-11-2	7.00E-02	S	2.00E-05	S	7.00E-05	I	-	-	1	0.14	-	-	1.36E+09	6.27E+02	4.66E+01	6.68E+07	4.34E+01	4.96E+01	8.81E+00	-	7.48E+00	2.24E+02	1.01E+01	-	9.64E+00	7.48E+00 max
Aroclor 1221	11104-28-2	2.00E+00	S	5.71E-04	S	-	-	-	-	1	0.14	9.16E+04	7.57E+02	1.36E+09	2.19E+01	1.63E+00	1.58E+02	1.50E+00	-	-	-	-	-	-	-	-	1.50E+00 ca**
Aroclor 1232	11141-16-5	2.00E+00	S	5.71E-04	S	-	-	-	-	1	0.14	9.16E+04	7.32E+01	1.36E+09	2.19E+01	1.63E+00	1.58E+02	1.50E+00	-	-	-	-	-	-	-	-	1.50E+00 ca**
Aroclor 1242	53469-21-9	2.00E+00	S	5.71E-04	S	-	-	-	-	1	0.14	-	-	1.36E+09	2.19E+01	1.63E+00	2.34E+06	1.52E+00	-	-	-	-	-	-	-	-	1.52E+00 ca**
Aroclor 1248	12672-29-6	2.00E+00	S	5.71E-04	S	-	-	-	-	1	0.14	-	-	1.36E+09	2.19E+01	1.63E+00	2.34E+06	1.52E+00	-	-	-	-	-	-	-	-	1.52E+00 ca**
Aroclor 1254	11097-69-1	2.00E+00	S	5.71E-04	S	2.00E-05	I	-	-	1	0.14	-	-	1.36E+09	2.19E+01	1.63E+00	2.34E+06	1.52E+00	1.42E+01	2.52E+00	-	2.14E+00	6.41E+01	2.88E+00	-	2.76E+00	1.52E+00 ca**
Aroclor 1260	11096-82-5	2.00E+00	S	5.71E-04	S	-	-	-	-	1	0.14	-	-	1.36E+09	2.19E+01	1.63E+00	2.34E+06	1.52E+00	-	-	-	-	-	-	-	-	1.52E+00 ca**
Polychlorinated Biphenyls (high risk)	1336-36-3	2.00E+00	I	5.71E-04	I	-	-	-	-	1	0.14	-	-	1.36E+09	2.19E+01	1.63E+00	2.34E+06	1.52E+00	-	-	-	-	-	-	-	-	1.52E+00 ca**
Acenaphthene	83-32-9	-	-	-	-	6.00E-02	I	-	-	1	0.13	1.51E+05	-	1.36E+09	-	-	-	-	4.25E+04	8.13E+03	-	6.83E+03	1.92E+05	9.30E+03	-	8.87E+03	6.83E+03 ca**
Anthracene	120-12-7	-	-	-	-	3.00E-01	I	-	-	1	0.13	5.63E+05	-	1.36E+09	-	-	-	-	2.13E+05	4.07E+04	-	3.41E+04	9.61E+05	4.65E+04	-	4.44E+04	3.41E+04 ca**
Benz[a]anthracene	56-55-3	7.30E-01	W	1.10E-04	C	-	-	-	-	1	0.13	-	-	1.36E+09	1.15E+01	1.74E+00	4.79E+06	1.51E+00	-	-	-	-	-	-	-	-	1.51E+00 ca**
Benzo[a]pyrene	50-32-8	7.30E+00	I	1.10E-03	C	-	-	-	-	1	0.13	-	-	1.36E+09	1.15E+00	1.74E-01	4.79E+05	1.51E-01	-	-	-	-	-	-	-	-	1.51E-01 ca**
Benzo[b]fluoranthene	205-99-2	7.30E-01	W	1.10E-04	C	-	-	-	-	1	0.13	-	-	1.36E+09	1.15E+01	1.74E+00	4.79E+06	1.51E+00	-	-	-	-	-	-	-	-	1.51E+00 ca**
Benzo[k]fluoranthene	207-08-9	7.30E-02	W	1.10E-04	C	-	-	-	-	1	0.13	-	-	1.36E+09	1.15E+02	1.74E+01	4.79E+06	1.51E+01	-	-	-	-	-	-	-	-	1.51E+01 ca**
Chrysene	218-01-9	7.30E-03	W	1.10E-05	C	-	-	-	-	1	0.13	-	-	1.36E+09	1.15E+03	1.74E+02	4.79E+07	1.51E+02	-	-	-	-	-	-	-	-	1.51E+02 ca**
Dibenz[a,h]anthracene	53-70-3	7.30E+00	W	1.20E-03	C	-	-	-	-	1	0.13	-	-	1.36E+09	1.15E+00	1.74E-01	4.39E+05	1.51E-01	-	-	-	-	-	-	-	-	1.51E-01 ca**
Fluoranthene	206-44-0	-	-	-	-	4.00E-02	I	-	-	1	0.13	-	-	1.36E+09	-	-	-	-	2.84E+04	5.42E+03	-	4.55E+03	1.28E+05	6.20E+03	-	5.91E+03	4.55E+03 ca**
Fluorene																											

Appendix E

Risk Estimates - On-Site Recreational User

Exposure Parameters:	Site-Specific Value		Justification	Value Used in Calculations				
	Adult	Child		Adult		Child		
General receptor parameters:								
	Units							
Body weight	kg	78	15	Adult value: male and female rounded average combined weight enHealth (2012b). Child value: average body weight of a 2-3 year old child enHealth (2012b).	78	bwa	15	bwc
Exposure duration	yr	35	6	95th percentile Australian adult residence enHealth (2012b)	35	eda	6	edc
Averaging time (carcinogens)	yr	70	70	enHealth (2012b)	70	atca	70	atcc
Averaging time (non-carcinogens)	yr	35	6	enHealth (2012b)	35	atnca	6	atncc
Incidental Soil Ingestion								
Daily soil ingestion rate	mg/day	60	100	Adult value: 95th percentile value for outside soil plus indoor dust. Child value: central tendency, outside soil plus indoor dust (enHealth, 2012).	60	irsa	100	irsc
Exposure frequency for soil ingestion	days/yr	52	52	Conservative assumption - based on 1 day per week per year	52	efsa	52	efsc
Fraction of daily soil intake from site	unitless	1	1	Assumes all soil ingestion is from the Site	1	fsa	1	fsc
Dermal Absorption of Soil								
Exposed skin surface area for soil contact	cm2	10500	4300	Adult value: Average value for adult male and female 95th percentile data forehead, hands, arms, lower legs and feet (enHealth, 2012). Child value: 95th percentile value for a 2-3yr old child for head, hands, arms, lower legs and feet (enHealth, 2012).	10500	sasa	4300	sasc
Soil to skin adherence factor	mg/cm2	0.5	0.5	enHealth (2012) default value for screening risk assessments	0.5	sada	0.5	sadc
Exposure frequency for dermal contact with soil	days/yr	52	52	Conservative assumption - based on 1 day per week per year	52	efdsa	52	efdsc
Indoor Inhalation								
Exposure time (indoor air)	hrs/day				8	etiaira	0	etiairc
Exposure frequency (indoor air)	days/yr				240	efiaira	0	efiairc
Particulate emission factor (indoor air)	m3/kg				1.02E+09	pefia	0	pefic
Outdoor Inhalation								
Exposure time (outdoor air)	hrs/day	2	2	Recommended value for 2-3 year old children (enHealth, 2012)	2	etoaira	2	etoairc
Exposure frequency (outdoor air)	days/yr	52	52	Conservative assumption - based on 1 day per week per year	52	efoaira	52	efoairc
Particulate emission factor (outdoor air)	m3/kg	1.36E+09	1.36E+09	USEPA (2002) Supplemental guidance for developing soil screening levels for Superfund sites, OSWER 9355.4-24, Soil Waste and Emergency Response, United States Environmental Protection Agency, Washington, DC.	1.36E+09	pefoa	1.36E+09	pefoc
Potable Water Ingestion								
Potable water intake rate	L/day				2	irdwa	0	irdwc
Exposure frequency for potable water intake	days/yr				240	efdwa	0	efdwc
Incidental Water Ingestion								
Incidental ingestion rate	L/day				0.005	irbwa	0	irbwc
Exposure frequency for incidental water ingestion	days/yr				0	efbwa	0	efbwc
Dermal Contact with Water								
Exposed skin surface for water contact	cm2				0	sawa	0	sawc
Exposure time for dermal water contact	hr/day				0	etbwa	0	etbwc
Exposure frequency for dermal water contact	days/yr				0	efdbwa	0	efdbwc
Vapour Inhalation Shower/ Sprinkler								
Exposure frequency	days/yr				0	efairscha	0	efairschc
Exposure time	min/ day				0	etairsha	0	etairshc
Inhalation rate	m3/hr				0.66	irshwa	0.454	irshwc
Inhalation Absorption Adjustment Factor	mg/mg				1	iaafa	1	iaafc
Lung retention factor	unitless				1	lrfa	1	lrfc
Ingestion of Home Grown Produce								
Proportion of homegrown produce ingested					0	prphga	0	prphgc
Exposure frequency	days/year				0	efhgpa	0	efhgpc
Inhalation of Vapours from Flowing Water								
Exposure time	hrs/day				0	etwfa	0	etwfc
Exposure frequency	days/year				0	efwfa	0	efwfc
Ingestion of Fish								
Daily fish ingestion rate	mg/day				0	irfa	0	irfc
Exposure frequency for fish ingestion	days/yr				0	effa	0	effc
Fraction of daily fish intake from site	unitless				0	ffa	0	ffc

Vapour Modelling - Soil to Outdoor Air & Direct Contact
Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
Darling Drive, Darling Harbour, Sydney NSW
On-Site Recreational User (version D) Scenario

[Back to User Input Sheet](#)

A. Model Input Parameters

Parameter Definition	Units	Default Value	Notes	Site-Specific Value	Label	Adopted Value for Model	Justification for Site-specific value, if applicable
Depth to subsurface soil sources (below building, ground surface or trench)	cm	100			LS	15	Autocalculates from layer thicknesses
<i>Vadose Zone Layer 1 (soil type where source is)</i>							
Thickness	cm	100		15	HV	15	Assumes impact is at surface
SCS Soil Type:				Sand and gravel (<12% fines)			Conservative assumption
Fraction of organic carbon	unitless	0.01		0.002	OC	0.002	
Soil bulk density	g/cm3	1.7		1.66	sbd	1.66	
Air-filled porosity (volumetric)	cm3/cm3	0.26		0.321	VACS	0.321	
Water-filled porosity (volumetric)	cm3/cm3	0.12		0.054	wvcvz	0.054	
Total soil porosity	cm3/cm3	0.38		0.375	TPOR	0.375	
Vapour phase source partitioning adjustment	unitless	1		1	VPPA	1	Not considered within vapour modelling
Vadose zone biodegradation adjustment	unitless	1		1	BioA	1	Not considered within vapour modelling
<i>Outdoor Air Characteristics</i>							
Wind speed in outdoor mixing zone (ambient air or trench, as appropriate)	cm/s	225		377.78	windsp	377.78	Based on annual average of 9am and 3pm at Sydney (Observation Hill) weather station
Width of source area parallel to wind or groundwater flow direction	cm	4500		1500	WSA	1500	ASTM 1739-95(2010)e1
Ambient air mixing zone height	cm	200		200	AAMZH	200	ASTM 1739-95(2010)e1

B. Chemical-Specific Fate and Transport Parameters

CHEMICAL	Koc (cm ³ /g)	Kd (cm ³ /g)	H' (cc-H ₂ O / cc-air)	S (mg/l-water)	D ^{air} (cm ² /s)	D ^{wat} (cm ² /s)	MW (g/mol)	VP (mmHg)	Volatile?
TPH C10-C14 aliphatic	1.12E+06	2.24E+03	6.26E+01	9.99E-02	1.00E-01	1.00E-05	1.70E+02	1.16E+00	Y
TPH C10-C14 aromatic	3.55E+03	7.10E+00	1.41E-01	2.53E+01	1.00E-01	1.00E-05	1.36E+02	1.16E+00	Y
TPH C15-C28 aliphatic	6.31E+08	1.26E+06	8.27E+01	1.11E-04	1.00E-01	1.00E-05	2.60E+02	5.93E-03	N
TPH C15-C28 aromatic	4.47E+04	8.93E+01	4.90E-03	1.06E+00	1.00E-01	1.00E-05	2.09E+02	5.51E-03	N
TPH C29-C36 aliphatic	6.31E+08	1.26E+06	8.50E+01	2.50E-06	1.00E-01	1.00E-05	2.70E+02	8.36E-04	N
TPH C29-C36 aromatic	1.26E+05	2.52E+02	1.70E-05	6.60E-03	1.00E-01	1.00E-05	2.40E+02	3.34E-07	N
Acenaphthene	5.03E+03	1.01E+01	7.52E-03	3.90E+00	5.06E-02	8.33E-06	1.54E+02	2.15E-03	N
Acenaphthylene	5.03E+03	1.01E+01	4.66E-03	1.61E+01	4.50E-02	6.98E-06	1.52E+02	6.68E-03	N
Anthracene	1.64E+04	3.28E+01	2.27E-03	4.34E-02	3.90E-02	7.85E-06	1.78E+02	6.53E-06	N
Benzo(a)anthracene	1.77E+05	3.54E+02	4.91E-04	9.40E-03	5.10E-02	9.00E-06	2.28E+02	2.10E-07	N
Benzo(a)pyrene	5.87E+05	1.17E+03	1.87E-05	1.62E-03	4.30E-02	9.00E-06	2.52E+02	5.49E-09	N
Benzo(b)fluoranthene	5.99E+05	1.20E+03	2.69E-05	1.50E-03	2.26E-02	5.56E-06	2.52E+02	5.00E-07	N
Benzo(k)fluoranthene	5.87E+05	1.17E+03	2.39E-05	8.00E-04	2.26E-02	5.56E-06	2.52E+02	9.65E-10	N
Benzo(g,h,i)perylene	1.95E+06	3.90E+03	1.35E-05	2.60E-04	4.90E-02	5.56E-06	2.76E+02	1.00E-10	N
Chrysene	1.81E+05	3.62E+02	2.14E-04	2.00E-03	2.48E-02	6.21E-06	2.28E+02	6.23E-09	N
Dibenz(a,h)anthracene	1.91E+06	3.82E+03	5.76E-06	2.49E-03	2.00E-02	5.24E-06	2.78E+02	9.55E-10	N
Fluoranthene	5.55E+04	1.11E+02	3.62E-04	2.60E-01	3.02E-02	6.35E-06	2.02E+02	9.22E-06	N
Fluorene	9.16E+03	1.83E+01	3.93E-03	1.83E+01	4.40E-02	7.89E-06	1.66E+02	6.00E-04	N
Indeno(1,2,3-cd)pyrene	1.95E+06	3.90E+03	1.42E-05	1.90E-04	2.30E-02	4.41E-06	2.76E+02	1.25E-10	N
Naphthalene	1.54E+03	3.08E+00	1.80E-02	3.10E+01	6.05E-02	8.38E-06	1.28E+02	8.50E-02	N
Phenanthrene	1.67E+04	3.34E+01	1.73E-03	1.15E+00	3.45E-02	6.69E-06	1.78E+02	1.21E-04	N
Pyrene	5.43E+04	1.09E+02	4.87E-04	1.35E-01	2.78E-02	7.25E-06	2.02E+02	4.50E-06	N

Definition of Parameters

Koc	Organic carbon partition coefficient	D ^{air}	Diffusion coefficient in air
Kd	Soil-water partition coefficient	D ^{wat}	Diffusion coefficient in water
H'	Dimensionless Henry's Law Constant	MW	Molecular weight
S	Solubility	VP	Vapour pressure

Vapour Modelling - Soil to Outdoor Air & Direct Contact
Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
Darling Drive, Darling Harbour, Sydney NSW
On-Site Recreational User (version D) Scenario

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C. Chemical-Specific Diffusion Coefficients

CHEMICAL	D _{s1} (cm ² /s)	D _{s2} (cm ² /s)	D _{s3} (cm ² /s)	D _{slot} (cm ² /s)	D _{crack} (cm ² /s)	C _{sat} (mg/kg)	Convective Factor (unitless)	D _a (cm ² /s)	M _{excav} (g)
TPH C10-C14 aliphatic	1.62E-02	-	-	1.62E-02	7.80E-03	2.25E+02	1.57E+01	2.69E-04	6.22E+05
TPH C10-C14 aromatic	1.62E-02	-	-	1.62E-02	7.80E-03	1.81E+02	1.57E+01	1.91E-04	5.24E+05
TPH C15-C28 aliphatic	NV	NV	NV	NV	NV	1.40E+02	NV	NV	NV
TPH C15-C28 aromatic	NV	NV	NV	NV	NV	9.47E+01	NV	NV	NV
TPH C29-C36 aliphatic	NV	NV	NV	NV	NV	3.16E+00	NV	NV	NV
TPH C29-C36 aromatic	NV	NV	NV	NV	NV	1.66E+00	NV	NV	NV
Acenaphthene	NV	NV	NV	NV	NV	3.94E+01	NV	NV	NV
Acenaphthylene	NV	NV	NV	NV	NV	1.63E+02	NV	NV	NV
Anthracene	NV	NV	NV	NV	NV	1.42E+00	NV	NV	NV
Benz(a)anthracene	NV	NV	NV	NV	NV	3.33E+00	NV	NV	NV
Benzo(a)pyrene	NV	NV	NV	NV	NV	1.90E+00	NV	NV	NV
Benzo(b)fluoranthene	NV	NV	NV	NV	NV	1.80E+00	NV	NV	NV
Benzo(k)fluoranthene	NV	NV	NV	NV	NV	9.39E-01	NV	NV	NV
Benzo(g,h,i)perylene	NV	NV	NV	NV	NV	1.01E+00	NV	NV	NV
Chrysene	NV	NV	NV	NV	NV	7.24E-01	NV	NV	NV
Dibenz(a,h)anthracene	NV	NV	NV	NV	NV	9.51E+00	NV	NV	NV
Fluoranthene	NV	NV	NV	NV	NV	2.89E+01	NV	NV	NV
Fluorene	NV	NV	NV	NV	NV	3.10E+01	NV	NV	NV
Indeno(1,2,3-cd)pyrene	NV	NV	NV	NV	NV	7.41E-01	NV	NV	NV
Naphthalene	NV	NV	NV	NV	NV	9.66E+01	NV	NV	NV
Phenanthrene	NV	NV	NV	NV	NV	3.84E+01	NV	NV	NV
Pyrene	NV	NV	NV	NV	NV	1.47E+01	NV	NV	NV

Definition of Parameters

D_s Effective diffusion coefficient in soil based on vapor-phase concentration
D_{crack} Effective diffusion coefficient through foundation cracks
C_{sat} Soil concentration at which dissolved pore-water and vapor phases become saturated

D_a = Apparent diffusivity (for construction scenario Volatilisation Factor)
M_{excav} = Cumulative unit mass emitted from excavation (Eq E-13 in USEPA, 2002).

D. Chemical-Specific Volatilisation Factors

CHEMICAL	VF _{as1} (mg/m ³ -air / mg/kg-soil)	VF _{as2} (mg/m ³ -air / mg/kg-soil)	VF _{p (indoor)} (mg/m ³ -air / mg/kg-soil)	VF _{p (outdoor)} (mg/m ³ -air / mg/kg-soil)	VF _{samb} (mg/m ³ -air / mg/kg-soil)	VF _{seep} (mg/m ³ -air / mg/kg-soil)
TPH C10-C14 aliphatic	4.44E-05	1.74E-05	9.80E-10	7.35E-10	5.94E-04	1.02E-02
TPH C10-C14 aromatic	3.75E-05	1.74E-05	9.80E-10	7.35E-10	4.22E-04	7.24E-03
TPH C15-C28 aliphatic	NV	NV	9.80E-10	7.35E-10	NV	NV
TPH C15-C28 aromatic	NV	NV	9.80E-10	7.35E-10	NV	NV
TPH C29-C36 aliphatic	NV	NV	9.80E-10	7.35E-10	NV	NV
TPH C29-C36 aromatic	NV	NV	9.80E-10	7.35E-10	NV	NV
Acenaphthene	NV	NV	9.80E-10	7.35E-10	NV	NV
Acenaphthylene	NV	NV	9.80E-10	7.35E-10	NV	NV
Anthracene	NV	NV	9.80E-10	7.35E-10	NV	NV
Benz(a)anthracene	NV	NV	9.80E-10	7.35E-10	NV	NV
Benzo(a)pyrene	NV	NV	9.80E-10	7.35E-10	NV	NV
Benzo(b)fluoranthene	NV	NV	9.80E-10	7.35E-10	NV	NV
Benzo(k)fluoranthene	NV	NV	9.80E-10	7.35E-10	NV	NV
Benzo(g,h,i)perylene	NV	NV	9.80E-10	7.35E-10	NV	NV
Chrysene	NV	NV	9.80E-10	7.35E-10	NV	NV
Dibenz(a,h)anthracene	NV	NV	9.80E-10	7.35E-10	NV	NV
Fluoranthene	NV	NV	9.80E-10	7.35E-10	NV	NV
Fluorene	NV	NV	9.80E-10	7.35E-10	NV	NV
Indeno(1,2,3-cd)pyrene	NV	NV	9.80E-10	7.35E-10	NV	NV
Naphthalene	NV	NV	9.80E-10	7.35E-10	NV	NV
Phenanthrene	NV	NV	9.80E-10	7.35E-10	NV	NV
Pyrene	NV	NV	9.80E-10	7.35E-10	NV	NV

Definition of Parameters

VF_{as} Volatilization factor from surficial soils to ambient air (vapors) - use lower of two values
VF_p Volatilization factor from surficial soils to ambient air (particulates)
VF_{samb} Volatilization factor from subsurface soils to ambient air
VF_{seep} Volatilization factor from soil to enclosed-space vapors

Health Risk Calculations - Incidental Soil Ingestion
Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
Darling Drive, Darling Harbour, Sydney NSW
On-Site Recreational User (version D) Scenario

Chemical	Soil Concentration (mg/kg)	Oral Soil Bioavailability Factor (unitless)	Threshold Intake and Risk Calculations							Non-Threshold Intake and Risk Calculations					
			Oral RfD (mg/kg/day)	Adult Intake Factor (threshold) (kg/kg/day)	Adult Intake (threshold) (mg/kg/day)	Hazard Index (Adult) (unitless)	Child Intake Factor (threshold) (kg/kg/day)	Child Intake (threshold) (mg/kg/day)	Hazard Index (Child) (unitless)	Oral CSF (mg/kg/day) ⁻¹	Adult Intake Factor (non-threshold) (kg/kg/day)	Child Intake Factor (non-threshold) (kg/kg/day)	Lifetime Intake Factor (non-threshold) (kg/kg/day)	Lifetime Intake (non-threshold) (mg/kg/day)	Lifetime Excess Cancer Risk (unitless)
TPH C10-C14 aliphatic	410	1.00E+00	1.00E-01	1.10E-07	4.49E-05	4.49E-04	9.50E-07	3.89E-04	3.89E-03	-	-	-	-	-	-
TPH C10-C14 aromatic	410	1.00E+00	4.00E-02	1.10E-07	4.49E-05	1.12E-03	9.50E-07	3.89E-04	9.74E-03	-	-	-	-	-	-
TPH C15-C28 aliphatic	2500	1.00E+00	2.00E+00	1.10E-07	2.74E-04	1.37E-04	9.50E-07	2.37E-03	1.19E-03	-	-	-	-	-	-
TPH C15-C28 aromatic	2500	1.00E+00	3.00E-02	1.10E-07	2.74E-04	9.13E-03	9.50E-07	2.37E-03	7.91E-02	-	-	-	-	-	-
TPH C29-C36 aliphatic	1600	1.00E+00	2.00E+00	1.10E-07	1.75E-04	8.77E-05	9.50E-07	1.52E-03	7.60E-04	-	-	-	-	-	-
TPH C29-C36 aromatic	1600	1.00E+00	3.00E-02	1.10E-07	1.75E-04	5.84E-03	9.50E-07	1.52E-03	5.07E-02	-	-	-	-	-	-
Acenaphthene	0.6	1.00E+00	6.00E-02	1.10E-07	6.58E-08	1.10E-06	9.50E-07	5.70E-07	9.50E-06	-	-	-	-	-	-
Acenaphthylene	1.3	1.00E+00	6.00E-02	1.10E-07	1.42E-07	2.37E-06	9.50E-07	1.23E-06	2.06E-05	-	-	-	-	-	-
Anthracene	3.4	1.00E+00	3.00E-01	1.10E-07	3.73E-07	1.24E-06	9.50E-07	3.23E-06	1.08E-05	-	-	-	-	-	-
Benz(a)anthracene	39	1.00E+00	-	-	-	-	-	-	-	4.30E-02	5.48E-08	8.14E-08	1.36E-07	5.31E-06	2.28E-07
Benzo(a)pyrene	6.5	1.00E+00	-	-	-	-	-	-	-	4.30E-01	5.48E-08	8.14E-08	1.36E-07	8.85E-07	3.81E-07
Benzo(b)fluoranthene	9.3	1.00E+00	-	-	-	-	-	-	-	4.30E-02	5.48E-08	8.14E-08	1.36E-07	1.27E-06	5.45E-08
Benzo(k)fluoranthene	62	1.00E+00	-	-	-	-	-	-	-	4.30E-02	5.48E-08	8.14E-08	1.36E-07	8.44E-06	3.63E-07
Benzo(g,h,i)perylene	3.9	1.00E+00	-	-	-	-	-	-	-	4.30E-03	5.48E-08	8.14E-08	1.36E-07	5.31E-07	2.28E-09
Chrysene	6	1.00E+00	--	-	-	-	-	-	-	4.30E-03	5.48E-08	8.14E-08	1.36E-07	8.17E-07	3.51E-09
Dibenz(a,h)anthracene	1	1.00E+00	--	-	-	-	-	-	-	4.30E-01	5.48E-08	8.14E-08	1.36E-07	1.36E-07	5.86E-08
Fluoranthene	94	1.00E+00	4.00E-02	1.10E-07	1.03E-05	2.58E-04	9.50E-07	8.93E-05	2.23E-03	-	-	-	-	-	-
Fluorene	1.8	1.00E+00	4.00E-02	1.10E-07	1.97E-07	4.93E-06	9.50E-07	1.71E-06	4.27E-05	-	-	-	-	-	-
Indeno(1,2,3-cd)pyrene	3.5	1.00E+00	--	-	-	-	-	-	-	4.30E-02	5.48E-08	8.14E-08	1.36E-07	4.77E-07	2.05E-08
Naphthalene	3.6	1.00E+00	2.00E-02	1.10E-07	3.95E-07	1.97E-05	9.50E-07	3.42E-06	1.71E-04	-	-	-	-	-	-
Phenanthrene	51	1.00E+00	4.00E-02	1.10E-07	5.59E-06	1.40E-04	9.50E-07	4.84E-05	1.21E-03	-	-	-	-	-	-
Pyrene	84	1.00E+00	3.00E-02	1.10E-07	9.21E-06	3.07E-04	9.50E-07	7.98E-05	2.66E-03	-	-	-	-	-	-
TOTAL						1.75E-02			1.52E-01						1.11E-06

Health Risk Calculations - Inhalation of Soil-Derived Dust (Particulates) in Outdoor Air
 Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
 Darling Drive, Darling Harbour, Sydney NSW
 On-Site Recreational User (version D) Scenario

Chemical	Soil Concentration (mg/kg)	Particulate Concentration in Outdoor Air (From Surface Soil) (m3/kg)	Threshold Intake and Risk Calculations							Non-Threshold Intake and Risk Calculations					
			Inhalation RfC (adjusted for background exposure) (mg/m3)	Adult Exposure Factor (threshold) (kg/m3)	Adult Exposure Adjusted Air Concentration (threshold) (mg/m3)	Hazard Index (Adult) (unitless)	Child Exposure Factor (threshold) (kg/m3)	Child Exposure Adjusted Air Concentration (threshold) (mg/m3)	Hazard Index (Child) (unitless)	Inhalation Unit Risk (ug/m3)-1	Adult Exposure Factor (non-threshold) (kg/m3)	Child Exposure Factor (non-threshold) (kg/m3)	Lifetime Exposure Factor (non-threshold) (kg/m3)	Lifetime Exposure Adjusted Air Concentration (non-threshold) (mg/m3)	Lifetime Excess Cancer Risk (unitless)
			(mg/m3)	(kg/m3)	(mg/m3)	(unitless)	(kg/m3)	(mg/m3)	(unitless)	(ug/m3)-1	(kg/m3)	(kg/m3)	(kg/m3)	(mg/m3)	(unitless)
TPH C10-C14 aliphatic	410	7.35E-10	9.00E-01	8.73E-12	3.58E-09	3.98E-09	8.73E-12	3.58E-09	3.98E-09	-	-	-	-	-	-
TPH C10-C14 aromatic	410	7.35E-10	1.80E-01	8.73E-12	3.58E-09	1.99E-08	8.73E-12	3.58E-09	1.99E-08	-	-	-	-	-	-
TPH C15-C28 aliphatic	2500	7.35E-10	6.30E+00	8.73E-12	2.18E-08	3.46E-09	8.73E-12	2.18E-08	3.46E-09	-	-	-	-	-	-
TPH C15-C28 aromatic	2500	7.35E-10	9.45E-02	8.73E-12	2.18E-08	2.31E-07	8.73E-12	2.18E-08	2.31E-07	-	-	-	-	-	-
TPH C29-C36 aliphatic	1600	7.35E-10	6.30E+00	8.73E-12	1.40E-08	2.22E-09	8.73E-12	1.40E-08	2.22E-09	-	-	-	-	-	-
TPH C29-C36 aromatic	1600	7.35E-10	9.45E-02	8.73E-12	1.40E-08	1.48E-07	8.73E-12	1.40E-08	1.48E-07	-	-	-	-	-	-
Acenaphthene	0.6	7.35E-10	2.10E-01	8.73E-12	5.24E-12	2.49E-11	8.73E-12	5.24E-12	2.49E-11	-	-	-	-	-	-
Acenaphthylene	1.3	7.35E-10	2.10E-01	8.73E-12	1.13E-11	5.40E-11	8.73E-12	1.13E-11	5.40E-11	-	-	-	-	-	-
Anthracene	3.4	7.35E-10	1.05E+00	8.73E-12	2.97E-11	2.83E-11	8.73E-12	2.97E-11	2.83E-11	-	-	-	-	-	-
Benz(a)anthracene	39	7.35E-10	-	-	-	-	-	-	-	8.70E-03	4.36E-12	7.48E-13	5.11E-12	1.99E-10	1.73E-09
Benzo(a)pyrene	6.5	7.35E-10	-	-	-	-	-	-	-	8.70E-02	4.36E-12	7.48E-13	5.11E-12	3.32E-11	2.89E-09
Benzo(b)fluoranthene	9.3	7.35E-10	-	-	-	-	-	-	-	8.70E-03	4.36E-12	7.48E-13	5.11E-12	4.76E-11	4.14E-10
Benzo(k)fluoranthene	62	7.35E-10	-	-	-	-	-	-	-	8.70E-03	4.36E-12	7.48E-13	5.11E-12	3.17E-10	2.76E-09
Benzo(g,h,i)perylene	3.9	7.35E-10	-	-	-	-	-	-	-	8.70E-04	4.36E-12	7.48E-13	5.11E-12	1.99E-11	1.73E-11
Chrysene	6	7.35E-10	-	-	-	-	-	-	-	8.70E-04	4.36E-12	7.48E-13	5.11E-12	3.07E-11	2.67E-11
Dibenz(a,h)anthracene	1	7.35E-10	-	-	-	-	-	-	-	8.70E-02	4.36E-12	7.48E-13	5.11E-12	5.11E-12	4.45E-10
Fluoranthene	94	7.35E-10	1.40E-01	8.73E-12	8.21E-10	5.86E-09	8.73E-12	8.21E-10	5.86E-09	-	-	-	-	-	-
Fluorene	1.8	7.35E-10	1.40E-01	8.73E-12	1.57E-11	1.12E-10	8.73E-12	1.57E-11	1.12E-10	-	-	-	-	-	-
Indeno(1,2,3-cd)pyrene	3.5	7.35E-10	-	-	-	-	-	-	-	8.70E-03	4.36E-12	7.48E-13	5.11E-12	1.79E-11	1.56E-10
Naphthalene	3.6	7.35E-10	3.70E-03	8.73E-12	3.14E-11	8.49E-09	8.73E-12	3.14E-11	8.49E-09	-	-	-	-	-	-
Phenanthrene	51	7.35E-10	1.40E-01	8.73E-12	4.45E-10	3.18E-09	8.73E-12	4.45E-10	3.18E-09	-	-	-	-	-	-
Pyrene	84	7.35E-10	1.05E-01	8.73E-12	7.33E-10	6.98E-09	8.73E-12	7.33E-10	6.98E-09	-	-	-	-	-	-
TOTAL						4.33E-07			4.33E-07						8.44E-09

Health Risk Calculations - Inhalation of Surface Soil-Derived Vapours in Outdoor Air
 Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
 Darling Drive, Darling Harbour, Sydney NSW
 On-Site Recreational User (version D) Scenario

Chemical	Soil Conc. (mg/kg)	Csat (mg/kg)	Volatilisation Factor from Subsurface Soil to Outdoor Air [(mg/m3)/(mg/kg)]	Vapour Concentration in Outdoor Air (From Surface Soil) (mg/m3)	Threshold Intake and Risk Calculations						
					Inhalation RfC (adjusted for background exposure) (mg/m3)	Adult Exposure Factor (threshold) (kg/m3)	Adult Exposure Adjusted Air Concentration (threshold) (mg/m3)	Hazard Index (Adult) (unitless)	Child Exposure Factor (threshold) (kg/m3)	Child Exposure Adjusted Air Concentration (threshold) (mg/m3)	Hazard Index (Child) (unitless)
TPH C10-C14 aliphatic	410	2.25E+02	1.74E-05	3.93E-03	9.00E-01	2.07E-07	4.66E-05	5.18E-05	2.07E-07	4.66E-05	5.18E-05
TPH C10-C14 aromatic	410	1.81E+02	1.74E-05	3.15E-03	1.80E-01	2.07E-07	3.74E-05	2.08E-04	2.07E-07	3.74E-05	2.08E-04
TPH C15-C28 aliphatic	2500	1.40E+02	NV	-	6.30E+00	-	-	-	-	-	-
TPH C15-C28 aromatic	2500	9.47E+01	NV	-	9.45E-02	-	-	-	-	-	-
TPH C29-C36 aliphatic	1600	3.16E+00	NV	-	6.30E+00	-	-	-	-	-	-
TPH C29-C36 aromatic	1600	1.66E+00	NV	-	9.45E-02	-	-	-	-	-	-
Acenaphthene	0.6	3.94E+01	NV	-	2.10E-01	-	-	-	-	-	-
Acenaphthylene	1.3	1.63E+02	NV	-	2.10E-01	-	-	-	-	-	-
Anthracene	3.4	1.42E+00	NV	-	1.05E+00	-	-	-	-	-	-
Benz(a)anthracene	39	3.33E+00	NV	-	-	-	-	-	-	-	-
Benzo(a)pyrene	6.5	1.90E+00	NV	-	-	-	-	-	-	-	-
Benzo(b)fluoranthene	9.3	1.80E+00	NV	-	-	-	-	-	-	-	-
Benzo(k)fluoranthene	62	9.39E-01	NV	-	-	-	-	-	-	-	-
Benzo(g,h,i)perylene	3.9	1.01E+00	NV	-	-	-	-	-	-	-	-
Chrysene	6	7.24E-01	NV	-	-	-	-	-	-	-	-
Dibenz(a,h)anthracene	1	9.51E+00	NV	-	-	-	-	-	-	-	-
Fluoranthene	94	2.89E+01	NV	-	1.40E-01	-	-	-	-	-	-
Fluorene	1.8	3.10E+01	NV	-	1.40E-01	-	-	-	-	-	-
Indeno(1,2,3-cd)pyrene	3.5	7.41E-01	NV	-	-	-	-	-	-	-	-
Naphthalene	3.6	9.66E+01	NV	-	3.70E-03	-	-	-	-	-	-
Phenanthrene	51	3.84E+01	NV	-	1.40E-01	-	-	-	-	-	-
Pyrene	84	1.47E+01	NV	-	1.05E-01	-	-	-	-	-	-
TOTAL								2.60E-04			2.60E-04

Summary of Estimated Health Risks[Back to User Input Sheet](#)**Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector****Darling Drive, Darling Harbour, Sydney NSW****On-Site Recreational User (version D) Scenario**

Exposure Pathway	Threshold Risk Estimates		Non-Threshold Risk Estimates (Lifetime Exposure)
	Adult Exposure	Childhood Exposure	
Incidental Ingestion of Soil	1.8E-02	1.5E-01	1.1E-06
Dermal Contact with Soil	3.0E-01	6.4E-01	6.9E-06
Inhalation of Surface Soil-Derived Dust in Outdoor Air	4.3E-07	4.3E-07	8.4E-09
Inhalation of Surface Soil-Derived Vapours in Outdoor Air	2.6E-04	2.6E-04	-
TOTAL	3.2E-01	7.9E-01	8.1E-06

Appendix F

Risk Estimates - On-Site Commercial Worker

Exposure Parameters:	Units	Site-Specific Value	Justification	Value Used in Calculations	
		Adult		Adult	
General receptor parameters:					
Body weight	kg	78	Adult value: male and female rounded average combined weight enHealth (2012b).	78	bwa
Exposure duration	yr	30	NEPC (1999) commercial/industrial exposure	30	eda
Averaging time (carcinogens)	yr	70	enHealth (2012b)	70	atca
Averaging time (non-carcinogens)	yr	30	NEPC (1999) commercial/industrial exposure	30	atnca
Incidental Soil Ingestion					
Daily soil ingestion rate	mg/day			0	irsa
Exposure frequency for soil ingestion	days/yr			240	efsa
Fraction of daily soil intake from site	unitless			1	fsa
Dermal Absorption of Soil					
Exposed skin surface area for soil contact	cm2			0	sasa
Soil to skin adherence factor	mg/cm2			0.5	sada
Exposure frequency for dermal contact with soil	days/yr			240	efdsa
Indoor Inhalation					
Exposure time (indoor air)	hrs/day	8	NEPC (1999)	8	etiaira
Exposure frequency (indoor air)	days/yr	240	NEPC (1999)	240	efiaira
Particulate emission factor (indoor air)	m3/kg			1.02E+09	pefia
Outdoor Inhalation					
Exposure time (outdoor air)	hrs/day			0	etoaira
Exposure frequency (outdoor air)	days/yr			240	efoaira
Particulate emission factor (outdoor air)	m3/kg			1.36E+09	pefoa
Potable Water Ingestion					
Potable water intake rate	L/day			2	irdwa
Exposure frequency for potable water intake	days/yr			240	efdwa
Incidental Water Ingestion					
Incidental ingestion rate	L/day			0.005	irbwa
Exposure frequency for incidental water ingestion	days/yr			0	efbwa
Dermal Contact with Water					
Exposed skin surface for water contact	cm2			0	sawa
Exposure time for dermal water contact	hr/day			0	etbwa
Exposure frequency for dermal water contact	days/yr			0	efdbwa
Vapour Inhalation Shower/ Sprinkler					
Exposure frequency	days/yr			0	efiairscha
Exposure time	min/ day			0	etiairsha
Inhalation rate	m3/hr			0.66	irshwa
Inhalation Absorption Adjustment Factor	mg/mg			1	iaafa
Lung retention factor	unitless			1	lrfa
Ingestion of Home Grown Produce					
Proportion of homegrown produce ingested				0	prphga
Exposure frequency	days/year			0	efhgpa
Inhalation of Vapours from Flowing Water					
Exposure time	hrs/day			0	etwfa
Exposure frequency	days/year			0	efwfa
Ingestion of Fish					
Daily fish ingestion rate	mg/day			0	irfa
Exposure frequency for fish ingestion	days/yr			0	effa
Fraction of daily fish intake from site	unitless			0	ffa

Vapour Modelling - Soil to Outdoor and Indoor Air
Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
Darling Drive, Darling Harbour, Sydney NSW
On-Site Commercial Worker (version D) Scenario

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A. Model Input Parameters

Parameter Definition	Units	Site-Specific Value	Label	Adopted Value for Model	Justification for Site-specific value, if applicable
Lower depth of surficial soil zone	cm	15	LDSSZ	15	Assumes impact is at surface
Depth to subsurface soil sources (below building, ground surface or trench)	cm		LS	15	Autocalculates from layer thicknesses
<u>Vadose Zone Layer 1 (soil type where source is)</u>					
Thickness	cm	15	HV	15	Assumes impact is at surface
SCS Soil Type:		Sand and gravel (<12% fines)			Conservative assumption
Fraction of organic carbon	unitless	0.002	OC	0.002	
Soil bulk density	g/cm3	1.66	sbd	1.66	
Air-filled porosity (volumetric)	cm3/cm3	0.321	VACS	0.321	
Water-filled porosity (volumetric)	cm3/cm3	0.054	wvcvz	0.054	
Total soil porosity	cm3/cm3	0.375	TPOR	0.375	
Vapour phase source partitioning adjustment	unitless	1	VPPA	1	Not considered within vapour modelling
Vadose zone biodegradation adjustment	unitless	1	BioA	1	Not considered within vapour modelling
<u>Building Characteristics</u>					
Enclosed-space volume/infiltration area (ratio)	cm	300	LB	300	ASTM 1739-95 (2010)e1 (default value) for commercial/industrial buildings
Enclosed-space foundation or wall thickness	cm	15	Lcrack	15	Building Code of Australia
Enclosed-space air exchange rate	1/s	0.0005556	ENCAER	5.56E-04	Minimum air exchange rate for commercial buildings (Building Code of Australia) - 2 exchanges per hour.
Areal fraction of cracks in foundations/walls	cm ² -cracks/cm ² -total area	0.00038	NU	0.00038	US EPA (2004) default for slab-on-grade buildings
Volumetric air content in found./wall cracks	cc/cc	0.26	VACF	0.26	ASTM 1739-95(2010)e1 (default value)
Volumetric water content in found./wall cracks	cc/cc	0.12	wvcfnd	0.12	ASTM 1739-95(2010)e1 (default value)
<u>Convective vapour flow term</u>					
Calculate convective flow term?		n	Conv	n	
Convective flow rate (if specified directly)	cm3/sec	83			Default USEPA (2004) 5L/ min
Slab Area	cm2	4.00E+06	Ab	4000000	Assumes building footprint of 20mx20m (CRC CARE, 2011, default commercial building)

Vapour Modelling - Soil to Outdoor and Indoor Air
Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
Darling Drive, Darling Harbour, Sydney NSW
On-Site Commercial Worker (version D) Scenario

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B. Chemical-Specific Fate and Transport Parameters

CHEMICAL	Koc (cm ³ /g)	Kd (cm ³ /g)	D ^{air} (cm ² /s)	D ^{wat} (cm ² /s)	MW (g/mol)	VP (mmHg)	Volatile?
TPH C10-C14 aromatic	3.55E+03	7.10E+00	1.00E-01	1.00E-05	1.36E+02	1.16E+00	Y
TPH C10-C14 aliphatic	1.12E+06	2.24E+03	1.00E-01	1.00E-05	1.70E+02	1.16E+00	Y

Definition of Parameters

Koc	Organic carbon partition coefficient	D ^{air}
Kd	Soil-water partition coefficient	D ^{wat}
H'	Dimensionless Henry's Law Constant	MW
S	Solubility	VP

C. Chemical-Specific Diffusion Coefficients

CHEMICAL	D _{s1} (cm ² /s)	D _{s2} (cm ² /s)	D _{crack} (cm ² /s)	C _{sat} (mg/kg)	Convective Factor (unitless)	D _a (cm ² /s)	M _{excav} (g)
TPH C10-C14 aromatic	1.62E-02	-	7.80E-03	1.81E+02	1.05E+02	1.91E-04	5.24E+05
TPH C10-C14 aliphatic	1.62E-02	-	7.80E-03	2.25E+02	1.05E+02	2.69E-04	6.22E+05

Definition of Parameters

D _s	Effective diffusion coefficient in soil based on vapor-phase concentration
D _{crack}	Effective diffusion coefficient through foundation cracks
C _{sat}	Soil concentration at which dissolved pore-water and vapor phases become saturated

D. Chemical-Specific Volatilisation Factors

CHEMICAL	VF _{as1} (mg/m ³ -air / mg/kg-soil)	VF _{as2} (mg/m ³ -air / mg/kg-soil)	VF _{samb} (mg/m ³ -air / mg/kg-soil)	VF _{sevp} (mg/m ³ -air / mg/kg-soil)	VF _{excav} (mg/m ³ -air / mg/kg-soil)
TPH C10-C14 aromatic	1.89E-04	1.32E-05	2.12E-03	2.41E-03	-2.26E+09
TPH C10-C14 aliphatic	2.24E-04	1.32E-05	2.99E-03	3.39E-03	-2.68E+09

Definition of Parameters

VF _{as}	Volatilization factor from surficial soils to ambient air (vapors) - use lower of two values
VF _p	Volatilization factor from surficial soils to ambient air (particulates)
VF _{samb}	Volatilization factor from subsurface soils to ambient air
VF _{sevp}	Volatilization factor from soil to enclosed-space vapors
VF _{excav}	Volatilisation factor from subsurface soil to trench air (where soil contamination is below base of trench)

Health Risk Calculations - Inhalation of Subsurface Soil-Derived Vapours in Indoor Air
 Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
 Darling Drive, Darling Harbour, Sydney NSW
 On-Site Commercial Worker (version D) Scenario

Chemical	Soil Conc. (mg/kg)	Csat (mg/kg)	Volatilisation Factor from Subsurface Soil to Indoor Air [(mg/m3)/(mg/kg)]	Vapour Concentration in Indoor Air (From Subsurface Soil) (mg/m3)	Threshold Intake and Risk Calculations			
					Inhalation RfC (Adjusted for background exposure) (mg/m3)	Adult Exposure Factor (threshold) (kg/m3)	Adult Exposure Adjusted Air Concentration (threshold) (mg/m3)	Hazard Index (Adult) (unitless)
TPH C10-C14 aromatic	410	1.81E+02	2.41E-03	4.36E-01	1.80E-01	5.27E-04	9.55E-02	5.31E-01
TPH C10-C14 aliphatic	410	2.25E+02	3.39E-03	7.64E-01	9.00E-01	7.43E-04	1.67E-01	1.86E-01
TOTAL								7.17E-01

Summary of Estimated Health Risks

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Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector

Darling Drive, Darling Harbour, Sydney NSW

On-Site Commercial Worker (version D) Scenario

Exposure Pathway	Threshold Risk Estimates	Non-Threshold Risk Estimates
	Adult Exposure	(Lifetime Exposure)
Inhalation of Subsurface Soil-Derived Vapours in Indoor Air	7.2E-01	-
TOTAL	7.2E-01	

Appendix G

Risk Estimates - On-Site Intrusive Maintenance Worker

Exposure Parameters:	Site-Specific Value		Justification	Value Used in Calculations			
	Adult	Child		Adult		Child	
General receptor parameters:							
	Units						
Body weight	kg	78	Adult value: male and female rounded average combined weight enHealth (2012b).	78	bwa	13	bwc
Exposure duration	yr	30	NEPC (1999) commercial/industrial exposure	30	eda	6	edc
Averaging time (carcinogens)	yr	70	enHealth (2012b)	70	atca	70	atcc
Averaging time (non-carcinogens)	yr	30	NEPC (1999) commercial/industrial exposure	30	atnca	6	atncc
Incidental Soil Ingestion							
Daily soil ingestion rate	mg/day	60	Adult value: 95th percentile value for outside soil plus indoor dust.	60	irsa	0	irsc
Exposure frequency for soil ingestion	days/yr	20	Conservative assumption - maintenance works for four weeks per year at the same location	20	efsa	0	efsc
Fraction of daily soil intake from site	unitless	1	Assumes all soil ingestion is from the Site	1	fsa	0	fsc
Dermal Absorption of Soil							
Exposed skin surface area for soil contact	cm2	6800	enHealth, 2012b - based on 95th percentile for adult males with head, hands, lower legs and forearms exposed.	6800	sasa	0	sasc
Soil to skin adherence factor	mg/cm2	0.5	enHealth (2012) recommended value for screening risk assessments	0.5	sada	0	sadc
Exposure frequency for dermal contact with soil	days/yr	20	Conservative assumption - maintenance works for four weeks per year at the same location	20	efdsa	0	efdsc
Indoor Inhalation							
Exposure time (indoor air)	hrs/day			8	etiaira	0	etiairc
Exposure frequency (indoor air)	days/yr			240	efiaira	0	efiairc
Particulate emission factor (indoor air)	m3/kg			1.02E+09	pefia	0	pefic
Outdoor Inhalation							
Exposure time (outdoor air)	hrs/day	10	Conservative assumption	10	etoaira	0	etoairc
Exposure frequency (outdoor air)	days/yr	20	Conservative assumption - maintenance works for four weeks per year at the same location	20	efoaira	0	efoairc
Particulate emission factor (outdoor air)	m3/kg	4.40E+08	USEPA (2002) Supplemental guidance for developing soil screening levels for Superfund sites, OSWER 9355.4-24, Solid Waste and Emergency Response, United States Environmental Protection Agency, Washington, DC.	440000000	pefoa	0	pefoc
Potable Water Ingestion							
Potable water intake rate	L/day			2	irdwa	0	irdwc
Exposure frequency for potable water intake	days/yr			240	efdwa	0	efdwc
Incidental Water Ingestion							
Incidental ingestion rate	L/day			0.005	irbwa	0	irbwc
Exposure frequency for incidental water ingestion	days/yr			0	efbwa	0	efbwc
Dermal Contact with Water							
Exposed skin surface for water contact	cm2			0	sawa	0	sawc
Exposure time for dermal water contact	hr/day			0	etbwa	0	etbwc
Exposure frequency for dermal water contact	days/yr			0	efdbwa	0	efdbwc
Vapour Inhalation Shower/ Sprinkler							
Exposure frequency	days/yr			0	efairscha	0	efairschc
Exposure time	min/ day			0	etiairsha	0	etiairshc
Inhalation rate	m3/hr			0.66	irshwa	0.454	irshwc
Inhalation Absorption Adjustment Factor	mg/mg			1	iaafa	1	iaafc
Lung retention factor	unitless			1	lrfa	1	lrfc
Ingestion of Home Grown Produce							
Proportion of homegrown produce ingested				0	prphga	0	prphgc
Exposure frequency	days/year			0	efhgpa	0	efhgpc
Inhalation of Vapours from Flowing Water							
Exposure time	hrs/day			0	etwfa	0	etwfc
Exposure frequency	days/year			0	efwfa	0	efwfc
Ingestion of Fish							
Daily fish ingestion rate	mg/day			0	irfa	0	irfc
Exposure frequency for fish ingestion	days/yr			0	effa	0	effc
Fraction of daily fish intake from site	unitless			0	ffa	0	ffc

Vapour Modelling - Soil to Outdoor Air & Direct Contact
Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
Darling Drive, Darling Harbour, Sydney NSW
On-Site Intrusive Maintenance Worker (version D) Scenario

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A. Model Input Parameters

Parameter Definition	Units	Default Value	Notes	Site-Specific Value	Label	Adopted Value for Model	Justification for Site-specific value, if applicable
Depth to subsurface soil sources (below building, ground surface or trench)	cm	100			LS	15	Autocalculates from layer thicknesses
<i>Vadose Zone Layer 1 (soil type where source is)</i>							
Thickness	cm	100		15	HV	15	Assumes impact is at surface
SCS Soil Type:				Sand and gravel (<12% fines)			Conservative assumption
Fraction of organic carbon	unitless	0.01		0.002	OC	0.002	
Soil bulk density	g/cm3	1.7		1.66	sbd	1.66	
Air-filled porosity (volumetric)	cm3/cm3	0.26		0.321	VACS	0.321	
Water-filled porosity (volumetric)	cm3/cm3	0.12		0.054	vwcwz	0.054	
Total soil porosity	cm3/cm3	0.38		0.375	TPOR	0.375	
Vapour phase source partitioning adjustment	unitless	1		1	VPPA	1	Not considered within vapour modelling
Vadose zone biodegradation adjustment	unitless	1		1	BioA	1	Not considered within vapour modelling
<i>Outdoor Air Characteristics</i>							
Wind speed in outdoor mixing zone (ambient air or trench, as appropriate)	cm/s	22.5		37.778	windsp	37.778	Based on 10% annual average of 9am and 3pm at Sydney (Observation Hill) weather station
Width of source area parallel to wind or groundwater flow direction	cm	600		1500	WSA	1500	ASTM 1739-95(2010)e1
Ambient air mixing zone height	cm	200		200	AAMZH	200	ASTM 1739-95(2010)e1

B. Chemical-Specific Fate and Transport Parameters

CHEMICAL	Koc (cm ³ /g)	Kd (cm ³ /g)	H' (cc-H ₂ O / cc-air)	S (mg/l-water)	D ^{air} (cm ² /s)	D ^{wat} (cm ² /s)	MW (g/mol)	VP (mmHg)	Volatile?
TPH C10-C14 aliphatic	1.12E+06	2.24E+03	6.26E+01	9.99E-02	1.00E-01	1.00E-05	1.70E+02	1.16E+00	Y
TPH C10-C14 aromatic	3.55E+03	7.10E+00	1.41E-01	2.53E+01	1.00E-01	1.00E-05	1.36E+02	1.16E+00	Y
TPH C15-C28 aliphatic	6.31E+08	1.26E+06	8.27E+01	1.11E-04	1.00E-01	1.00E-05	2.60E+02	5.93E-03	N
TPH C15-C28 aromatic	4.47E+04	8.93E+01	4.90E-03	1.06E+00	1.00E-01	1.00E-05	2.09E+02	5.51E-03	N
TPH C29-C36 aliphatic	6.31E+08	1.26E+06	8.50E+01	2.50E-06	1.00E-01	1.00E-05	2.70E+02	8.36E-04	N
TPH C29-C36 aromatic	1.26E+05	2.52E+02	1.70E-05	6.60E-03	1.00E-01	1.00E-05	2.40E+02	3.34E-07	N
Acenaphthene	5.03E+03	1.01E+01	7.52E-03	3.90E+00	5.06E-02	8.33E-06	1.54E+02	2.15E-03	N
Acenaphthylene	5.03E+03	1.01E+01	4.66E-03	1.61E+01	4.50E-02	6.98E-06	1.52E+02	6.68E-03	N
Anthracene	1.64E+04	3.28E+01	2.27E-03	4.34E-02	3.90E-02	7.85E-06	1.78E+02	6.53E-06	N
Benz(a)anthracene	1.77E+05	3.54E+02	4.91E-04	9.40E-03	5.10E-02	9.00E-06	2.28E+02	2.10E-07	N
Benzo(a)pyrene	5.87E+05	1.17E+03	1.87E-05	1.62E-03	4.30E-02	9.00E-06	2.52E+02	5.49E-09	N
Benzo(b)fluoranthene	5.99E+05	1.20E+03	2.69E-05	1.50E-03	2.26E-02	5.56E-06	2.52E+02	5.00E-07	N
Benzo(k)fluoranthene	5.87E+05	1.17E+03	2.39E-05	8.00E-04	2.26E-02	5.56E-06	2.52E+02	9.65E-10	N
Benzo(g,h,i)perylene	1.95E+06	3.90E+03	1.35E-05	2.60E-04	4.90E-02	5.56E-06	2.76E+02	1.00E-10	N
Chrysene	1.81E+05	3.62E+02	2.14E-04	2.00E-03	2.48E-02	6.21E-06	2.28E+02	6.23E-09	N
Dibenz(a,h)anthracene	1.91E+06	3.82E+03	5.76E-06	2.49E-03	2.00E-02	5.24E-06	2.78E+02	9.55E-10	N
Fluoranthene	5.55E+04	1.11E+02	3.62E-04	2.60E-01	3.02E-02	6.35E-06	2.02E+02	9.22E-06	N
Fluorene	9.16E+03	1.83E+01	3.93E-03	1.69E+00	4.40E-02	7.89E-06	1.66E+02	6.00E-04	N
Indeno(1,2,3-cd)pyrene	1.95E+06	3.90E+03	1.42E-05	1.90E-04	2.30E-02	4.41E-06	2.76E+02	1.25E-10	N
Naphthalene	1.54E+03	3.08E+00	1.80E-02	3.10E+01	6.05E-02	8.38E-06	1.28E+02	8.50E-02	N
Phenanthrene	1.67E+04	3.34E+01	1.73E-03	1.15E+00	3.45E-02	6.69E-06	1.78E+02	1.21E-04	N
Pyrene	5.43E+04	1.09E+02	4.87E-04	1.35E-01	2.78E-02	7.25E-06	2.02E+02	4.50E-06	N

Definition of Parameters

Koc	Organic carbon partition coefficient	D ^{air}	Diffusion coefficient in air
Kd	Soil-water partition coefficient	D ^{wat}	Diffusion coefficient in water
H'	Dimensionless Henry's Law Constant	MW	Molecular weight
S	Solubility	VP	Vapour pressure

Vapour Modelling - Soil to Outdoor Air & Direct Contact
Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
Darling Drive, Darling Harbour, Sydney NSW
On-Site Intrusive Maintenance Worker (version D) Scenario

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C. Chemical-Specific Diffusion Coefficients

CHEMICAL	D_{s1}	D_{s2}	D_{s3}	D_{slot}	D_{crack}	C_{sat}	Convective Factor	D_a	M_{excav}
	(cm ² /s)	(mg/kg)	(unitless)	(cm ² /s)	(g)				
TPH C10-C14 aliphatic	1.62E-02	-	-	1.62E-02	7.80E-03	2.25E+02	1.57E+01	2.69E-04	6.22E+05
TPH C10-C14 aromatic	1.62E-02	-	-	1.62E-02	7.80E-03	1.81E+02	1.57E+01	1.91E-04	5.24E+05
TPH C15-C28 aliphatic	NV	NV	NV	NV	NV	1.40E+02	NV	NV	NV
TPH C15-C28 aromatic	NV	NV	NV	NV	NV	9.47E+01	NV	NV	NV
TPH C29-C36 aliphatic	NV	NV	NV	NV	NV	3.16E+00	NV	NV	NV
TPH C29-C36 aromatic	NV	NV	NV	NV	NV	1.66E+00	NV	NV	NV
Acenaphthene	NV	NV	NV	NV	NV	3.94E+01	NV	NV	NV
Acenaphthylene	NV	NV	NV	NV	NV	1.63E+02	NV	NV	NV
Anthracene	NV	NV	NV	NV	NV	1.42E+00	NV	NV	NV
Benz(a)anthracene	NV	NV	NV	NV	NV	3.33E+00	NV	NV	NV
Benzo(a)pyrene	NV	NV	NV	NV	NV	1.90E+00	NV	NV	NV
Benzo(b)fluoranthene	NV	NV	NV	NV	NV	1.80E+00	NV	NV	NV
Benzo(k)fluoranthene	NV	NV	NV	NV	NV	9.39E-01	NV	NV	NV
Benzo(g,h,i)perylene	NV	NV	NV	NV	NV	1.01E+00	NV	NV	NV
Chrysene	NV	NV	NV	NV	NV	7.24E-01	NV	NV	NV
Dibenz(a,h)anthracene	NV	NV	NV	NV	NV	9.51E+00	NV	NV	NV
Fluoranthene	NV	NV	NV	NV	NV	2.89E+01	NV	NV	NV
Fluorene	NV	NV	NV	NV	NV	3.10E+01	NV	NV	NV
Indeno(1,2,3-cd)pyrene	NV	NV	NV	NV	NV	7.41E-01	NV	NV	NV
Naphthalene	NV	NV	NV	NV	NV	9.66E+01	NV	NV	NV
Phenanthrene	NV	NV	NV	NV	NV	3.84E+01	NV	NV	NV
Pyrene	NV	NV	NV	NV	NV	1.47E+01	NV	NV	NV

Definition of Parameters

D_s Effective diffusion coefficient in soil based on vapor-phase concentration
 D_{crack} Effective diffusion coefficient through foundation cracks
 C_{sat} Soil concentration at which dissolved pore-water and vapor phases become saturated

D_a = Apparent diffusivity (for construction scenario Volatilisation Factor)
 M_{excav} = Cumulative unit mass emitted from excavation (Eq E-13 in USEPA, 2002).

D. Chemical-Specific Volatilisation Factors

CHEMICAL	VF_{as1}	VF_{as2}	$VF_{p (indoor)}$	$VF_{p (outdoor)}$	VF_{samb}	VF_{seps}
	(mg/m ³ -air / mg/kg-soil)					
TPH C10-C14 aliphatic	4.44E-04	1.74E-04	9.80E-10	2.27E-09	5.94E-03	1.02E-02
TPH C10-C14 aromatic	3.75E-04	1.74E-04	9.80E-10	2.27E-09	4.21E-03	7.24E-03
TPH C15-C28 aliphatic	NV	NV	9.80E-10	2.27E-09	NV	NV
TPH C15-C28 aromatic	NV	NV	9.80E-10	2.27E-09	NV	NV
TPH C29-C36 aliphatic	NV	NV	9.80E-10	2.27E-09	NV	NV
TPH C29-C36 aromatic	NV	NV	9.80E-10	2.27E-09	NV	NV
Acenaphthene	NV	NV	9.80E-10	2.27E-09	NV	NV
Acenaphthylene	NV	NV	9.80E-10	2.27E-09	NV	NV
Anthracene	NV	NV	9.80E-10	2.27E-09	NV	NV
Benz(a)anthracene	NV	NV	9.80E-10	2.27E-09	NV	NV
Benzo(a)pyrene	NV	NV	9.80E-10	2.27E-09	NV	NV
Benzo(b)fluoranthene	NV	NV	9.80E-10	2.27E-09	NV	NV
Benzo(k)fluoranthene	NV	NV	9.80E-10	2.27E-09	NV	NV
Benzo(g,h,i)perylene	NV	NV	9.80E-10	2.27E-09	NV	NV
Chrysene	NV	NV	9.80E-10	2.27E-09	NV	NV
Dibenz(a,h)anthracene	NV	NV	9.80E-10	2.27E-09	NV	NV
Fluoranthene	NV	NV	9.80E-10	2.27E-09	NV	NV
Fluorene	NV	NV	9.80E-10	2.27E-09	NV	NV
Indeno(1,2,3-cd)pyrene	NV	NV	9.80E-10	2.27E-09	NV	NV
Naphthalene	NV	NV	9.80E-10	2.27E-09	NV	NV
Phenanthrene	NV	NV	9.80E-10	2.27E-09	NV	NV
Pyrene	NV	NV	9.80E-10	2.27E-09	NV	NV

Definition of Parameters

VF_{as} Volatilization factor from surficial soils to ambient air (vapors) - use lower of two values
 VF_p Volatilization factor from surficial soils to ambient air (particulates)
 VF_{samb} Volatilization factor from subsurface soils to ambient air
 VF_{seps} Volatilization factor from soil to enclosed-space vapors

Health Risk Calculations - Incidental Soil Ingestion
Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
Darling Drive, Darling Harbour, Sydney NSW
On-Site Intrusive Maintenance Worker (version D) Scenario

Chemical	Soil Concentration (mg/kg)	Oral Soil Bioavailability Factor (unitless)	Threshold Intake and Risk Calculations				Non-Threshold Intake and Risk Calculations			
			Oral RfD (mg/kg/day)	Adult Intake Factor (threshold) (kg/kg/day)	Adult Intake (threshold) (mg/kg/day)	Hazard Index (Adult) (unitless)	Oral CSF (mg/kg/day) ⁻¹	Adult Intake Factor (non-threshold) (kg/kg/day)	Lifetime Intake (non-threshold) (mg/kg/day)	Lifetime Excess Cancer Risk (unitless)
TPH C10-C14 aliphatic	410	1.00E+00	1.00E-01	4.21E-08	1.73E-05	1.73E-04	-	-	-	-
TPH C10-C14 aromatic	410	1.00E+00	4.00E-02	4.21E-08	1.73E-05	4.32E-04	-	-	-	-
TPH C15-C28 aliphatic	2500	1.00E+00	2.00E+00	4.21E-08	1.05E-04	5.27E-05	-	-	-	-
TPH C15-C28 aromatic	2500	1.00E+00	3.00E-02	4.21E-08	1.05E-04	3.51E-03	-	-	-	-
TPH C29-C36 aliphatic	1600	1.00E+00	2.00E+00	4.21E-08	6.74E-05	3.37E-05	-	-	-	-
TPH C29-C36 aromatic	1600	1.00E+00	3.00E-02	4.21E-08	6.74E-05	2.25E-03	-	-	-	-
Acenaphthene	0.6	1.00E+00	6.00E-02	4.21E-08	2.53E-08	4.21E-07	-	-	-	-
Acenaphthylene	1.3	1.00E+00	6.00E-02	4.21E-08	5.48E-08	9.13E-07	-	-	-	-
Anthracene	3.4	1.00E+00	3.00E-01	4.21E-08	1.43E-07	4.78E-07	-	-	-	-
Benz(a)anthracene	39	1.00E+00	-	-	-	-	4.30E-02	1.81E-08	7.05E-07	3.03E-08
Benzo(a)pyrene	6.5	1.00E+00	-	-	-	-	4.30E-01	1.81E-08	1.17E-07	5.05E-08
Benzo(b)fluoranthene	9.3	1.00E+00	-	-	-	-	4.30E-02	1.81E-08	1.68E-07	7.22E-09
Benzo(k)fluoranthene	62	1.00E+00	-	-	-	-	4.30E-02	1.81E-08	1.12E-06	4.82E-08
Benzo(g,h,i)perylene	3.9	1.00E+00	-	-	-	-	4.30E-03	1.81E-08	7.05E-08	3.03E-10
Chrysene	6	1.00E+00	--	-	-	-	4.30E-03	1.81E-08	1.08E-07	4.66E-10
Dibenz(a,h)anthracene	1	1.00E+00	--	-	-	-	4.30E-01	1.81E-08	1.81E-08	7.77E-09
Fluoranthene	94	1.00E+00	4.00E-02	4.21E-08	3.96E-06	9.91E-05	-	-	-	-
Fluorene	1.8	1.00E+00	4.00E-02	4.21E-08	7.59E-08	1.90E-06	-	-	-	-
Indeno(1,2,3-cd)pyrene	3.5	1.00E+00	--	-	-	-	4.30E-02	1.81E-08	6.32E-08	2.72E-09
Naphthalene	3.6	1.00E+00	2.00E-02	4.21E-08	1.52E-07	7.59E-06	-	-	-	-
Phenanthrene	51	1.00E+00	4.00E-02	4.21E-08	2.15E-06	5.37E-05	-	-	-	-
Pyrene	84	1.00E+00	3.00E-02	4.21E-08	3.54E-06	1.18E-04	-	-	-	-
TOTAL						6.73E-03				1.47E-07

Health Risk Calculations - Dermal Contact with Soil
Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
Darling Drive, Darling Harbour, Sydney NSW
On-Site Intrusive Maintenance Worker (version D) Scenario

Chemical	Soil Concentration (mg/kg)	Dermal Absorption Factor (DAF) (unitless)	Threshold Intake and Risk Calculations			
			Dermal RfD (mg/kg/day)	Adult Intake Factor (threshold) (kg/kg/day)	Adult Intake (threshold) (mg/kg/day)	Hazard Index (Adult) (unitless)
TPH C10-C14 aliphatic	410	0.2	1.00E-01	4.78E-07	1.96E-04	1.96E-03
TPH C10-C14 aromatic	410	0.2	4.00E-02	4.78E-07	1.96E-04	4.90E-03
TPH C15-C28 aliphatic	2500	0.2	2.00E+00	4.78E-07	1.19E-03	5.97E-04
TPH C15-C28 aromatic	2500	0.2	3.00E-02	4.78E-07	1.19E-03	3.98E-02
TPH C29-C36 aliphatic	1600	0.2	2.00E+00	4.78E-07	7.64E-04	3.82E-04
TPH C29-C36 aromatic	1600	0.2	3.00E-02	4.78E-07	7.64E-04	2.55E-02
Acenaphthene	0.6	0.13	6.00E-02	3.11E-07	1.86E-07	3.11E-06
Acenaphthylene	1.3	0.13	6.00E-02	3.11E-07	4.04E-07	6.73E-06
Anthracene	3.4	0.13	3.00E-01	3.11E-07	1.06E-06	3.52E-06
Benz(a)anthracene	39	0.13	-	-	-	-
Benzo(a)pyrene	6.5	0.13	-	-	-	-
Benzo(b)fluoranthene	9.3	0.13	-	-	-	-
Benzo(k)fluoranthene	62	0.13	-	-	-	-
Benzo(g,h,i)perylene	3.9	0.13	-	-	-	-
Chrysene	6	0.13	-	-	-	-
Dibenz(a,h)anthracene	1	0.13	-	-	-	-
Fluoranthene	94	0.13	4.00E-02	3.11E-07	2.92E-05	7.30E-04
Fluorene	1.8	0.13	4.00E-02	3.11E-07	5.59E-07	1.40E-05
Indeno(1,2,3-cd)pyrene	3.5	0.13	-	-	-	-
Naphthalene	3.6	0.13	2.00E-02	3.11E-07	1.12E-06	5.59E-05
Phenanthrene	51	0.13	4.00E-02	3.11E-07	1.58E-05	3.96E-04
Pyrene	84	0.13	3.00E-02	3.11E-07	2.61E-05	8.69E-04
TOTAL					7.52E-02	

Non-Threshold Intake and Risk Calculations			
Dermal CSF (mg/kg/day) ⁻¹	Adult Intake Factor (non-threshold) (kg/kg/day)	Lifetime Intake (non-threshold) (mg/kg/day)	Lifetime Excess Cancer Risk (unitless)
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
4.30E-02	1.33E-07	5.19E-06	2.23E-07
4.30E-01	1.33E-07	8.65E-07	3.72E-07
4.30E-02	1.33E-07	1.24E-06	5.32E-08
4.30E-02	1.33E-07	8.25E-06	3.55E-07
4.30E-03	1.33E-07	5.19E-07	2.23E-09
4.30E-03	1.33E-07	7.98E-07	3.43E-09
4.30E-01	1.33E-07	1.33E-07	5.72E-08
-	-	-	-
-	-	-	-
4.30E-02	1.33E-07	4.66E-07	2.00E-08
-	-	-	-
-	-	-	-
-	-	-	-
TOTAL			1.09E-06

**Health Risk Calculations - Inhalation of Soil-Derived Dust (Particulates) in Outdoor Air
 Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
 Darling Drive, Darling Harbour, Sydney NSW
 On-Site Intrusive Maintenance Worker (version D) Scenario**

Chemical	Soil Concentration (mg/kg)	Particulate Concentration in Outdoor Air (From Surface Soil) (m3/kg)	Threshold Intake and Risk Calculations			
			Inhalation RfC (adjusted for background exposure) (mg/m3)	Adult Exposure Factor (threshold) (kg/m3)	Adult Exposure Adjusted Air Concentration (threshold) (mg/m3)	Hazard Index (Adult) (unitless)
TPH C10-C14 aliphatic	410	2.27E-09	9.00E-01	5.19E-11	2.13E-08	2.36E-08
TPH C10-C14 aromatic	410	2.27E-09	1.80E-01	5.19E-11	2.13E-08	1.18E-07
TPH C15-C28 aliphatic	2500	2.27E-09	6.30E+00	5.19E-11	1.30E-07	2.06E-08
TPH C15-C28 aromatic	2500	2.27E-09	9.45E-02	5.19E-11	1.30E-07	1.37E-06
TPH C29-C36 aliphatic	1600	2.27E-09	6.30E+00	5.19E-11	8.30E-08	1.32E-08
TPH C29-C36 aromatic	1600	2.27E-09	9.45E-02	5.19E-11	8.30E-08	8.79E-07
Acenaphthene	0.6	2.27E-09	2.10E-01	5.19E-11	3.11E-11	1.48E-10
Acenaphthylene	1.3	2.27E-09	2.10E-01	5.19E-11	6.75E-11	3.21E-10
Anthracene	3.4	2.27E-09	1.05E+00	5.19E-11	1.76E-10	1.68E-10
Benz(a)anthracene	39	2.27E-09	-	-	-	-
Benzo(a)pyrene	6.5	2.27E-09	-	-	-	-
Benzo(b)fluoranthene	9.3	2.27E-09	-	-	-	-
Benzo(k)fluoranthene	62	2.27E-09	-	-	-	-
Benzo(g,h,i)perylene	3.9	2.27E-09	-	-	-	-
Chrysene	6	2.27E-09	-	-	-	-
Dibenz(a,h)anthracene	1	2.27E-09	-	-	-	-
Fluoranthene	94	2.27E-09	1.40E-01	5.19E-11	4.88E-09	3.48E-08
Fluorene	1.8	2.27E-09	1.40E-01	5.19E-11	9.34E-11	6.67E-10
Indeno(1,2,3-cd)pyrene	3.5	2.27E-09	-	-	-	-
Naphthalene	3.6	2.27E-09	3.70E-03	5.19E-11	1.87E-10	5.05E-08
Phenanthrene	51	2.27E-09	1.40E-01	5.19E-11	2.65E-09	1.89E-08
Pyrene	84	2.27E-09	1.05E-01	5.19E-11	4.36E-09	4.15E-08
TOTAL					2.57E-06	

Non-Threshold Intake and Risk Calculations			
Inhalation Unit Risk (ug/m3)-1	Adult Exposure Factor (non-threshold) (kg/m3)	Lifetime Exposure Adjusted Air Concentration (non-threshold) (mg/m3)	Lifetime Excess Cancer Risk (unitless)
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
8.70E-03	2.22E-11	8.67E-10	7.55E-09
8.70E-02	2.22E-11	1.45E-10	1.26E-08
8.70E-03	2.22E-11	2.07E-10	1.80E-09
8.70E-03	2.22E-11	1.38E-09	1.20E-08
8.70E-04	2.22E-11	8.67E-11	7.55E-11
8.70E-04	2.22E-11	1.33E-10	1.16E-10
8.70E-02	2.22E-11	2.22E-11	1.93E-09
-	-	-	-
-	-	-	-
8.70E-03	2.22E-11	7.78E-11	6.77E-10
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
-	-	-	-
TOTAL			3.67E-08

Health Risk Calculations - Inhalation of Surface Soil-Derived Vapours in Outdoor Air
 Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector
 Darling Drive, Darling Harbour, Sydney NSW
 On-Site Intrusive Maintenance Worker (version D) Scenario

Chemical	Soil Conc. (mg/kg)	Csat (mg/kg)	Volatilisation Factor from Subsurface Soil to Outdoor Air [(mg/m3)/(mg/kg)]	Vapour Concentration in Outdoor Air (From Surface Soil) (mg/m3)	Threshold Intake and Risk Calculations			
					Inhalation RfC (adjusted for background exposure) (mg/m3)	Adult Exposure Factor (threshold) (kg/m3)	Adult Exposure Adjusted Air Concentration (threshold) (mg/m3)	Hazard Index (Adult) (unitless)
TPH C10-C14 aliphatic	410	2.25E+02	1.74E-04	3.93E-02	9.00E-01	3.98E-06	8.96E-04	9.96E-04
TPH C10-C14 aromatic	410	1.81E+02	1.74E-04	3.15E-02	1.80E-01	3.98E-06	7.20E-04	4.00E-03
TPH C15-C28 aliphatic	2500	1.40E+02	NV	-	6.30E+00	-	-	-
TPH C15-C28 aromatic	2500	9.47E+01	NV	-	9.45E-02	-	-	-
TPH C29-C36 aliphatic	1600	3.16E+00	NV	-	6.30E+00	-	-	-
TPH C29-C36 aromatic	1600	1.66E+00	NV	-	9.45E-02	-	-	-
Acenaphthene	0.6	3.94E+01	NV	-	2.10E-01	-	-	-
Acenaphthylene	1.3	1.63E+02	NV	-	2.10E-01	-	-	-
Anthracene	3.4	1.42E+00	NV	-	1.05E+00	-	-	-
Benz(a)anthracene	39	3.33E+00	NV	-	-	-	-	-
Benzo(a)pyrene	6.5	1.90E+00	NV	-	-	-	-	-
Benzo(b)fluoranthene	9.3	1.80E+00	NV	-	-	-	-	-
Benzo(k)fluoranthene	62	9.39E-01	NV	-	-	-	-	-
Benzo(g,h,i)perylene	3.9	1.01E+00	NV	-	-	-	-	-
Chrysene	6	7.24E-01	NV	-	-	-	-	-
Dibenz(a,h)anthracene	1	9.51E+00	NV	-	-	-	-	-
Fluoranthene	94	2.89E+01	NV	-	1.40E-01	-	-	-
Fluorene	1.8	3.10E+01	NV	-	1.40E-01	-	-	-
Indeno(1,2,3-cd)pyrene	3.5	7.41E-01	NV	-	-	-	-	-
Naphthalene	3.6	9.66E+01	NV	-	3.70E-03	-	-	-
Phenanthrene	51	3.84E+01	NV	-	1.40E-01	-	-	-
Pyrene	84	1.47E+01	NV	-	1.05E-01	-	-	-
TOTAL								5.00E-03

Summary of Estimated Health Risks[Back to User Input Sheet](#)**Sydney International Convention Exhibition and Entertainment Precinct - PPP Sector****Darling Drive, Darling Harbour, Sydney NSW****On-Site Intrusive Maintenance Worker (version D) Scenario**

Exposure Pathway	Threshold Risk Estimates	Non-Threshold Risk Estimates
	Adult Exposure	(Lifetime Exposure)
Incidental Ingestion of Soil	6.7E-03	1.5E-07
Dermal Contact with Soil	7.5E-02	1.1E-06
Inhalation of Surface Soil-Derived Dust in Outdoor Air	2.6E-06	3.7E-08
Inhalation of Surface Soil-Derived Vapours in Outdoor Air	5.0E-03	-
TOTAL	8.7E-02	1.3E-06

Appendix H

Methodology and Algorithms

Risk Assessment Equations

1.1 Estimate of Exposure

Incidental Ingestion of Soil

$$CDI_{ing,s} = \frac{C_s * IngR_s * EF * ED * CF}{365 \frac{\text{days}}{\text{year}} * AT * BW}$$

Where:

- $CDI_{ing,s}$ = Chronic Daily Intake for Soil Ingestion (mg/kg/day)
- C_s = Chemical Concentration in Soil (mg/kg)
- $IngR_s$ = Soil Ingestion Rate (mg/day)
- EF = Exposure Frequency (days/year)
- ED = Exposure Duration (years)
- CF = Unit conversion factor (kg/10⁶ mg)
- AT = Averaging Time (years)
 - = 70 years for non-threshold carcinogens
 - = ED for chemicals assessed based on threshold effects
- BW = Body weight (kg)

Dermal Contact with Soil

$$CDI_{der,s} = \frac{C_s * AH * SA * AF * EF * ED * CF}{365 \frac{\text{days}}{\text{year}} * AT * BW}$$

Where:

- $CDI_{der,s}$ = Chronic Daily Intake for Dermal Contact with Soil (mg/kg/day)
- AH = Soil Adherence Factor (mg/cm²/day)
- SA = Skin Surface Available for Contact (cm²)
- AF = Dermal Absorption Factor (chemical-specific; unitless)

and other factors are as defined earlier.

Inhalation of Particulates or Vapours

The following equation has been adopted to estimate intakes associated with inhalation of chemicals in air (particulates or vapours). Inhalation exposures have been estimated using the revised methodology recently published by the USEPA (USEPA, 2009), which recommends adjustment of the measured or estimated contaminant concentration in air to account for site-specific exposure considerations, rather than estimation of a chronic daily intake of contaminant via the inhalation pathway.

For particulates, it is assumed that all particulates inhaled are small enough to penetrate deep into the lungs (i.e., are inspirable), and that the particulate air EPCs have been estimated as inspirable (PM₁₀) dust concentrations.

$$EC_{inh} = \frac{C_a * ET * EF * ED}{AT * 365 \frac{\text{days}}{\text{year}} * 24 \frac{\text{hours}}{\text{day}}}$$

Where:

EC_{inh} = Exposure Adjusted Air Concentration (mg/m^3)

C_a = Chemical Concentration in Air (mg/m^3)

ET = Exposure Time (hours/day)

and other factors are as defined earlier.

Note that when assessing inhalation of particulates derived from soil, the chemical concentration in airborne particulates ($C_{a,part}$) is calculated as:

$$C_{a,part} = \frac{C_s}{PEF}$$

Where PEF is the particulate emission factor in units of m^3/kg .

When assessing inhalation of vapours derived from soil or groundwater, the vapour concentration in air ($C_{a,vap}$) is calculated as:

$$C_{a,vap,s} = C_s * VF_s$$

Where:

$C_{a,vap,s}$ = Vapour concentration in air deriving from soil (mg/m^3)

VF_s = Volatilisation Factor from soil to air ($\text{mg}/\text{m}^3 / \text{mg}/\text{kg}$)

OR

$$C_{a,vap,gw} = C_{gw} * VF_{gw}$$

Where:

$C_{a,vap,gw}$ = Vapour concentration in air deriving from groundwater (mg/m^3)

VF_{gw} = Volatilisation Factor from groundwater to air ($\text{mg}/\text{m}^3 / \text{mg}/\text{L}$)

1.2 Estimation of Risks

Threshold Risk Estimates

Risks to human health for CoPCs assessed on the basis of a threshold approach were estimated by comparison of the daily chemical intake or exposure adjusted air concentration of each CoPC with its respective TDI or Reference Concentration allowable from the Site (i.e., the TDI minus background intakes). The resulting ratio, referred to as the hazard quotient, is derived in the following manner:

$$HQ = \frac{CDI_t}{TDI - \text{background}}$$

or

$$HQ = \frac{EC_{inh}}{RfC - \text{background}}$$

Where:

- HQ = Hazard Quotient (unitless)
- CDI_t = Chronic Daily Intake (calculated based on threshold averaging time) (mg/kg/day)
- TDI = Tolerable Daily Intake (mg/kg/day) – adjusted for background intake
- EC_{inh} = Exposure adjusted air concentration (mg/m³)
- RfC = Tolerable Concentration in air (mg/m³) – adjusted for background intake

A potentially unacceptable chemical intake/exposure is indicated if the exposure level exceeds the TDI or TC (i.e. if the hazard quotient is greater than 1).

To assess the overall potential for adverse health effects posed by exposure to multiple chemicals, the hazard quotients for each chemical and exposure pathway relevant to a receptor are summed. The resulting sum is referred to as the hazard index (HI), and is calculated using the following equation.

$$HI = \sum_{i=1, j=1}^n HQ_{i,j}$$

Where:

- HI = Hazard Index (unitless)
- HQ_{i,j} = Hazard Quotient for pathway *i* and chemical *j* (unitless)
- n = Number of chemicals and/or pathways relevant to land use scenario

If the HI is less than one, then cumulative exposure to the CoPC is considered unlikely to result in an adverse effect. If the sum is greater than one, a more detailed and critical evaluation of the hazards may be required, or appropriate risk management measures at the Site may need to be implemented.

Non-Threshold (Carcinogenic) Risk Estimates

Risks to human health for CoPC considered to be genotoxic carcinogens were estimated as the incremental probability of an individual developing cancer over a lifetime as a result of chemical exposure. The numerical estimate of incremental lifetime carcinogenic risk was calculated using the following relationship:

$$ILCR = CDI_{nt} * SF$$

or

$$ILCR = EC_{inh} * IUR * 10^3 \frac{\mu g}{mg}$$

Where:

- ILCR = Incremental Lifetime Cancer Risk (unitless)
- CDI_{nt} = Chronic Daily Intake (calculated based on non-threshold averaging time) (mg/kg/day)
- SF = Cancer Slope Factor (mg/kg/day)⁻¹
- EC_{inh} = Exposure adjusted air concentration (mg/m³)
- IUR = Inhalation Unit Risk (μg/m³)⁻¹

To assess the overall potential for effects posed by simultaneous exposure to more than one chemical that is associated with non-threshold carcinogenic effects, the risk for each chemical and pathway relevant to a receptor, and for adults and children (as relevant), were summed. The resulting sum is referred to as the cumulative incremental lifetime carcinogenic risk and is estimated as follows:

$$ILCR_{cum} = \sum_{i=1, j=1}^n ILCR_{i,j}$$

Where

$ILCR_{cum}$ = Cumulative ILCR for a given receptor (unitless)

$ILCR_{i,j}$ = ILCR for chemical i and pathway j

n = Number of chemicals and/or pathways relevant to land use scenario.

This approach assumes that exposure to multiple carcinogens over a lifetime results in a cumulative effect, and therefore, exposures are summed over all intake routes.

References

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Vapour Intrusion Model Algorithms

The following algorithms, where applicable, were used in the current risk assessment.

1.1 Vapour Emissions in Indoor Air

The algorithms used to predict indoor air vapour concentrations are those described by Johnson and Ettinger (1991). The algorithms estimate a unitless 'vapour attenuation coefficient' (α), which represents the ratio of the indoor vapour concentration (C_{indoor}) to the vapour concentration at the source (C_{source}) found at some depth (L_T) below a foundation. The attenuation coefficient is then used to calculate a volatilization factor (VF), which represents the ratio of concentration of vapour phase hydrocarbon in indoor air to that in the source media (soil, groundwater or PSH), i.e.:

$$VF = \frac{C_{\text{vapour}(m)}}{C_m} \alpha \quad \text{Equation 1}$$

where $C_{\text{vapour}(m)}$ indicates the vapour phase concentration at source media m (soil, groundwater and/or PSH), and C_m indicates the concentration in source media m .

The estimated air concentration is then given by:

$$C_{\text{air}(m)} = C_m VF \quad \text{Equation 2}$$

where $C_{\text{air}(m)}$ is the chemical concentration associated with vapours derived from media m .

Parameter definitions used in the equations below can be found in **Table 1**.

The attenuation coefficient, α , is estimated by:

$$\alpha = \frac{\left[\frac{D_T^{\text{eff}} A_B}{Q_B L_T} \right] \exp\left(\frac{Q_{\text{soil}} L_{\text{crack}}}{D_{\text{crack}}^{\text{eff}} \eta A_B} \right)}{\exp\left(\frac{Q_{\text{soil}} L_{\text{crack}}}{D_{\text{crack}}^{\text{eff}} \eta A_B} \right) + \left[\frac{D_T^{\text{eff}} A_B}{Q_B L_T} \right] + \left[\frac{D_T^{\text{eff}} A_B}{Q_{\text{soil}} L_T} \right] \left(\exp\left(\frac{Q_{\text{soil}} L_{\text{crack}}}{D_{\text{crack}}^{\text{eff}} \eta A_B} \right) - 1 \right)} \quad \text{Equation 3}$$

where $\alpha = C_{\text{indoor}}/C_{\text{source}}$ and other parameters are as listed in **Table 1**.

Q_{soil} can be specified directly, or can be calculated using the following analytical solution of Nazaroff (1988):

$$Q_{\text{soil}} = \frac{2\pi\Delta P k_v X_{\text{crack}}}{\mu \ln(2Z_{\text{crack}}/r_{\text{crack}})} \quad \text{Equation 4}$$

Where r_{crack} is equal to:

$$r_{\text{crack}} = \eta(A_B / X_{\text{crack}}) \quad \text{Equation 5}$$

For cases where Q_{soil} is zero, the above equation simplifies to:

$$\alpha = \frac{\left[\frac{D_T^{eff} A_B}{Q_B L_T} \right]}{1 + \left[\frac{D_T^{eff} A_B}{Q_B L_T} \right] + \left[\frac{D_T^{eff} L_{crack}}{D_{crack}^{eff} L_T \eta} \right]} \quad \text{Equation 6}$$

In the case of a groundwater source, the vapour phase concentration at the source ($C_{source(gw)}$) (mg/cm^3) is estimated by:

$$C_{source(gw)} = C_{gw} H \cdot 10^3 \frac{L}{m^3} \quad \text{Equation 7}$$

If the dissolved phase concentration entered in the model exceeds the pure component aqueous solubility for the chemical, residual phase chemical is considered to be present in the groundwater sample, and the vapour phase concentration is limited to that in equilibrium with the aqueous solubility limit. In this case, the model calculates the vapour source term based on the chemical-specific aqueous solubility, rather than on the groundwater concentration entered in the model.

In the case of a soil source, the vapour phase concentration at the source ($C_{source(s)}$) in mg/m^3 is estimated by:

$$C_{source(s)} = C_{soil} \frac{H \rho_s}{\theta_{ws} + k_{oc} f_{oc} \rho_s + H \theta_{as}} 10^6 \frac{cm^3}{m^3} \frac{kg}{g} \quad \text{Equation 8}$$

When calculating the vapour phase concentration in equilibrium with a soil source, the bulk soil concentration entered in the model is compared to the estimated concentration at which dissolved pore water and vapour phases become saturated (C_{sat}). Where the soil concentration is greater than C_{sat} , residual liquid phase chemical is considered to be present in the soil source area, and the vapour phase concentration is limited to that at C_{sat} . In this case, the model calculates the vapour source term based on C_{sat} , rather than on the bulk soil concentration entered in the model.

In the case of PSH, the vapour phase concentration at the source ($C_{source(P)}$) is estimated from weight fractions in the hydrocarbon source applying Raoult's law, as follows:

$$C_{source(P)} = \left(\frac{\frac{wt.frac_{i,L}}{MW_i}}{\frac{wt.frac_{i,L}}{MW_i} + \frac{(1-wt.frac_{i,L})}{MW_T}} \right) \left(\frac{VP \cdot MW}{P_T MV} \right) 10^6 \frac{mg}{g} \frac{L}{m^3} \quad \text{Equation 9}$$

For groundwater, L_T is equal to the thickness of the capillary fringe (h_{cap}) plus the thickness of the vadose zone (h_v). In practice, the thickness of the vadose zone for the groundwater to indoor air model is estimated based on reported depth to groundwater, less the assumed h_{cap} (typically obtained from literature sources). For soil, L_T is equal to the depth to the subsurface soil contamination source.

For a subsurface soil source, the effective vapour phase diffusion coefficient between the contaminated soil source and the building foundation ($D_{T(s)}^{eff}$) is estimated by:

$$D_{T(s)}^{eff} = D^{air} \frac{\theta_{as}^{3.33}}{\theta_T^2} + D^{wat} \frac{1}{H} \frac{\theta_{ws}^{3.33}}{\theta_T^2} \quad \text{Equation 10}$$

For groundwater, the effective vapour phase diffusion coefficient between the groundwater source and the building foundation ($D_{T(gw)}^{eff}$) is estimated by:

$$D_{T(gw)}^{eff} = \frac{(h_{cap} + h_v)}{\frac{h_{cap}}{D_{cap}^{eff}} + \frac{h_v}{D_s^{eff}}} \quad \text{Equation 11}$$

with

$$D_{cap}^{eff} = D^{air} \frac{\theta_{acap}^{3.33}}{\theta_T^2} + D^{wat} \frac{1}{H} \frac{\theta_{wcap}^{3.33}}{\theta_T^2} \quad \text{Equation 12}$$

For both soil and groundwater sources, the effective diffusion through foundation cracks (D_{crack}^{eff}) is estimated by:

$$D_{crack}^{eff} = D^{air} \frac{\theta_{acrack}^{3.33}}{\theta_T^2} + D^{wat} \frac{1}{H} \frac{\theta_{wcrack}^{3.33}}{\theta_T^2} \quad \text{Equation 13}$$

The algorithms used to predict outdoor vapour concentrations which may result from a surface soil, subsurface soil or groundwater source are those described by ASTM (2010).

1.2 Vapour Emissions in Outdoor Air

The algorithm for groundwater to outdoor air vapour concentration is presented in Equation 14 below based on ASTM (2002):

$$VF_{wamb} = \frac{H}{1 + \left[\frac{U \cdot D \cdot L_T}{W \cdot D_{T(gw)}^{eff}} \right]} \cdot 10^3 \quad \text{Equation 14}$$

Where $D_{T(gw)}^{eff}$ is the estimated groundwater diffusion coefficient in accordance with Equation 11.

The estimated outdoor air concentration is determined by Equation 2 above.

The algorithm for soil to outdoor air vapour concentration is presented in Equation 15 below based on ASTM (2010).

$$VF_{samb} = \frac{H \cdot \rho_s}{\left[\theta_{ws} + k_s \cdot \rho_s + H \cdot \theta_{as} \right] \left[1 + \frac{U_{air} \cdot D \cdot L_T}{D_{T(s)}^{eff} \cdot W} \right]} \quad \text{Equation 15}$$

Where $D_{T(s)}^{eff}$ is estimated by Equation 10.

The estimated outdoor air concentration is determined by Equation 2 above.

Table 1 Vapour Model Parameter Definitions

Parameter/Symbol	Definition
A_B	surface area of the enclosed space in contact with soil (cm^2)
$C_{\text{source}(m)}$	vapour phase concentration at the media (m) source (mg/m^3)
$C_{\text{source}(gw)}$	vapour phase concentration at the groundwater source (mg/m^3)
$C_{\text{source}(s)}$	vapour phase concentration at the soil source (mg/m^3)
$C_{\text{source}(P)}$	vapour phase concentration at the PSH source (mg/m^3)
C_{soil}	Chemical concentration in bulk soil (mg/kg)
C_{gw}	Chemical concentration in groundwater (mg/L)
D^{air}	Chemical-specific diffusion coefficient in air (cm^2/s)
D^{wat}	Chemical-specific diffusion coefficient in water (cm^2/s)
$D^{\text{eff}}_{\text{cap}}$	effective overall vapour-phase diffusion coefficient through capillary fringe (cm^2/s)
$D^{\text{eff}}_{\text{crack}}$	effective overall vapour-phase diffusion coefficient through walls and foundation cracks (cm^2/s)
D^{eff}_T	effective overall vapour-phase diffusion coefficient in soil between the foundation and the depth L_T (cm^2/s)
E_B	Indoor air exchange rate with outdoor air (1/s)
f_{oc}	weight fraction of organic carbon in soil (unitless)
H or HL	unitless Henry's law constant
k_{oc}	Chemical-specific carbon-water sorption coefficient (cm^3/g)
k_v	soil vapour permeability (cm^2)
L_{crack}	the enclosed space foundation thickness (cm)
L_T	the distance (depth) to the vapour source or other point of interest below foundation (cm) (equal to $h_{\text{cap}}+h_v$ for groundwater-derived vapour, or the depth of soil contamination for soil-derived vapour)
MV	gas molar volume (22.4 L/mole at standard temperature and pressure)
MW_i	molecular weight of component i
MW_T	molecular weight of the total mixture
P_T	total pressure of system (assumed to be atmospheric pressure, or 760 mm Hg)
Q_B	the enclosed space volumetric air flow rate (cm^3/s) of fresh air; usually estimated to be the product of the enclosed space volume (V_B) and the indoor air exchange rate with outdoor air (E_B)
Q_{soil}	the pressure-driven soil gas flow rate from the subsurface into the enclosed space (cm^3/s)
V_B	Enclosed space volume (cm^3)
VP	Chemical specific vapour pressure of pure liquid chemical i (mm Hg)
$Wt.\text{frac.}_{i,L}$	weight fraction of component i in the liquid (PSH) source

Parameter/Symbol	Definition
X_{crack}	Building perimeter (cm)
$X_{i,g}$	mole fraction of component i in the vapour phase
$X_{i,L}$	mole fraction of component i in the liquid (PSH) source
Z_{crack}	Depth to bottom of slab (cm)
ΔP	pressure differential between soil surface and the enclosed space ($\text{g}/\text{cm}^2\text{-s}$)
η	the fraction of the enclosed space surface area open for vapour intrusion (unitless), i.e., the areal fraction of cracks in the foundation/slab
μ	viscosity of air ($1.81 \times 10^{-4} \text{ g}/\text{cm-s}$)
ρ_s	soil bulk density (g/cm^3)
θ_{as}	volumetric air content in vadose zone soils (unitless)
θ_{ws}	volumetric water content in vadose zone soils (unitless)
θ_{ws}	volumetric water content in vadose zone soils (unitless)
θ_T	total soil porosity (unitless)
θ_{acap}	volumetric air content in capillary zone soils (unitless)
θ_{wcap}	volumetric water content in capillary zone soils (unitless)
θ_{acrack}	volumetric air content in foundation/wall cracks (unitless)
θ_{wcrack}	volumetric water content in foundation/wall cracks (unitless)
E	air emissions from the liquid surface (g/s)
VF	volatisation factor
VF_{wamb}	volatisation factor groundwater to ambient (outdoor air) vapours
VF_{samb}	volatisation factor surficial soils to ambient (outdoor air) vapours
K	overall mass transfer coefficient (m/s)
A	liquid surface area (m^2)
C_L	concentration of constituent in the liquid phase
U_{10}	windspeed at 10 m above the liquid surface (m/s)
D_w	diffusivity of constituent in water (cm^2/s)
D_{ether}	diffusivity of ether in water (cm^2/s), adopted $8.5 \times 10^{-5} \text{ cm}^2/\text{s}$
K	overall mass transfer coefficient (m/s)
k_L	liquid phase mass transfer coefficient (m/s)
k_G	gas phase mass transfer coefficient (m/s)
K_{eq}	equilibrium constant or partition coefficient, concentration in gas phase/ concentration in liquid phase
K_s	soil-water sorption coefficient $\text{gH}_2\text{O}/\text{g soil}$
U	windspeed (m/s)
W	width of source area parallel to wind or groundwater flow direction (cm)

Parameter/Symbol	Definition
Sc_G	schmidt number on gas side
μ_G	viscosity of air (g/cm sec): 1.86×10^{-4}
ρ_G	density of air (g/cm ³): 1.29×10^{-3}
D	ambient air mixing height (cm)
D_a	diffusivity of constituent in air (cm ² /s)
D_e	effective diameter of impoundment
A	area of impoundment (m ²)
Q	volumetric flow rate (m ³ /s)
C_i	initial concentration in waste (g/m ³)
C_L	equilibrium of bulk concentration in the impoundment (g/m ³)
K	overall mass transfer coefficient (m/s)
A	liquid surface area (m ²)
Vol _B	Volume of the basement (m ³)
Air _{Exc}	Air exchange rate in basement (exchanges per day)

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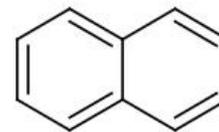
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Appendix I

Toxicity Profiles



Naphthalene

The majority of the following information has been sourced from ATSDR (2005), with other sources listed in the references.

Chemical Identification

Synonyms: White tar, mothballs, naphthalin, moth flakes, camphor tar, tar camphor, naphthaline, antimite, albocarbon

CAS: 91-20-3

Molecular Formula: C₁₀H₈

Molecular Weight: 128.18 g/mol

General

Naphthalene occurs naturally in fossil fuels such as petroleum and coal, and is produced when organic materials (e.g., fossil fuels, wood, tobacco) are burned. Naphthalene is also produced commercially from either coal tar or petroleum. Commercially-produced naphthalene is predominately used in the production of phthalic anhydride, which is used as an intermediate for polyvinyl chloride plasticizers such as di(2-ethylhexyl) phthalate. Other uses of naphthalene include production of naphthalene sulfonates (used in concrete additives and synthetic tanning agents), pesticides (e.g., carbaryl insecticides and moth repellents), and dye intermediates.

Naphthalene is frequently present in industrial and automobile emissions and effluents and in various media in the general environment due to its natural occurrence in coal and petroleum products and emissions, its use as an intermediate in the production of plasticizers, resins, and insecticides, and its use in a variety of consumer products such as moth repellants.

Significance of Exposure Pathways and Background

The most significant exposure pathway is inhalation of contaminated air and tobacco smoke from both active and passive smoking. Although naphthalene has been detected in certain foods, beverages and tap water, these do not constitute major sources of exposure for most people (ATSDR, 2005).

Typical air concentrations for naphthalene are low, 0.2 ppb or less. Studies of outdoor air reported concentrations of 0.09 ppb 1-methylnaphthalene and 0.011 ppb 2-methylnaphthalene. In homes or businesses where cigarettes are smoked, wood is burned, or moth repellents are used, the levels of naphthalene, 1-methylnaphthalene, and 2-methylnaphthalene in the air are higher. Studies of indoor air typically report that average indoor air concentrations of these contaminants are less than 1 ppb (ATSDR, 2005).

As summarised in **Table 1** below, estimated chemical intakes from background exposure to PAHs (including naphthalene) in soil, drinking water, food and air are less than 1% of the lowest TDI adopted in this assessment. Background exposure was therefore not considered to be significant in comparison to the adopted dose-response criteria and TDIs were not corrected for background exposure.

Table 1 Estimated Background Exposure to PAHs

Concentration of Intake	Estimated Intake (mg/kg/day)	Notes
Drinking Water		
623 ng/L	1.78×10^{-5}	Water concentration is the maximum reported individual PAH concentration for a range of drinking water sources monitored in the USA, United Kingdom and Europe (ATSDR, 1995). Use of this value is considered conservative given that PAHs have not been reported in Australian drinking water supplies (NHMRC, 2011). Intake has been estimated for a 70 kg adult, assuming that 2 L of water per day is ingested.

Concentration of Intake	Estimated Intake (mg/kg/day)	Notes
Food		
10 µg/day	1.4×10^{-4}	Food intake is the maximum reported for individual PAHs based on a range of studies in the USA, United Kingdom and Europe (ATSDR, 1995). Intake has been calculated assuming a 70 kg body weight.
Air		
10.9 ng/m ³	3.1×10^{-5}	Air concentration is maximum annual average of any individual PAH reported in Australian cities by Environmental Australia (DEH, 1999). Intake has been calculated assuming a 70 kg adult respire 20 m ³ of air per day.
Total Intake		0.00019 mg/kg/day
Minimum TDI¹		0.02 mg/kg/day
Fraction of TDI Due to Background Exposure		0.0096 (<1%)

Notes

¹Value is minimum TDI of those adopted for PAHs assessed on the basis of threshold dose-response criteria. TDIs ranged from 0.02 mg/kg/day (naphthalene) to 0.3 mg/kg/day (anthracene).

It is noted that the PAH background exposure analysis presented in this report has not explicitly considered background exposure to PAH by smokers. However, ATSDR (1995) report that concentrations of individual PAHs in cigarette smoke range from less than 1 µg per 100 cigarettes to 62 µg per 100 cigarettes. For a 70 kg individual who smokes one pack (20 cigarettes) per day, the maximum expected intake of any individual PAH is therefore estimated to be 0.00018 mg/kg/day. This additional intake due to smoking also represents less than 1% of the lowest PAH TDI considered in this assessment (0.02 mg/kg/day for naphthalene).

Background exposure is not considered in the assessment of carcinogenic (non-threshold) risks, as non-threshold risks are estimated *incremental* lifetime cancer risks. However, it should be noted that the maximum background intake estimated for individual PAHs, if assumed to apply to benzo(a)pyrene or benzo(a)pyrene toxic equivalents, would result in an estimated incremental lifetime cancer risk of approximately 9 in 100,000, which is greater than the acceptable incremental cancer risk of 1 in 100,000 adopted for this assessment. However, this cancer risk estimate should not be interpreted as an indication that exposure to background levels of PAH may cause a significant increase in cancer rates above baseline levels as the baseline lifetime risk of cancer is reported to be approximately 50% (one in two; NRC, 2006). Thus the adopted acceptable incremental cancer risk of 1 in 100,000 and the estimated cancer risk due to background concentrations of PAHs in the environment are still very low in comparison to baseline lifetime cancer risks for the population as a whole.

Non-Carcinogenic Health Effects

Reports that establish associations between naphthalene exposure and health effects in humans are restricted to numerous reports of hemolytic anemia or cataracts following acute exposure or occupational exposure to naphthalene, either by ingestion or by inhalation of naphthalene vapours, but these reports have not identified exposure levels associated with these effects. A relationship appears to exist between an inherited deficiency in the enzyme, glucose 6-phosphate dehydrogenase (G6PD), and susceptibility to naphthalene-induced hemolysis. Newborn infants also appear to be susceptible to naphthalene-induced hemolysis presumably due to a decreased ability to conjugate and excrete naphthalene metabolites.

Results from animal studies exposed to naphthalene by oral administration, by inhalation exposure, or by parenteral administration identify several health effects of potential concern for humans, including maternal toxicity during pregnancy with acute oral exposure, decreased body weight (without lesions developing in any tissues or organs) with intermediate oral exposure, and increased incidence of non-neoplastic and neoplastic lesions in the nose (in rats and mice) and the lung (in mice only) with chronic inhalation exposure.

Identification of Non-Carcinogenic Toxicity Reference Values

The dose-response values provide an estimate of exposure to the human population that is likely to be without an appreciable risk of deleterious effects during a lifetime. The oral exposure represents a daily value and the inhalation exposure represents continuous inhalation. These values are intended for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action (USEPA, 2012). Available chronic dose-response values published by sources recognised and endorsed by enHealth (enHealth, 2012), NEPC (NEPC, 1999) and the USEPA and are summarised below.

Oral

The oral dose-response value is expressed in units of mg/kg/day. This value may be termed a Reference Dose (RfD), Tolerable Daily Intake (TDI), or Minimal Risk Level for oral exposure (MRL) depending on the agency of derivation.

Table 1 Published Threshold Dose-Response Values for Naphthalene - Oral

Agency	Oral Dose-Response Value (mg/kg/day)	Source	Target Endpoint	Test Animal	UF	Notes
ATSDR	0.6	ATSDR (2005)	Body weight, organ weight, haematological parameters	Rats and mice	100	Intermediate duration MRL. Based on three studies by NTP, 1980a; NTP, 1980b and Shopp et al., 1984,
IRIS	0.02	USEPA (1998)	Decreased body weight	Rats	3000	RfD. Based on Study by BCL, 1980
NSF International	0.02	NSF International (1998)	Decreased body weight	Rats	3000	RfD. Based on IRIS (USEPA, 2013)
RIVM	0.04	RIVM (2000)	-	-	-	TDI. Aromatic compound with equivalent carbon No. of >C ₉ -C ₁₆ .

ATSDR – Agency for Toxic Substances and Disease Registry

IRIS – Integrated Risk Information System.

RIVM – National Institute of Public Health and the Environment (Netherlands)

UF – Uncertainty Factor.

Inhalation

The inhalation dose-response value is expressed in units of mg/m³. This value may be termed a Reference Concentration (RfC), Tolerable Concentration (TC), or Minimal Risk Level for inhalation (MRL) depending on the agency of derivation.

Table 2 Published Threshold Dose-Response Values for Naphthalene - Inhalation

Agency	Inhalation Dose-Response Value (mg/m ³)	Source	Target Endpoint	Test Animal	UF	Notes
ATSDR	0.0037*	ATSDR (2005)	Nonneoplastic lesions in nasal olfactory epithelium	Rats and mice	300	Chronic MRL. Based on study by NTP, 1992; NTP, 2000 and Abdo et al., 2001.
IRIS	0.003	USEPA (1998)	Nasal effects	Mice	3000	RfC. Based on Study by NTP, 1991

ATSDR – Agency for Toxic Substances and Disease Registry

IRIS – Integrated Risk Information System.

*Converted from 0.0007 ppm to mg/m³ using the molecular weight of 128.18g/mol and assuming 25°C and 760 mm Hg

Carcinogenicity and Genotoxicity

The only studies of cancer in humans exposed to naphthalene are two case series reports of cancer; one report of four laryngeal cancer cases (all of whom were smokers) among workers in a naphthalene purification plant in East Germany, and another report of 23 cases of colorectal carcinoma admitted to a hospital in Nigeria. NTP, USEPA, and IARC concur that these studies provide inadequate evidence of naphthalene carcinogenicity in humans. No cohort mortality or morbidity studies or case-control studies examining possible associations between naphthalene exposure and increased risk of cancer (or other health effects) are available.

In animals, chronic inhalation studies have found increased incidences of non-neoplastic and neoplastic lesions in the nose of rats, non-neoplastic lesions in the nose of mice, and neoplastic and non-neoplastic lesions in the lungs of mice. In mice of both sexes, chronic inhalation of 10 or 30 ppm naphthalene induced inflammation of the nose and lung, metaplasia of the olfactory epithelium, and hyperplasia of the nasal respiratory epithelium. In female mice (but not male mice), exposure to 30 ppm (but not 10 ppm) increased the incidence of benign lung tumors (alveolar/bronchiolar adenomas) compared with controls. One other female mouse exposed to 30 ppm showed a malignant lung tumor (alveolar/bronchiolar carcinoma). In rats of both sexes, inhalation of 10, 30, or 60 ppm naphthalene induced non-neoplastic and neoplastic lesions only in the nasal cavity. Non-neoplastic nasal lesions included (1) hyperplasia, atrophy, chronic inflammation, and hyaline degeneration of the olfactory epithelium and (2) hyperplasia, metaplasia or degeneration of the respiratory epithelium or glands. Neoplastic lesions associated with naphthalene exposure in rats were olfactory epithelial neuroblastoma (a rare malignant tumor) and respiratory epithelial adenoma.

The mechanisms by which naphthalene causes non-neoplastic or neoplastic lesions in the respiratory tract of rodents are incompletely understood, but are thought to involve reactive metabolites of naphthalene, including 1,2-naphthalene oxide, 1,2-naphthoquinone, 1,4-naphthoquinone, and possibly 1,2-dihydroxy-3,4-epoxy-1,2,3,4-tetrahydronaphthalene.

Comparison of species susceptibility to naphthalene-induced non-neoplastic lung damage suggests that mice are much more sensitive than rats (e.g., non-neoplastic or neoplastic lung lesions were not found in chronically exposed rats in an NTP study) and that differences in rates and stereoselectivity of naphthalene metabolism to epoxide intermediates may be involved in this species difference. Acute (4-hour) inhalation exposure of mice to naphthalene concentrations as low as 2–10 ppm induced lung injury, whereas rats exposed to naphthalene concentrations as high as 110 ppm showed no signs of lung injury. Some evidence has been reported that rates and stereoselectivity of naphthalene metabolism in primate lung tissue may be more like rats than mice. In vitro studies with microsomes from lymphoblastoid cells, which expressed recombinant human CYP2F1, metabolism of naphthalene to epoxide intermediates was demonstrated, but the predominant enantiomeric form produced (1S,2R-oxide) was different from the form (1R,2S-oxide) produced by mouse CYP2F2. Although these observations on epoxide formation may suggest that mice may be more sensitive than humans to acute naphthalene lung toxicity from epoxide intermediates, the possible role of other potentially reactive metabolites of naphthalene (e.g., the naphthoquinone metabolites) is unknown with chronic exposure scenarios. To date, mechanistic understanding of species differences in naphthalene bioactivation in the lung is too incomplete to definitively rule out the possible human relevance of naphthalene-induced lung lesions in mice.

In contrast, the olfactory epithelium and respiratory epithelium of the nose of rats and mice do not appear to differ in sensitivity to naphthalene non-neoplastic toxicity from chronic inhalation exposure.

Non-neoplastic nasal lesions were found in nearly all exposed animals of both species at the lowest exposure level, 10 ppm, in both chronic studies. CYP monooxygenases, which might be involved in naphthalene metabolism and bioactivation, have been demonstrated to exist in nasal respiratory epithelial and olfactory epithelial tissue from rodents and humans. Studies designed to specifically characterize metabolism of naphthalene in nasal tissue, however, have not been conducted, with the exception of a single study, which examined in vitro rates of metabolism of naphthalene to naphthalene oxides in postmitochondrial supernatants from mouse, rat, and hamster olfactory tissue. Metabolic rates (units of nmol/min/mg protein) showed the following order: mouse (87.1) > rat (43.5) > hamster (3.9). This order did not correspond with species differences in sensitivity to single intraperitoneal injections of naphthalene in a companion study. The lowest dose levels producing substantial necrosis and exfoliation in olfactory epithelium were 200 mg/kg in rats and 400 mg/kg in mice and hamsters. To date, mechanistic understanding of species differences in naphthalene bioactivation in the

respiratory tissues is too incomplete to definitively rule out the possible human relevance of naphthalene-induced nasal lesions in rodents (non-neoplastic lesions in rats and mice and neoplastic lesions in rats).

It is unknown whether the naphthalene-induced neoplastic lesions found in mice (lung adenomas) and rats (nose respiratory epithelial adenomas and olfactory epithelial neuroblastomas) are produced via a genotoxic mode of action or a non-genotoxic mode requiring tissue damage and regenerative responses as precursor events. Results from genotoxicity tests for naphthalene have been predominately (but not completely) negative, and the general sites of neoplastic lesions, the nose in rats and the lungs in mice, show some correspondence (but not complete) with the general sites of non-neoplastic lesions. However, mechanistic understanding of naphthalene's carcinogenic mode of action is too incomplete to rule out the possibility of a genotoxic mode of action. Key issues that remain unexplained or unstudied include:

- The possible significance of the few positive genotoxicity results that have been obtained, including: reverse mutations in *Salmonella typhimurium* by 1,2-naphthoquinone; in vitro formation of N-7 guanine adducts of DNA by 1,2-naphthoquinone; reverse mutations for luminescence in the marine bacteria, *Vibrio fischeri*, by naphthalene; induction of sister chromatid exchanges in Chinese hamster ovary cells by naphthalene and in human mononuclear leukocytes by 1,2- or 1,4-naphthoquinone; induction of chromosomal aberrations in Chinese hamster ovaries and preimplantation mouse embryos by naphthalene; induction of somatic mutations and recombination in *Drosophila melanogaster* by naphthalene; and weak (about 2-fold) induction of micronuclei in red blood cells from *Pleurodeles waltl* larvae by naphthalene;
- The lack of a mechanistic explanation of why nearly all rats and mice develop nasal non-neoplastic lesions following chronic exposure to naphthalene at concentrations ≥ 10 ppm, but only some rats develop nasal tumors;
- The lack of a mechanistic explanation of why both male and female mice exposed to naphthalene show similar incidences of chronic lung inflammation following chronic exposure to 10 or 30 ppm, but only female mice showed statistically significant increased incidence of lung tumors;
- The lack of in vivo genotoxicity assays involving target tissues of naphthalene carcinogenicity (nose and lung); and
- The lack of information on the possible threshold exposure levels for nonneoplastic nasal lesions in rats and mice at air concentrations < 10 ppm.

The National Toxicology Program 11th Report on Carcinogens includes naphthalene in its list of chemicals reasonably anticipated to be human carcinogen.

International Agency for Research on Cancer concluded that naphthalene is possibly carcinogenic to humans (Group 2B) based on specific evaluations that there is inadequate evidence in humans and sufficient evidence in animals for the carcinogenicity of naphthalene. IARC considered the findings for nasal tumors in male and female rats and lung tumors in female mice in the NTP bioassays as sufficient evidence, noting that both nasal tumor types (olfactory epithelial neuroblastomas and respiratory epithelial adenomas) are rare in untreated rats.

The USEPA last assessed the carcinogenicity of naphthalene before the availability of the results from the chronic rat bioassay. In the EPA (1998) Toxicological Review on Naphthalene, it was concluded that there was inadequate evidence in humans and limited evidence in animals of naphthalene carcinogenicity (increased incidence of lung tumors in female mice). Under the EPA 1986 cancer guidelines, naphthalene was assigned to Group C - possible human carcinogen. Under the EPA 1996 proposed cancer guidelines, it was judged that the human carcinogenic potential of naphthalene via the oral or inhalation routes "cannot be determined", but it was noted that there was suggestive evidence of potential human carcinogenicity based on increased lung tumors in female mice. Currently, the EPA Integrated Risk Information System (IRIS) Office is reassessing the inhalation carcinogenicity of naphthalene.

Identification of Carcinogenic Toxicity Reference Values

Oral and Inhalation

ATSDR, RIVM, and USEPA have evaluated the carcinogenicity data for naphthalene. EPA classifies this compound as Group C, a possible human carcinogen, using criteria of the 1986 cancer guidelines (USEPA, 1986). Using the 1996 Proposed Guidelines for Carcinogen Risk Assessment (USEPA, 1996), the human carcinogenic potential of naphthalene via the oral or inhalation routes "cannot be determined" at this time based on human and animal data; however, there is suggestive evidence [observations of benign respiratory tumors and

one carcinoma in female mice only exposed to naphthalene by inhalation (NTP, 1992)]. Additional support includes increase in respiratory tumors associated with exposure to 1-methylnaphthalene. An oral slope factor or inhalation unit risk for naphthalene was not derived because of a lack of chronic oral naphthalene studies. RIVM determined that naphthalene is not carcinogenic, and therefore, based its risk estimate on the threshold approach. ATSDR has published a Toxicological Profile for Naphthalene. Although ATSDR discusses the carcinogenicity data in its Toxicological Profiles, it does not currently assess cancer potency or perform cancer risk assessments.

Adopted Dose-Response Values

Dose-response values for threshold and non-threshold effects associated with oral or inhalation exposure to naphthalene have not been published by Australian regulatory bodies.

Threshold (Non-Carcinogenic)

For assessment of potential threshold effects associated with oral exposure to naphthalene, AECOM has adopted the reference dose of **0.02 mg/kg/day** published by USEPA (IRIS database).

For assessment of potential threshold effects associated with inhalation exposure to naphthalene, AECOM has adopted the inhalation reference concentration of **0.0037 mg/m³** published by ATSDR (ATSDR, 2005).

Non-Threshold (Carcinogenic)

For assessment of potential non-threshold effects associated with oral and inhalation exposure to naphthalene, no international values were available to adopt.

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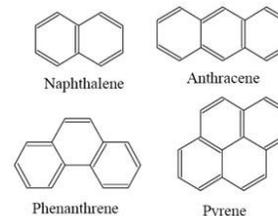
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Polycyclic Aromatic Hydrocarbons (PAHs)

General

Polycyclic aromatic hydrocarbons (PAHs) occur ubiquitously in the environment from both synthetic and natural sources. PAHs occur in the atmosphere most commonly in the products of incomplete combustion. These products include fossil fuels; cigarette smoke; industrial processes (such as coke production and refinement of crude oil); and exhaust emissions from gasoline engines, oil-fired heating, and burnt coals. PAHs are present in groundwater, surface water, drinking water, waste water, and sludge. They are found in foods, particularly charbroiled, broiled, or pickled food items, and refined fats and oils. Individuals living in the vicinity of hazardous waste sites where PAHs have been detected at levels above background may experience exposure to these chemicals via inhalation of contaminated air or ingestion of contaminated food, soil, or water.

Within Australia, the following 16 PAHs are typically analysed for and considered as a group in contaminated site assessment work:

- acenaphthene
- acenaphthylene
- anthracene
- benz[a]anthracene
- benzo[a]pyrene
- benzo[b]fluoranthene
- benzo[g,h,i]perylene
- benzo[k]fluoranthene
- chrysene
- dibenz[a,h]anthracene
- fluoranthene
- fluorene
- naphthalene
- indeno[1,2,3-c,d]pyrene
- phenanthrene
- pyrene

These PAHs are the most commonly assessed based on the following considerations:

- More information is available on them than other PAHs.
- They are suspected to be more harmful than other PAHs, and they exhibit harmful effects that are representative of the PAHs.
- There is considered to be a greater chance of exposure to these PAHs than to the others.

The above PAHs with the exception of naphthalene are considered in this toxicity profile. The toxicity of naphthalene (if considered in this risk assessment) is discussed separately.

Significance of Exposure Pathways and Background

The most significant exposure pathways are inhalation of contaminated air and ingestion of certain foods, beverages and tap water (ATSDR, 1995).

Background levels of some representative PAHs in the air are reported to be 0.02-1.2 nanograms per cubic meter (ng/m^3) in rural areas and 0.15-19.3 ng/m^3 in urban areas (ATSDR, 1995).

As summarised in **Table 1** below, estimated chemical intakes from background exposure to PAHs in soil, drinking water, food and air are less than 1% of the lowest TDI adopted in this assessment. Background exposure was therefore not considered to be significant in comparison to the adopted dose-response criteria and TDIs were not corrected for background exposure.

Table 1 Estimated Background Exposure to PAHs

Concentration of Intake	Estimated Intake (mg/kg/day)	Notes
Drinking Water		
623 ng/L	1.78×10^{-5}	Water concentration is the maximum reported individual PAH concentration for a range of drinking water sources monitored in the USA, United Kingdom and Europe (ATSDR, 1995). Use of this value is considered conservative given that PAHs have not been reported in Australian drinking water supplies (NHMRC, 2011). Intake has been estimated for a 70 kg adult, assuming that 2 L of water per day is ingested.
Food		
10 µg/day	1.4×10^{-4}	Food intake is the maximum reported for individual PAHs based on a range of studies in the USA, United Kingdom and Europe (ATSDR, 1995). Intake has been calculated assuming a 70 kg body weight.
Air		
10.9 ng/m ³	3.1×10^{-5}	Air concentration is maximum annual average of any individual PAH reported in Australian cities by Environmental Australia (DEH, 1999). Intake has been calculated assuming a 70 kg adult respire 20 m ³ of air per day.
Total Intake		0.00019 mg/kg/day
Minimum TDI¹		0.02 mg/kg/day
Fraction of TDI Due to Background Exposure		0.0096 (<1%)

Notes

¹Value is minimum TDI of those adopted for PAHs assessed on the basis of threshold dose-response criteria. TDIs ranged from 0.02 mg/kg/day (naphthalene) to 0.3 mg/kg/day (anthracene).

It is noted that the PAH background exposure analysis presented in this report has not explicitly considered background exposure to PAH by smokers. However, ATSDR (1995) report that concentrations of individual PAHs in cigarette smoke range from less than 1 µg per 100 cigarettes to 62 µg per 100 cigarettes. For a 70 kg individual who smokes one pack (20 cigarettes) per day, the maximum expected intake of any individual PAH is therefore estimated to be 0.00018 mg/kg/day. This additional intake due to smoking also represents less than 1% of the lowest PAH TDI considered in this assessment (0.02 mg/kg/day for naphthalene).

Background exposure is not considered in the assessment of carcinogenic (non-threshold) risks, as non-threshold risks are estimated *incremental* lifetime cancer risks. However, it should be noted that the maximum background intake estimated for individual PAHs, if assumed to apply to benzo(a)pyrene or benzo(a)pyrene toxic equivalents, would result in an estimated incremental lifetime cancer risk of approximately 9 in 100,000, which is greater than the acceptable incremental cancer risk of 1 in 100,000 adopted for this assessment. However, this cancer risk estimate should not be interpreted as an indication that exposure to background levels of PAH may cause a significant increase in cancer rates above baseline levels as the baseline lifetime risk of cancer is reported to be approximately 50% (one in two; NRC, 2006). Thus the adopted acceptable incremental cancer risk of 1 in 100,000 and the estimated cancer risk due to background concentrations of PAHs in the environment are still very low in comparison to baseline lifetime cancer risks for the population as a whole.

Non-Carcinogenic Health Effects

Noncancer adverse health effects associated with PAH exposure have been observed in animals but generally not in humans (with the exception of adverse hematological and dermal effects). Animal studies demonstrate that PAHs tend to affect proliferating tissues such as bone marrow, lymphoid organs, gonads, and intestinal epithelium.

Identification of Non-Carcinogenic Toxicity Reference Values

The dose-response values provide an estimate of exposure to the human population that is likely to be without an appreciable risk of deleterious effects during a lifetime. The oral exposure represents a daily value and the inhalation exposure represents continuous inhalation. These values are intended for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action (USEPA, 2012). Available chronic dose-response values published by sources recognised and endorsed by enHealth (enHealth, 2012), NEPC (NEPC, 1999) and the USEPA and are summarised below.

Oral

The oral dose-response value is expressed in units of mg/kg/day. This value may be termed a Reference Dose (RfD), Tolerable Daily Intake (TDI), or Minimal Risk Level for oral exposure (MRL) depending on the agency of derivation.

Table 1 Published Threshold Dose-Response Values for PAHs - Oral

PAH	Agency	Oral Dose-Response Value (mg/kg/day)	Source	Target Endpoint	Test Animal	UF	Notes
Acenaphthene	IRIS	0.06	USEPA, 1994	Hepatotoxicity	Mice	3000	RfD. USEPA, data last reviewed in 1994
	ATSDR	0.6	ATSDR, 1995	Liver weight	Mice	300	MRL for intermediate duration exposure. USEPA, 1989
Anthracene	RIVM	0.04	RIVM, 2000	-	-	-	TDI based on the RIVM TDI for TPH >C9-C16. Baars et.al., 2001.
	IRIS	0.3	USEPA, 1993	No observed effects	Mice	3000	RfD. USEPA, data last reviewed in 1993
	ATSDR	10	ATSDR, 1995	Liver effects.	Mice	100	MRL for intermediate duration exposure. USEPA, 1989
Fluoranthene	IRIS	0.04	USEPA, 1993	Nephropathy, increased liver weights, hematological alterations, and clinical effects	Mice	3000	RfD. USEPA, data last reviewed in 1993.
	ATSDR	0.4	ATSDR, 1995	Liver weight	Mice	300	MRL for intermediate duration exposure. USEPA, 1988
Fluorene	RIVM	0.04	RIVM, 2000	-	-	-	TDI based on the RIVM TDI for TPH >C9-C16. Baars et.al., 2001.

PAH	Agency	Oral Dose-Response Value (mg/kg/day)	Source	Target Endpoint	Test Animal	UF	Notes
	IRIS	0.04	USEPA, 1990	Decreased red blood cells, packed cell volume and hemoglobin	Mice	3000	RfD. USEPA, data last reviewed in 1990.
	ATSDR	0.4	ATSDR, 1995	Liver weight	Mice	300	MRL for intermediate duration exposure. USEPA, 1989
Phenanthrene	RIVM	0.04	RIVM, 2000	-	-	-	TDI based on the RIVM TDI for TPH >C9-C16. Baars et.al., 2001.
Pyrene	IRIS	0.03	USEPA, 1993	Kidney effects	Mice	3000	RfD USEPA, data last reviewed in 1993.

ATSDR – Agency for Toxic Substances and Disease Registry

IRIS – Integrated Risk Information System.

RIVM – National Institute of Public Health and the Environment (Netherlands)

UF – Uncertainty Factor.

Inhalation

US EPA and other agencies have not published dose-response values for assessment of threshold health effects associated with inhalation exposure to PAHs. Cancer health effects of PAHs were considered to be the primary risk driver via the inhalation pathway, and will be assessed assuming carcinogenic health effects (see below).

Carcinogenicity and Genotoxicity

Carcinogenicity

Evidence exists to indicate that mixtures of PAHs are carcinogenic in humans. The evidence in humans comes primarily from occupational studies of workers exposed to mixtures containing PAHs as a result of their involvement in such processes as coke production, roofing, oil refining, or coal gasification (e.g., coal tar, roofing tar, soot, coke oven emissions, soot, crude oil) (Hammond et al. 1976; Lloyd 1971; Maclure and MacMahon 1980; Mazumdar et al. 1975; Redmond et al. 1976; Wynder and Hoffmann 1967). PAHs, however, have not been clearly identified as the causative agent. Cancer associated with exposure to PAH-containing mixtures in humans occurs predominantly in the lung and skin following inhalation and dermal exposure, respectively. Some ingestion of PAHs is likely because of swallowing of particles containing PAHs subsequent to mucociliary clearance of these particulates from the lung.

Certain PAHs are carcinogenic to animals by the oral route (e.g., benz[a]anthracene, benzo[a]pyrene, and dibenz[a,h]anthracene). The results of dermal studies indicate that benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-c,d]pyrene are tumorigenic in mice following dermal exposure. The sensitivity of mouse skin to PAH tumorigenesis forms the basis for the extensive studies performed using dermal administration. This tumorigenicity can be enhanced or modified with concomitant exposure to more than one PAH, long straight-chain hydrocarbons (i.e., dodecane), or similar organic compounds commonly found at hazardous waste sites. Thus, humans exposed to PAHs in combination with these substances could be at risk for developing skin cancer.

For many of the carcinogenic PAHs, it appears that the site of tumor induction is influenced by the route of administration and site of absorption, i.e., forestomach tumors are observed following ingestion, lung tumors following inhalation, and skin tumors following dermal exposure. However, the observations that (1) mammary tumors are induced following intravenous injection in Sprague-Dawley rats, (2) the susceptibility to tumor development on the skin after dermal application is not similar in rats and mice, and (3) oral cavity tumors are not

observed when benzo[a]pyrene is administered in the diet, suggest that the point of first contact may not always be the site of PAH-induced tumors.

Genotoxicity

Benzo[a]pyrene has been thoroughly studied in genetic toxicology test systems, and has been found to induce genetic damage in prokaryotes, eukaryotes, and mammalian cells in vitro, and to produce a wide range of genotoxic effects (gene mutations in somatic cells, chromosome damage in germinal and somatic cells, DNA adduct formation, UDS, sister chromatid exchange, and neoplastic cell transformation). In cultured human cells, benzo[a]pyrene binds to DNA and causes gene mutations, chromosome aberrations, sister chromatid exchange, and UDS.

The results of in vivo studies indicate that many of the same types of adverse effects observed in vitro were seen in mice, rats, and hamsters exposed to benzo[a]pyrene via the oral, dermal, or intraperitoneal routes. The available data also indicate that benzo[a]pyrene is genotoxic in both somatic and germinal cells of intact animals. The only study that was found regarding genotoxic effects in humans following exposure to benzo[a]pyrene reported no correlation between aluminium plant workers' exposure to PAHs, including benzo[a]pyrene, and sister chromatid exchange frequency. The findings from assays using human cells as the target, in conjunction with the data from whole animal experiments, suggest that benzo[a]pyrene would probably have similar deleterious effects on human genetic material.

Because the genotoxic activity of benzo[a]pyrene is well established, it is frequently used as a positive control to demonstrate the sensitivity of various test systems to detect the genotoxic action of unknown compounds. It also serves as the model compound for PAHs, and the available information on the formation of metabolites and structure of benzo[a]pyrene can theoretically be used to predict potential genotoxicity/carcinogenicity of other PAHs that have not been as extensively studied.

Epoxidation is thought to be the major pathway for benzo[a]pyrene metabolism pertinent to macromolecular interaction. The metabolic attack consists of the cytochrome P-450/P-448-dependent MFO system converting the benzo[a]pyrene molecule into an epoxide; the epoxide is acted upon by epoxide hydrolase to form a dihydrodiol, and a second cytochrome MFO reaction gives rise to the ultimate mutagenic/carcinogenic form, benzo[a]pyrene 7,8-diol-9,10-epoxide. One of the unique structural features of the diol epoxide is that it appears to form in the area of the PAH molecule referred to as the bay region (i.e., a deep-pocketed area formed when a single benzo ring is joined to the remainder of the multiple ring system to form a phenanthrene nucleus).

Analysis of the bay region diol epoxides and their contribution to the DNA binding, genotoxicity, and carcinogenicity of various PAHs has provided the basis for the bay region hypothesis. For example, DNA adducts formed with non-bay region diol epoxides of benzo[a]pyrene have low mutagenic potential. The hypothesis further predicts that structures with more reactive bay regions would probably be more genotoxic and more carcinogenic. The body of evidence on the mutagenic and tumorigenic activity of the PAHs that form bay region diol epoxides (benzo[a]pyrene, benz[a]anthracene, chrysene, dibenz[a,h]anthracene; benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, and indeno[1,2,3-c,d]pyrene) supports this hypothesis.

In summary, several general conclusions can be reached for the unsubstituted PAHs evaluated in this profile. The formation of diol epoxides that covalently bind to DNA appears to be the primary mechanism of action for both genotoxicity and carcinogenicity of several of the unsubstituted PAHs that are genotoxins (benzo[a]pyrene, benz[a]anthracene, dibenz[a,h]anthracene, chrysene, benzo[b]fluoranthene, benzo[j]fluoranthene). There was insufficient evidence to draw meaningful conclusions regarding the genotoxic potential of benzo[g,h,i]perylene, although some evidence does exist.

With regard to the unsubstituted PAHs that either lack a bay region configuration (acenaphthene, acenaphthylene, anthracene, fluorene, and pyrene) or appear to have a weakly reactive bay region (phenanthrene), there is no compelling evidence to suggest that they interact with or damage DNA.

The five PAHs that appear to be exceptions to the bay region diol epoxide hypothesis are fluoranthene, benzo[k]fluoranthene, benzo[j]fluoranthene, and indeno[1,2,3-cd]pyrene (no bay region), and benzo[e]pyrene (two bay regions). The evidence does suggest, however, that fluoranthene possesses genotoxic properties while benzo[e]pyrene is either weakly mutagenic or nonmutagenic.

Identification of Carcinogenic Toxicity Reference Values

Oral

The oral dose-response value is expressed in units of (mg/kg/day)⁻¹. This value may be termed a Cancer Slope Factor (CSF), a Cancer Risk (CR), Slope Factor (SF), or Risk Specific Dose (RSD) depending on the agency of derivation. Some agencies present guideline values which may be converted to dose-response values. Breath

Table 2 Published Non-Threshold Dose-Response Values for Benzo(a)pyrene – Oral

Agency	Oral Cancer Slope Factor (mg/kg/day) ⁻¹	Source	Target Endpoint	Test Animal	Notes
NHMRC	0.43	NHMRC, 2011	Weight change	Mouse	Derived from drinking water unit risk of 1x10 ⁻⁶ per 0.00007 mg/L, assuming 70 kg body weight and 2 L/day water ingestion rate.
WHO	0.5	WHO, 2008	Oral carcinogenicity	Mouse	Derived from drinking water unit risk of 1 x 10 ⁻⁵ per 0.0007 mg/L.
IRIS	7.3	USEPA (last reviewed in 1994)	Forestomach, squamous cell papillomas and carcinomas	Mouse and rat	RSD. Neal and Rigdon, 1967; Rabstein et al., 1973; Brune et al., 1981.
CCME	2.3	CCME (2008)	Forestomach, squamous cell papillomas and carcinomas	Mouse and rat	RSD. Neal and Rigdon, 1967; Rabstein et al., 1973; Brune et al., 1981.
RIVM	0.2	RIVM, 2001	Tumor development	rat	Kroese et al., 1999 and Kalberlah et al., 1995

IRIS – Integrated Risk Information System.

RIVM – National Institute of Public Health and the Environment (Netherlands)

In order to assess potential oral health effects associated with exposure to potentially carcinogenic PAHs other than benzo(a)pyrene, toxic equivalency factors (TEFs) have been adopted. It is noted that enHealth (2012) state “At this time, no one set of PAH TEFs has been recommended for use in Australia, although it is likely that the Canadian set [CCME, 2010] is becoming more widely used, based on the fact that it is the most recent compilation of such values” (p149). The TEFs presented below were adopted from the relative potency scheme recommended by the WHO (1998) based on a detailed critical review by CCME of more than a dozen sets of TEF numbers published over the last twenty years (CCME, 2010). These TEFs represent one of the most recent reviews of relative PAH potency undertaken by an international regulatory agency. It was noted by CCME (2010) that more than a dozen sets of equivalency numbers have been proposed over the past two decades and cautions that there can only be limited confidence in the derived potency estimates (enHealth, 2012).

TEFs were adopted, where available, for carcinogenic PAHs considered to be genotoxic carcinogens (benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(g,h,i)perylene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene; see genotoxicity discussion above). The adopted TEFs are shown in **Table 3** below.

Table 3 Toxic Equivalency Factors Used to Derive Oral Dose-Response Criteria for Carcinogenic PAHs Relative to Benzo(a)pyrene (CCME, 2010)

PAH	Toxic Equivalency Factor (TEF)
Benzo(a)pyrene (index compound)	1.0
Benz(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(g,h,i)perylene	0.01
Benzo(k)fluoranthene	0.1
Chrysene	0.01
Dibenzo(a,h)anthracene	1
Indeno(1,2,3-c,d)pyrene	0.1

Inhalation

The inhalation dose-response value is expressed in units of mg/m^3 . This value may be termed a Unit Risk (UR), Cancer Risk (CR), Risk Specific Concentration (RSC), Toxic Dose (that corresponds to a 5% increase in mortality) ($\text{TD}_{0.05}$) or Tumourigenic Concentration 5% (TC05) depending on the agency of derivation.

The WHO (2000) inhalation unit risk (IUR) is based on observations in coke oven workers to mixtures of PAHs. It is noted that the composition of PAHs to which coke oven workers are exposed to may differ from that present in ambient air, or derived from soil contamination. It is noted that an inhalation unit risk is in the same order of magnitude as that derived using a linear multistage model associated with lung tumours in a rat inhalation study from coal tar/pitch condensation aerosols.

Table 4 Published Non-Threshold Dose-Response Values for PAHs - Inhalation

PAH	Agency	Inhalation Unit Risk Factor ($\mu\text{g}/\text{m}^3\text{-}1$)	Source	Target Endpoint	Test Animal	UF	Notes
Benzo[a]pyrene	WHO	8.7×10^{-2}	WHO 2000a, WHO 2010	Lung cancer	Human	-	WHO, 1999

WHO – World Health Organisation

Adopted Dose-Response Values

The adopted toxicological data were chosen in accordance with the enHealth (2012) hierarchical guidance for selection of toxicological data (Section 5.12). It is noted that this guidance states that "...it may be assumed that Australian guidance values accorded Level 1 status should take precedence over other sources, provided they are reasonably current. Other Level 1 sources may be more useful where it can be established that they are based on more contemporary risk assessment methodologies".

Threshold (Non-Carcinogenic)

Table 5 Adopted Dose-Response Values for Threshold (Non-Carcinogenic) PAHs

PAHs	Dose-Response Value (mg/kg/day)	Published By
Oral		
Acenaphthene	0.06	USEPA, 1994 (IRIS database)
Anthracene	0.3	USEPA, 1993 (IRIS database)
Fluoranthene	0.04	US EPA, 1993 (IRIS database)
Fluorene	0.04	USEPA, 1990 (IRIS database)
Phenanthrene	0.04	RIVM, 2000
Pyrene	0.03	USEPA, 1993

Inhalation

US EPA and other agencies have not published dose-response values for assessment of threshold health effects associated with inhalation exposure to PAHs. Cancer health effects of PAHs were considered to be the primary risk driver via the inhalation pathway, and will be assessed assuming carcinogenic health effects (see below).

Non-Threshold (Carcinogenic)

Table 6 Adopted Dose-Response Values for Non-Threshold (Carcinogenic) PAHs

PAHs	Dose-Response Value	Published By
Oral (mg/kg/day)⁻¹		
Benzo[a]pyrene	0.43	NHMRC, 2011
Inhalation Unit Risk (µg/m³)⁻¹		
Benzo[a]pyrene	8.7 x 10 ⁻²	WHO 2000a, WHO 2010

In order to assess potential oral and inhalation health effects associated with exposure to potentially carcinogenic PAHs other than benzo(a)pyrene, the TEFs presented in **Table 3** were adopted. TEFs were adopted only for carcinogenic PAHs considered to be genotoxic carcinogens, i.e. benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, chrysene, dibenzo(a,h)anthracene and indeno(1,2,3-cd)pyrene.

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Total Petroleum Hydrocarbons

The following information was sourced primarily from literature collated and discussed by CRCCARE Technical Report no. 10 Health screening levels for petroleum hydrocarbons in soil and groundwater (Friebel, E. and Nadebaum, P., 2011a) and by the US Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG) in volumes 1 through 5 of the series (TPHCWG, 1997;1998). Information was also sourced from the US Agency for Toxic Substances and Disease Registry (ATSDR) profile (ASTDR, 1999).

Chemical Identification

Synonyms: total petroleum hydrocarbons (TPH), total recoverable hydrocarbons (TRH)

CAS: None

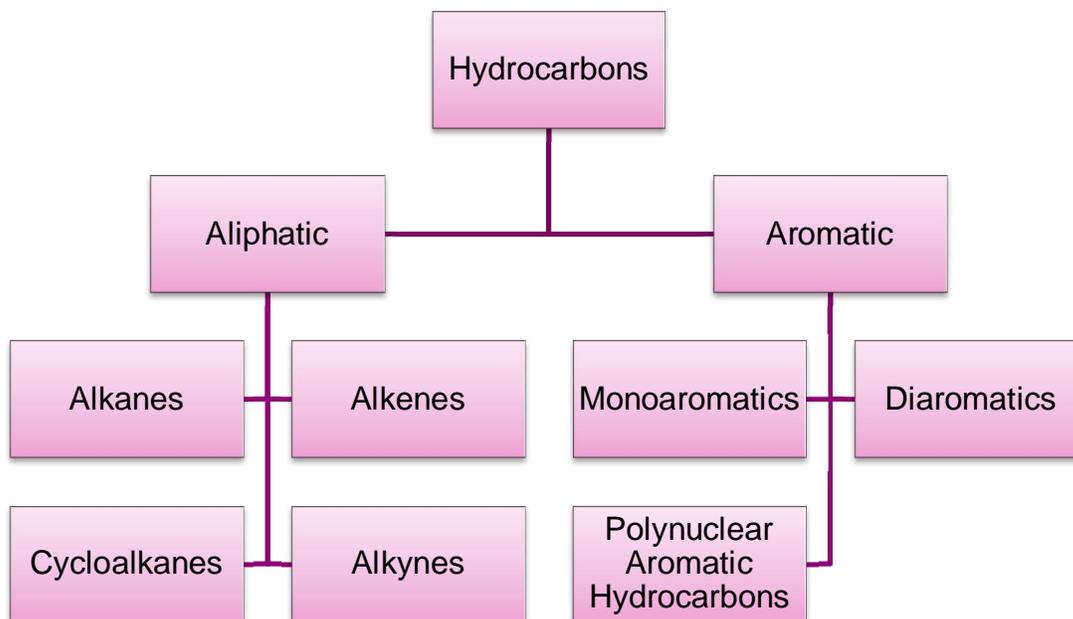
Molecular Formula: various, dependant on TPH mixture.

Molecular Weight: various, dependant on TPH mixture.

General Information

Petroleum hydrocarbons are organic compounds comprising carbon and hydrogen atoms arranged in varying structural configurations. Petroleum hydrocarbons are complex mixtures of hundreds of hydrocarbon compounds. In Volume 2 of the TPHCWG series, a list of 250 individual compounds found in TPH is presented (TPHCWG, 1998). Further information on the physical and chemical properties of each compound can be found in Appendix E of the US Agency for Toxic Substances and Disease Registry (ATSDR) profile (ASTDR, 1999). Due to the quantity of information, these tables have not been repeated in this toxicity profile.

Petroleum hydrocarbons can be generally divided into two families, aliphatics (fatty) and aromatics (fragrant). Aliphatics are further divided into three main classes, alkanes, alkenes and cycloalkanes. Alkynes, another type of aliphatic structure, are not commonly found in petroleum hydrocarbons and are not discussed further. Aromatic hydrocarbons have one or more benzene rings as structural components. Benzene is a six-membered carbon ring with the chemical formula C_6H_6 (TPHCWG, 1998).



On a molecular level, aliphatic and aromatic compounds differ by the patterns of bonding between adjacent carbon atoms. Aromatic molecules have ring structures. They are flat and symmetric with clouds of electrons above and below the plane of the molecule. Aromatic carbon-carbon bonds are termed resonance bonds, as electrons are shared between multiple carbon atoms. In these compounds, the electrons are “delocalized” (participating in several bonds), which imparts chemical stability. Aliphatic structures are characterized by highly directional bonds, in which carbon atoms share electrons only with adjacent carbons. The molecules are essentially free to rotate around these bonds, thus the aliphatic structures can assume many different conformations (TPHCWG, 1998).

The complex mixtures of petroleum hydrocarbons range from light, volatile, short-chained organic compounds to heavy, long-chained, branched compounds. During the refining process, crude oil is separated into fractions having similar boiling points. These fractions are then modified by cracking, condensation, polymerization, and alkylation processes, and are formulated into commercial products such as naphtha, gasoline, jet fuel, and fuel oils. Industry specifications for refined products, such as gasoline and diesel fuel, are based upon physical and performance-based criteria, not upon a specific chemical formulation (TPHCWG, 1998).

Due to the industry-driven nature of petroleum products, the composition of petroleum products released to the environment are complex and variable, and are a result of:

- the origin and chemistry of the parent crude oil;
- refining and blending processes; and
- the use of performance enhancing additives.

Once released to the environment, the chemistry of a petroleum product is further altered by contaminant fate and transport processes, such as leaching, volatilization, and biodegradation (TPHCWG, 1998).

TPH Fractions

Due to the complexity of petroleum products it is not feasible to quantify each compound in a given petroleum mixture. The Massachusetts Department of Environmental Protection (MA DEP) originally introduced a solution to this problem. MA DEP split TPH into a relatively small number of fractions with similar physical-chemical properties, simplifying modelling of their movement in the environment and allowing toxicity characteristics to be assigned to the fractions (MA DEP, 1994).

Independently the TPHCWG, (TPHCWG, 1997a) came up with a similar methodology and grouping, adding support to their conclusions. The MA DEP report was state-specific and has since been updated, whereas the TPHCWG work was more generically applicable and now forms an international basis for TPH evaluation. Thus the TPHCWG volumes (TPHCWG, 1997a; 1997b; 1998) form the basis for the remainder of this toxicity profile.

TPHCWG Fractions

More than 200 hydrocarbons were considered by TPHCWG in the development of fraction specific properties. A simple screening-level partitioning model, based on the ASTM Standard Guide for Risk-Based Corrective Action Applied at Petroleum Release Sites, “RBCA” (ASTM, 1995) was applied to each chemical in order to quantify, individually, the chemical's relative ability to leach from soil to groundwater and volatilise from soil to air. Based on the modelling results, the chemicals were grouped into fractions (using an order of magnitude as the cut-off point) (TPHCWG, 1997a).

Within each of the initial fractions the hydrocarbons were then grouped relative to their equivalent carbon (EC) number. The equivalent carbon number, EC, is related to the boiling point of a chemical normalised to the boiling point of the n-alkanes. This can also be determined from retention time in a boiling point gas chromatographic (GC) column. This relationship was empirically determined. Thus, for chemicals where only boiling points are known, an equivalent carbon number can be easily calculated (TPHCWG, 1997a).

For example, hexane contains six carbons and has a boiling point of 69°C. Its equivalent carbon number is six. Benzene, also containing six carbons, has a boiling point of 80°C. Based on benzene's boiling point and its retention time in a boiling point GC column, benzene's equivalent carbon number is 6.5. This approach is consistent with methods routinely used in the petroleum industry for separating complex mixtures and is a more

appropriate differentiation technique than the carbon number of the chemical. Additionally this is consistent with the way analytical laboratories report carbon numbers when chemicals are evaluated on a boiling point GC column (TPHCWG, 1997a).

Once the fractions were defined, typical fate and transport properties were assigned to each fraction based on an empirical relationship between fate and transport properties of chemicals within each fraction and boiling point. These properties could be used to estimate fraction-specific exposure potential at petroleum hydrocarbon contaminated sites (TPHCWG, 1997a).

Volume 3 of the TPHCWG series (TPHCWG, 1997a) describes the process of defining the fractions. Fraction-specific properties can then be used to estimate the partitioning of the specific fraction in soil-water-air systems. Fate and transport models (either simple or complex) can then be applied as well. This revolutionary approach is now the accepted international basis for TPH evaluation.

The representative physical parameters for the TPH fractions are presented in Table 1 below. Table 7 in Volume 3 (TPHCWG, 1997a) presents physical parameters for fractions based on simple averaging, composition-weighted averaging, and correlation to relative boiling point index. Although each method yields similar results, AECOM has chosen the 'averaging of fractions' method to be consistent with the CRC CARE methodology. Therefore, the physical parameters for the averaging methodology are presented in Table 1. Note that consistent with paragraph 4.3.5 in Volume 3 (TPHCWG, 1997a), the diffusivity in air should be set to 0.1 cm²/sec for all fractions, and the diffusivity in water should be set to 0.00001 cm²/sec for all fractions.

Table 1 Physical Parameters for Hydrocarbon Fractions (TPHCWG, 1997a)

Fraction	Molecular Weight (g/mol)	Solubility (mg/l)	Vapour Pressure (atm)	Henry's Law Constant (cm ³ /cm ³)	Log K _{oc}
Aliphatics					
EC ₅ -EC ₆	80	100	0.38	41	2.8
>EC ₆ -EC ₈	110	160	0.07	77	3.5
>EC ₈ -EC ₁₀	130	0.69	0.0069	160	4.5
>EC ₁₀ -EC ₁₂	160	0.053	0.00072	160	5.5
>EC ₁₂ -EC ₁₆	210	0.00035	0.000039	160	6.7
>EC ₁₆ -EC ₃₅	280	0.0000015	0.0000011	110	8.6
Aromatic					
EC ₅ -EC ₇	78	1800	0.13	0.22	1.9
>EC ₇ -EC ₈	92	520	0.038	0.27	2.4
>EC ₈ -EC ₁₀	120	110	0.006	0.42	3.1
>EC ₁₀ -EC ₁₂	140	30	0.00094	0.34	3.5
>EC ₁₂ -EC ₁₆	150	9.3	0.00006	0.097	3.8
>EC ₁₆ -EC ₂₁	180	0.56	0.0000023	0.0099	4.2

EC – Equivalent carbon number index.

Draft NEPM Fractions

Australian TPH fractions are assessed according to the TPHCWG fractions and then compressed into four final fractions, which is based on the Canadian approach (CCME, 2008). Australian fuel-specific weight fractions were determined by analysis of fuels provided by British Petroleum Australia and Shell Company of Australia. Petrol and diesel fuel were analysed and representative weights were determined. These fractions generally conform to the Canadian system of approximately 20% aromatic hydrocarbons and 80% aliphatic hydrocarbons.

Table 2 Australian Fraction Compression (Friebel, E. and Nadebaum, P., 2011a)

CRC CARE Fraction	TPHCWG Fractions	TPHCWG Weight Fractions
C ₆ -C ₁₀	Aliphatic C ₆ -C ₈	0.231
	Aliphatic >C ₈ -C ₁₀	0.641
	Aromatic >C ₈ -C ₁₀	0.128
	Sum of fractions for C ₆ -C ₁₀ = 1	
>C ₁₀ -C ₁₆	Aliphatic >C ₁₀ -C ₁₂	0.208
	Aliphatic >C ₁₂ -C ₁₆	0.598
	Aromatic >C ₁₀ -C ₁₂	0.04
	Aromatic >C ₁₂ -C ₁₆	0.153
	Sum of fractions for C ₁₀ -C ₁₆ = 1	
>C ₁₆ -C ₃₄	Aliphatic >C ₁₆ -C ₂₁	0.709
	Aliphatic >C ₂₁ -C ₃₄	0.023
	Aromatic >C ₁₆ -C ₂₁	0.235
	Aromatic >C ₂₁ -C ₃₄	0.033
	Sum of fractions for C ₁₆ -C ₃₄ = 1	
>C ₃₄ -C ₄₀	Aliphatic >C ₃₄ -C ₄₀	0.8
	Aromatic >C ₃₄ -C ₄₀	0.2
	Sum of fractions for C ₃₄ -C ₄₀ = 1	

Health screening levels (HSLs) for the Draft NEPM fractions are generated by first deriving HSLs for the TPHCWG fractions in order to use the TPHCWG toxicity and fate & transport parameters, then the fractions were collapsed by applying the weighting factors (Friebel, E. and Nadebaum, P., 2011a).

Weighting fractions are also applied when undertaking a 'forwards' risk assessment (i.e. assessing the potential health risk), whereby the weighting factors are applied directly to the source concentration. For example a source concentration of TPH fraction >C₃₄ - C₄₀ of 10 mg/kg would be assessed in a 'forwards' assessment as 8 mg/kg aliphatic >C₃₄-C₄₀ and 2 mg/kg aromatic >C₃₄-C₄₀ (Friebel, E. and Nadebaum, P., 2011b).

Significance of Exposure Pathways and Background

The extent of TPH absorption via inhalation, oral and/or dermal routes varies greatly due to the wide range of physical and chemical properties within a TPH mixture (ATSDR, 1999). Background levels in Australian air were estimated at less than 10% of the benchmark dose for inhalation (Friebel, E. and Nadebaum, P., 2011a).

The adopted skin absorption factor was set at 20%, which is consistent with CRC CARE methodology (Friebel, E. and Nadebaum, P., 2011a) and is based on CCME (2008) guidance.

Non-Carcinogenic Health Effects

As TPH consists of a vast number of compounds, many different health effects are possible including: central nervous system effects, blood effects, immune effects, lung effects, skin effects, eye effects, reproductive effects, liver effects and kidney effects. The toxicity criteria adopted primarily focuses on liver, kidney, body weight and blood effects.

Identification of Non-Carcinogenic Toxicity Reference Values

The dose-response values provide an estimate of exposure to the human population that is likely to be without an appreciable risk of deleterious effects during a lifetime. The oral exposure represents a daily value and the

inhalation exposure represents continuous inhalation. These values are intended for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action (USEPA, 2012). Available chronic dose-response values published by sources recognised and endorsed by the USEPA, NEPC (1999), and enHealth (2012) are summarised below.

The TPHCWG identified 250 compounds commonly found in TPH; of these only 95 had reliable toxicity data. Of the 95, only 25 had sufficient data to develop toxicity criteria. The paucity of data for many petroleum hydrocarbon compounds limits the usefulness of identifying each individual compound. There is some limited toxicity data available on some of the fuel mixtures; however encountered site contaminants are typically weathered. This can vastly change the chemical composition making the comparison results uncertain.

The TPHCWG solved this problem with the indicator/surrogate approach. This involves an initial evaluation of certain carcinogenic indicator compounds such as benzene and certain polycyclic aromatic hydrocarbons (PAHs). Following this, and if applicable, the whole product approach is applied. However only in rare cases is there toxicity data available for the product encountered. In addition the whole product method is appropriate only for fresh spills of a single known product. The third step is to use the TPHCWG surrogates to evaluate the TPH fractions, non-carcinogenic mixtures, which represent the mass of petroleum remaining after evaluation of the carcinogenic indicators.

TPH was broken down into 15 fractions with respect to fate and transport parameters. However due to limited availability of toxicity data and the similarity of toxic effects, the number of fractions was reduced to 8 with respect to toxicity. The eight fractions are summarised below:

Aliphatic C₅-C₈

Aliphatic EC₅-EC₈. n-Hexane was the only compound in this range for which toxicity data was available. However n-hexane is typically found between 0.05% and 15.7% in mixtures of TPH, and thus using n-hexane to represent the entire range was considered overly conservative. n-Heptane was considered a more appropriate surrogate for this range, assuming separate evaluation of hexane. However the basis used to derive the toxicity data was a commercial hexane mixture, which contains hexane isomers. Mixture data is assumed more appropriate as it considers some interactive effects between compounds.

A reference concentration (RfC) was derived for commercial hexane of 18.4 milligrams per cubic meter (mg/m³). A reference dose (RfD) of 5 milligrams per kilogram body weight per day (mg/kg bw/day) was derived through route-to-route extrapolation from the RfC. An uncertainty factor of 100 was applied to both RfD and RfC. The toxic endpoint of neurotoxicity was listed for both the RfD and RfC in Table 1 of Volume 4 (TPHCWG, 1997b).

TPHCWG recommended that when peer-reviewed toxicity data for cyclohexane became available, the evaluation for this fraction should be re-evaluated.

Aliphatic >C₈-C₁₆

Aliphatic >EC₈-EC₁₆. Limited data were available for this range. Data for n-nonane and 10 mixture studies on Jet fuel JP-8 (EC₉-EC₁₆) and dearomatised petroleum hydrocarbon streams were also available. These mixture studies were preferred as they take into account additive effects and between them cover the entire range of the fraction.

Consequently a RfD of 0.1 mg/kg bw/day was recommended. Uncertainty factors of between 1000 - 5000 were applied in developing the RfD. The recommended RfC of 1 mg/m³ was developed from the study on JP-8 and supported by findings from the mixture studies. No toxic effects were reported in the JP-8 study, and an uncertainty factor of 1000 was used. The toxic endpoints listed in Table 1, Volume 4 for both the RfD and RfC were hepatic and haematological changes.

Aliphatic >C₁₆-C₃₅

Aliphatic >EC₁₆-EC₃₅. An extensive study on white mineral oil (lower molecular weight) was used as the basis for this range. Consequently a RfD of 2 mg/kg bw/day was recommended. An uncertainty factor of 100 was applied, and the toxic endpoint was liver granulomas. No RfC was established, as compounds in this range are not considered volatile.

Aliphatic >C₃₅

Aliphatic >EC₃₅. An extensive study on white mineral oil (higher molecular weight) was used as the basis for this range. Consequently a RfD of 20 mg/kg bw/day was recommended. An uncertainty factor of 100 was applied, and

the toxic endpoint was liver granulomas. No RfC was established, as compounds in this range are not considered volatile.

Aromatic C₇-C₈

Seven compounds were identified in this range, six of which had USEPA RfDs. Two of these (toluene and styrene) had a RfD of 0.2 mg/kg bw/day. Ethylbenzene had a lower RfD of 0.1 mg/kg bw/day. As toluene is likely to exist in TPH mixtures at 10 times the concentration of ethylbenzene, the toluene RfD was considered appropriate. Consequently a RfD of 0.2 mg/kg bw/day was recommended. An uncertainty factors of 100 (xylenes) to 1000 (toluene, ethylbenzene, and styrene) were applied, and the toxic endpoint was hepatotoxicity and nephrotoxicity. A RfC of 0.4 mg/m³, the USEPA value for toluene, was recommended for this range.

Aromatic >C₈-C₁₆

Aromatic >EC₈-EC₁₆. RfDs were available for eight of the 77 compounds identified in this range. These ranged from 0.03 to 0.3 mg/kg bw/day. Four of the RfDs were 0.04 mg/kg bw/day, thus this was considered representative of the fraction. Consequently an RfD of 0.04 mg/kg bw/day was recommended. Uncertainty factors range from 100 (biphenyl), to 1000 (naphthalene, fluorine), to 3000 (isopropylbenzene, methylnaphthalene, anthracene). The toxic endpoint was decreased body weight. RfC data for this range were very limited. Data were available for naphthalene and isopropylbenzene, but it was felt that these were not representative of the entire fraction. Data on EC₉ aromatic mixtures were available which was considered more appropriate. Consequently a RfC of 0.2 mg/m³ was recommended. An uncertainty factor of 1000 was applied and the toxic endpoint was decreased body weight.

Aromatic >C₁₆-C₃₅

Aromatic >EC₁₆-EC₃₅. No USEPA RfDs were available for compounds in this range. Additionally a literature search failed to identify data from which an RfD could be established. As a result pyrene (C₁₆H₁₀) was selected to represent this fraction. Consequently a RfD of 0.03 mg/kg bw/day was recommended. An uncertainty factor of 3000 was applied and the toxic endpoint was nephrotoxicity. No RfC was established, as compounds in this range are not considered volatile.

Aromatic >C₃₅

It was noted that, compounds > C₃₅ are not likely to be bioavailable by the oral or dermal routes (Brainard and Beck, 1992).

Summary

Table 3 Threshold Dose-Response Values for TPH as developed by TPHCWG 1997b

Fraction	Oral RfD (mg/kg/day)	Inhalation RfC (mg/m ³)	Target Endpoints (Oral and Inhalation)
Aliphatic Fractions			
EC ₅ -C ₆	5.0	18.4	Neurotoxicity
>EC ₆ -EC ₈			
>EC ₈ -EC ₁₀	0.1	1.0	Hepatic and haematological Changes
>EC ₁₀ -EC ₁₂			
>EC ₁₂ -EC ₁₆			
>EC ₁₆ -EC ₂₁	2.0	NA	Hepatic (foreign body reaction) granuloma
>EC ₂₁ -EC ₃₅			
>EC ₃₅	20	-	Hepatic (foreign body reaction) granuloma
Aromatic Fractions			
EC ₇ -C ₈	0.2	0.4	Hepatotoxicity and Nephrotoxicity
>EC ₈ -EC ₁₀	0.04	0.2	Decreased Body Weight
>EC ₁₀ -EC ₁₂			

Fraction	Oral RfD (mg/kg/day)	Inhalation RfC (mg/m ³)	Target Endpoints (Oral and Inhalation)
>EC ₁₂ -EC ₁₆			
>EC ₁₆ -EC ₂₁	0.03	NA	Nephrotoxicity
>EC ₂₁ -EC ₃₅			
>EC ₃₅	0.03	NA	Nephrotoxicity

EC – Equivalent carbon number index.

Carcinogenicity and Genotoxicity

The indicator approach covers only threshold effects. Some petroleum hydrocarbons are known human carcinogens, ie benzene. This approach assumes that non-threshold compounds will be evaluated on a compound specific basis in addition to the fraction evaluation. This method also assumes that potentially carcinogenic PAHs will be evaluated based on the toxicity equivalence method outlined by the USEPA (USEPA, 1993). This is consistent with the approach adopted in Australia (Friebel, E. and Nadebaum, P., 2011a).

Largely due to the presence of PAHs and/or benzene, the International Agency for Research on Cancer (IARC) determined that occupational exposures in petroleum refining are probably carcinogenic to humans (Group 2A), gasoline and marine diesel fuel were determined as possibly carcinogenic to humans (Group 2B) and crude oil was determined as not classifiable as carcinogenic to humans (Group 3) (IARC, 1989).

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Appendix J

Background Exposure

Consideration of Background Exposure

Background levels of contamination comprise chemical concentrations present in the environment as a result of everyday activities or natural sources. These chemicals may be present in food, air, water and consumer products and represent the non-Site sources of contamination exposure. This is commonly referred to as background exposure. enHealth (2012a) and NEPM (1999) requires that 'background exposure' be taken into account during the assessment of potential human health risk.

Background exposure is only applied to threshold contaminants (i.e. non-carcinogens) because intakes of non-threshold contaminants (i.e. carcinogens) are considered on the basis of an increase in risk, which is irrespective of background exposure. The allocation of background exposure is undertaken on a chemical-specific basis by applying a factor (%) to the threshold toxicity reference value (TRV or Reference Dose), as illustrated in the equation below:

$$TRV \text{ (adjusted for background exposure)} = (1 - \text{Background (\%)}) \times TRV$$

In cases where background exposure is considered to be essentially negligible (contributing to less than 5% of the threshold TRV), no background exposure has been applied. Where background exposure is considered to comprise greater than 50% of the threshold TRV, the background exposure is considered to be 50% of the TRV.

The background exposure allocated for each of the CoPC assessed in the Tier 2 HHRA is summarised below.

1.1 TPH and BTEX

Quantification of background population exposures to TPH is complicated by the ubiquitous nature of petroleum hydrocarbons (including fuel mixtures) in the environment (Turczynowicz, 2003).

Background exposure to TPH is generally limited to background air concentrations and the inhalation pathway (Friebel, E. and Nadebaum, P., 2011). During development of the HSLs for petroleum hydrocarbons, CRC CARE reviewed the air background levels in Australia for BTEX compounds and below is a summary of this review and the assumptions adopted during development of the HSLs:

- For toluene, ethylbenzene and xylenes, it was concluded that the inclusion of background ambient air concentrations (between 3% and less than 1% of the threshold toxicity criteria) does not contribute significantly to the total allowable exposure used to derive the HSLs and therefore background exposure for these chemicals were not included.
- For benzene, an allocation of 20% of the RfC was attributed to background benzene in air which was considered to be protective of the majority of residential properties in Sydney and Melbourne.
- It was acknowledged that background concentration data for TPH is more uncertain in comparison to BTEX compounds, with limited data available. Consequently, 10% of the RfC for TPH was attributed to background exposure to account for any uncertainty with the limited data.

For the purpose of this HHRA, AECOM adopted the CRC CARE (2011) background exposure assumptions as described for BTEX and TPH above.

1.2 Polycyclic Aromatic Hydrocarbons (PAHs)

As summarised in **Table 1**, estimated chemical intakes from background exposure to PAHs in soil, drinking water, food and air are less than 1% of the lowest TDI adopted in this assessment. Background exposure was therefore not considered to be significant in comparison to the adopted dose-response criteria and TDIs were not corrected for background exposure.

Table 1 Estimated Background Exposure to PAHs

Concentration of Intake	Estimated Intake (mg/kg/day)	Notes
Drinking Water		
623 ng/L	1.78×10^{-5}	Water concentration is the maximum reported individual PAH concentration for a range of drinking water sources monitored in the USA, United Kingdom and Europe (ATSDR, 1995). Use of this value is considered conservative given that PAHs have not been reported in Australian drinking water supplies (NHMRC, 2011). Intake has been estimated for a 70 kg adult, assuming that 2 L of water per day is ingested.
Food		
10 µg/day	1.4×10^{-4}	Food intake is the maximum reported for individual PAHs based on a range of studies in the USA, United Kingdom and Europe (ATSDR, 1995). Intake has been calculated assuming a 70 kg body weight.
Air		
10.9 ng/m ³	3.1×10^{-5}	Air concentration is maximum annual average of any individual PAH reported in Australian cities by Environmental Australia (DEH, 1999). Intake has been calculated assuming a 70 kg adult respire 20 m ³ of air per day.
Total Intake		0.00019 mg/kg/day
Minimum TDI¹		0.02 mg/kg/day
Fraction of TDI Due to Background Exposure		0.0096 (<1%)

Notes

¹Value is minimum TDI of those adopted for PAHs assessed on the basis of threshold dose-response criteria. TDIs ranged from 0.02 mg/kg/day (naphthalene) to 0.3 mg/kg/day (anthracene).

It is noted that the PAH background exposure analysis presented in this report has not explicitly considered background exposure to PAH by smokers. However, ATSDR (1995) report that concentrations of individual PAHs in cigarette smoke range from less than 1 µg per 100 cigarettes to 62 µg per 100 cigarettes. For a 70 kg individual who smokes one pack (20 cigarettes) per day, the maximum expected intake of any individual PAH is therefore estimated to be 0.00018 mg/kg/day. This additional intake due to smoking also represents less than 1% of the lowest PAH TDI considered in this assessment (0.02 mg/kg/day for naphthalene).

Background exposure is not considered in the assessment of carcinogenic (non-threshold) risks, as non-threshold risks are estimated *incremental* lifetime cancer risks. However, it should be noted that the maximum background intake estimated for individual PAHs, if assumed to apply to benzo(a)pyrene or benzo(a)pyrene toxic equivalents, would result in an estimated incremental lifetime cancer risk of approximately 9 in 100,000, which is greater than the acceptable incremental cancer risk of 1 in 100,000 adopted for this assessment. However, this cancer risk estimate should not be interpreted as an indication that exposure to background levels of PAH may cause a significant increase in cancer rates above baseline levels as the baseline lifetime risk of cancer is reported to be approximately 50% (one in two; NRC, 2006). Thus the adopted acceptable incremental cancer risk of 1 in 100,000 and the estimated cancer risk due to background concentrations of PAHs in the environment are still very low in comparison to baseline lifetime cancer risks for the population as a whole.

1.3 References

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Turczynowicz, 2003. *Establishing Health-based Investigation Levels for benzene, toluene, ethyl benzene and xylenes, naphthalene and aromatic and aliphatic \leq EC16 TPH fractions*. Proceedings of the Fifth National Workshop on the Assessment of Site Contamination. Published by the Environment Protection and Heritage Council.

Appendix K

Uncertainty and Sensitivity Analysis

Uncertainty and Sensitivity Analysis

The risk assessment process involves a number of assumptions regarding Site conditions, human exposure and chemical toxicity. These assumptions are based on Site-specific information (where available), but it is not always possible to fully predict or describe site conditions and human activities at a site for the exposure period considered in the risk assessment. The assumptions adopted for this risk assessment were therefore generally selected to be conservative in nature, in order to evaluate an assumed reasonable maximum exposure scenario and provide a deliberate margin of safety.

A discussion of some of the key uncertainties associated with different components of the risk assessment process is provided in the following sections.

1.1 Sampling Density and Analysis

The soil and groundwater data used in the risk assessment was from all previous investigations conducted within the PPP sector between June 2012 and January 2013. Fill at the Site was noted to be considerably heterogeneous and due to the paucity of samples in the PPP sector, in relation to the NSW EPA Sampling Design Guidelines (1995), there is a possibility that concentrations of CoPC may not have been fully characterised in the PPP sector and concentrations higher than that reported may be present. Should further investigation and sample analysis be undertaken, at significantly greater concentrations and/or other COPC be identified, the conclusions of the risk assessment may require revision.

However, the limited data available to characterise the heterogeneous fill material is unlikely to influence the HHERA conclusions because:

- it is understood that the existing surface covering will remain in place following the redevelopment works, and consequently any impacts within any fill material that have not been characterised will be inaccessible for direct contact; and
- no volatile compounds were detected within groundwater, and only TPH C₁₀-C₁₅ was detected at one location in soil in an outdoor location. Therefore, it is unlikely that volatile compounds are present in any uncharacterised fill material beneath the existing building foundation slabs.

Whilst the current data set is limited, this HHERA has made a number of robust conservative assumptions as compensation, such as the exposure frequency, assumptions relating to direct surface soil exposure, and adoption of maximum reported site-wide concentrations as exposure point concentrations; further assumptions are detailed throughout the report. In addition, it was assumed that receptors have the potential to come into contact with saturated and unsaturated soil down to depths of 7.8 m bgs which is an unlikely scenario.

AECOM has only considered CoPC for which analytical data was provided and has relied upon the quality assessment of the data by Coffey Geotechnics Pty Ltd.

1.2 Toxicity Assessment

In general, the available scientific information is insufficient to provide a thorough understanding of all of the potential toxic properties of chemicals to which humans may be exposed. It is necessary, therefore, to extrapolate these properties from data obtained under other conditions of exposure and involving experimental laboratory animals. This may introduce two types of uncertainties into the risk assessment, as follows:

- a) Those related to extrapolating from one species to another; and
- b) Those related to extrapolating from the high exposure doses, usually used in experimental animal studies, to the lower doses usually estimated for human exposure situations.

The majority of the toxicological knowledge of chemicals comes from experiments with laboratory animals, although there may be interspecies differences in chemical absorption, metabolism, excretion and toxic response. There may also be uncertainties concerning the relevance of animal studies using exposure routes that differ from human exposure routes. In addition, the frequent necessity to extrapolate results of short-term or subchronic animal studies to humans exposed over a lifetime has inherent uncertainty.

In order to adjust for these uncertainties, ADIs and RfDs incorporate safety factors that may vary from 10 to 10,000. The USEPA assumes that humans are as sensitive to carcinogens as the most sensitive animal species.

The policy decision, while designed to minimise the potential for underestimating risk, introduces the potential to overestimate carcinogenic risk. Conversely, it also does not allow for the possibility that humans may be more sensitive than the most sensitive animal species.

The approach for evaluating risks to mixtures of chemicals assesses dose additively and does not account for potential synergism, antagonism or differences in target organ specificity and mechanism of action. In general, the additive approach had the effect of overestimating the risks. This is because chemicals that have no additive effects are included together as well as chemicals that may have additive effects.

The derivation of toxicity values for TPH fractions (TPHCWG, 1997b) also incorporates a number of uncertainties including:

- The composition of the TPH fractions present at the Site may vary from the surrogate chemical or chemical mixture upon which adopted toxicity criteria are based.
- The composition of the TPH fractions present at the Site may change with weathering in the environment.

1.3 Background TPH Exposures

Background exposure to TPH by the general population was considered to be 10%. This is in accordance with recommendations made by CRC CARE (2011) during development of the HSLs for petroleum hydrocarbons. CRC CARE (2011) acknowledged that there is limited data concerning TPH background exposure, and that the 10% background allocation to the RfC was a conservative approach to account for any uncertainty with limited data.

1.4 Human Exposure Parameters

Risk assessment requires the adoption of several assumptions in order to assess potential human exposure. This risk assessment included assumptions about general characteristics and patterns of human exposure relevant to the Site. The assumptions used for the identified on-Site receptors were conservative and developed to provide an estimate of reasonable maximum exposures rather than the actual exposures. This approach tends to overestimate the associated risks.

For the recreational user in the PPP area, it was assumed that direct contact with impacted surface soil is possible which is likely to overestimate the potential risks because any exposed surface soil is likely to be covered with grass, landscaping or soft-fall in the playground areas.

1.5 Vapour Transport Modelling

The modelling of vapour migration from a groundwater or soil source to indoor and outdoor air has relied on a model to estimate the concentration in air, based on concentrations in the subsurface. The use of a model requires the simplification of many complex processes in the subsurface as well as the potential for entry and dispersion within a building or outdoor air. To address this simplification, the vapour models available (and adopted in this HHRA) are considered to be conservative such that uncertainties are addressed through the overestimation of actual concentrations. The vapour model was applied assuming that the maximum reported CoPC concentrations in soil may be present beneath entire building foundations and that no degradation of chemicals occurred during migration through the vadose zone. These assumptions are considered highly and potentially unrealistically conservative, especially for assessment of petroleum hydrocarbons which are subject to aerobic degradation. Despite the conservatism associated with this modelling these assumptions were adopted to account for potentially significant data gaps.

Also the vapour model used (in conjunction with reported soil concentrations) is considered to be a first tier screening tool and is considered likely to overestimate air concentrations.

The future building proposed for the PPP area is likely to have multiple floors, lifts and permanent temperature controls; therefore, there is the potential for vapour intrusion via advective processes. The vapour modelling adopted in this HHRA therefore assumes that vapours move into the building via diffusion and advection. Advection processes are likely to draw soil vapours and ambient air (i.e. oxygen) into the building, and therefore the assumption that advection is occurring, without including biodegradation, is a conservative approach. A building on a slab foundation can have advective effects if under-pressurisation is present (which is more likely to occur in well-sealed structures), where the source is directly below and close to the building foundation and where no low permeability lens is available to direct the vapours laterally away from the foundation.

1.5.1 Sensitivity Analysis

When undertaking quantitative risk modelling it is important to understand the potential effects that exposure parameters may have on the overall calculated risks. **Table 1** provides a summary of the main assumptions adopted in the current risk assessment.

Table 1 Sensitivity of Modelling Input Parameters

Parameter	Range of Values	Value Adopted in Risk Assessment	Outcome in Risk Assessment
Depth to soil contamination	-	0.20 m	The only exceedance for volatile CoPC was reported in BH104 at a depth of 0.12-0.22 m.
Geology	Fill overlying Alluvium/Estuarine Deposits and/or Residual Soil, overlying Hawkesbury sandstone	Sand and Gravel	Sand and gravel was assumed for the fill as this is the most conservative value and representative of the heterogeneous nature of the fill. Adopting a denser geology will reduce the reported health risks as less vapour will migrate up through the geological profile and enter the building and/or ambient air.
Wind speed	-	377.78 cm/s for recreational users and 37.778 cm/s for intrusive maintenance workers	Based on the annual average 9 am and 3 pm wind speed at the Sydney (Observation Hill). Adopting a higher wind speed will reduce the reported health risks as vapour concentrations will assume to disperse more readily.
Width of source area	-	1500 cm	Based on ASTM E1739-95(2010)e1. Adopting a larger width area will increase the potential health risks as there will be a greater source area from which dust can be generated.
Ambient air mixing zone height	-	200 cm	Based on ASTM E1739-95(2010)e1.
Enclosed space air exchange rate	0.2 – 2/hour	2/hour	Based on the minimum air exchange rate for commercial buildings (Building Code of Australia). Adopting a lower exchange rate would increase the concentrations of vapour inside the building.
Qsoil	1-10L/min (coarse grained soil)	5L/min	Based on the US EPA (2004) default value. Adopting a lower value would reduce the vapour flow rate into the building.

1.6 Overall

The quantification of potential risks to human health presented in this report has considered a range of issues that are associated with uncertainties inherent in the Site-specific data, toxicological data and assumptions adopted. A number of these uncertainties and issues that warrant consideration in the interpretation of the risk estimates have been identified.

In addition to these uncertainties, a number of exposure and vapour modelling parameter values were selected to represent a variable range of physiological, behavioural, chemical and physical conditions. These variables are considered to be better represented as a distribution rather than a single point value. The outcome of the assessment can therefore be affected by the variability associated with key parameters (most sensitive values). However, it should be highlighted that the assessment presented in this report has adopted conservative or reasonable upper-bound values for these variables in most cases. The compounding effect of utilising multiple reasonable upper bound limits for quantitative parameters in the risk assessment is expected to give rise to an overestimation of actual exposure and associated health risk.

1.7 References

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Appendix L

ProUCL Calculations

Appendix L

	A	B	C	D	E	F	G	H	I	J	K	L		
1	General UCL Statistics for Full Data Sets													
9	Copper													
10														
11	General Statistics													
12	Number of Valid Observations						67	Number of Distinct Observations						48
13	Number of Missing Values						2							
14														
15	Raw Statistics						Log-transformed Statistics							
16	Minimum						1	Minimum of Log Data						0
17	Maximum						130	Maximum of Log Data						4.868
18	Mean						29.92	Mean of log Data						2.848
19	Geometric Mean						17.25	SD of log Data						1.143
20	Median						15							
21	SD						30.46							
22	Std. Error of Mean						3.722							
23	Coefficient of Variation						1.018							
24	Skewness						1.459							
25														
26	Relevant UCL Statistics													
27	Normal Distribution Test						Lognormal Distribution Test							
28	Lilliefors Test Statistic						0.216	Lilliefors Test Statistic						0.0837
29	Lilliefors Critical Value						0.108	Lilliefors Critical Value						0.108
30	Data not Normal at 5% Significance Level						Data appear Lognormal at 5% Significance Level							
31														
32	Assuming Normal Distribution						Assuming Lognormal Distribution							
33	95% Student's-t UCL						36.13	95% H-UCL						45.36
34	95% UCLs (Adjusted for Skewness)						95% Chebyshev (MVUE) UCL						57.11	
35	95% Adjusted-CLT UCL (Chen-1995)						36.75	97.5% Chebyshev (MVUE) UCL						67.7
36	95% Modified-t UCL (Johnson-1978)						36.24	99% Chebyshev (MVUE) UCL						88.52
37														
38	Gamma Distribution Test						Data Distribution							
39	k star (bias corrected)						1.006	Data appear Lognormal at 5% Significance Level						
40	Theta Star						29.73							
41	MLE of Mean						29.92							
42	MLE of Standard Deviation						29.82							
43	nu star						134.8							
44	Approximate Chi Square Value (.05)						109	Nonparametric Statistics						
45	Adjusted Level of Significance						0.0464	95% CLT UCL						36.04
46	Adjusted Chi Square Value						108.5	95% Jackknife UCL						36.13
47								95% Standard Bootstrap UCL						35.93
48	Anderson-Darling Test Statistic						0.919	95% Bootstrap-t UCL						36.99
49	Anderson-Darling 5% Critical Value						0.779	95% Hall's Bootstrap UCL						36.81
50	Kolmogorov-Smirnov Test Statistic						0.136	95% Percentile Bootstrap UCL						36.31
51	Kolmogorov-Smirnov 5% Critical Value						0.112	95% BCA Bootstrap UCL						36.53
52	Data not Gamma Distributed at 5% Significance Level						95% Chebyshev(Mean, Sd) UCL						46.14	
53								97.5% Chebyshev(Mean, Sd) UCL						53.16
54	Assuming Gamma Distribution						99% Chebyshev(Mean, Sd) UCL						66.95	
55	95% Approximate Gamma UCL (Use when n >= 40)						37							
56	95% Adjusted Gamma UCL (Use when n < 40)						37.18							
57														

Appendix L

	A	B	C	D	E	F	G	H	I	J	K	L		
1	General UCL Statistics for Full Data Sets													
70	Mercury													
71														
72	General Statistics													
73	Number of Valid Observations						67	Number of Distinct Observations						19
74	Number of Missing Values						2							
75														
76	Raw Statistics						Log-transformed Statistics							
77	Minimum						0.05	Minimum of Log Data						-2.996
78	Maximum						1.3	Maximum of Log Data						0.262
79	Mean						0.132	Mean of log Data						-2.467
80	Geometric Mean						0.0848	SD of log Data						0.782
81	Median						0.05							
82	SD						0.203							
83	Std. Error of Mean						0.0248							
84	Coefficient of Variation						1.538							
85	Skewness						4.298							
86														
87	Relevant UCL Statistics													
88	Normal Distribution Test						Lognormal Distribution Test							
89	Lilliefors Test Statistic						0.343	Lilliefors Test Statistic						0.303
90	Lilliefors Critical Value						0.108	Lilliefors Critical Value						0.108
91	Data not Normal at 5% Significance Level						Data not Lognormal at 5% Significance Level							
92														
93	Assuming Normal Distribution						Assuming Lognormal Distribution							
94	95% Student's-t UCL						0.174	95% H-UCL						0.141
95	95% UCLs (Adjusted for Skewness)						95% Chebyshev (MVUE) UCL						0.168	
96	95% Adjusted-CLT UCL (Chen-1995)						0.187	97.5% Chebyshev (MVUE) UCL						0.191
97	95% Modified-t UCL (Johnson-1978)						0.176	99% Chebyshev (MVUE) UCL						0.237
98														
99	Gamma Distribution Test						Data Distribution							
100	k star (bias corrected)						1.219	Data do not follow a Discernable Distribution (0.05)						
101	Theta Star						0.108							
102	MLE of Mean						0.132							
103	MLE of Standard Deviation						0.12							
104	nu star						163.4							
105	Approximate Chi Square Value (.05)						134.9	Nonparametric Statistics						
106	Adjusted Level of Significance						0.0464	95% CLT UCL						0.173
107	Adjusted Chi Square Value						134.3	95% Jackknife UCL						0.174
108							95% Standard Bootstrap UCL						0.173	
109	Anderson-Darling Test Statistic						8.443	95% Bootstrap-t UCL						0.208
110	Anderson-Darling 5% Critical Value						0.774	95% Hall's Bootstrap UCL						0.355
111	Kolmogorov-Smirnov Test Statistic						0.287	95% Percentile Bootstrap UCL						0.176
112	Kolmogorov-Smirnov 5% Critical Value						0.111	95% BCA Bootstrap UCL						0.19
113	Data not Gamma Distributed at 5% Significance Level						95% Chebyshev(Mean, Sd) UCL						0.241	
114							97.5% Chebyshev(Mean, Sd) UCL						0.287	
115	Assuming Gamma Distribution						99% Chebyshev(Mean, Sd) UCL						0.379	
116	95% Approximate Gamma UCL (Use when n >= 40)						0.16							
117	95% Adjusted Gamma UCL (Use when n < 40)						0.161							
118														

Appendix L

	A	B	C	D	E	F	G	H	I	J	K	L		
1	General UCL Statistics for Full Data Sets													
126	Nickel													
127														
128	General Statistics													
129	Number of Valid Observations						67	Number of Distinct Observations						48
130	Number of Missing Values						2							
131														
132	Raw Statistics						Log-transformed Statistics							
133	Minimum						0.6	Minimum of Log Data						-0.511
134	Maximum						180	Maximum of Log Data						5.193
135	Mean						24.43	Mean of log Data						2.21
136	Geometric Mean						9.116	SD of log Data						1.462
137	Median						10							
138	SD						38.17							
139	Std. Error of Mean						4.664							
140	Coefficient of Variation						1.562							
141	Skewness						2.386							
142														
143	Relevant UCL Statistics													
144	Normal Distribution Test						Lognormal Distribution Test							
145	Lilliefors Test Statistic						0.319	Lilliefors Test Statistic						0.0816
146	Lilliefors Critical Value						0.108	Lilliefors Critical Value						0.108
147	Data not Normal at 5% Significance Level						Data appear Lognormal at 5% Significance Level							
148														
149	Assuming Normal Distribution						Assuming Lognormal Distribution							
150	95% Student's-t UCL						32.21	95% H-UCL						40.37
151	95% UCLs (Adjusted for Skewness)						95% Chebyshev (MVUE) UCL						52.64	
152	95% Adjusted-CLT UCL (Chen-1995)						33.56	97.5% Chebyshev (MVUE) UCL						64.29
153	95% Modified-t UCL (Johnson-1978)						32.44	99% Chebyshev (MVUE) UCL						87.16
154														
155	Gamma Distribution Test						Data Distribution							
156	k star (bias corrected)						0.605	Data appear Lognormal at 5% Significance Level						
157	Theta Star						40.37							
158	MLE of Mean						24.43							
159	MLE of Standard Deviation						31.41							
160	nu star						81.11							
161	Approximate Chi Square Value (.05)						61.35	Nonparametric Statistics						
162	Adjusted Level of Significance						0.0464	95% CLT UCL						32.11
163	Adjusted Chi Square Value						60.98	95% Jackknife UCL						32.21
164								95% Standard Bootstrap UCL						32.21
165	Anderson-Darling Test Statistic						2.062	95% Bootstrap-t UCL						34.05
166	Anderson-Darling 5% Critical Value						0.805	95% Hall's Bootstrap UCL						33.29
167	Kolmogorov-Smirnov Test Statistic						0.182	95% Percentile Bootstrap UCL						32.51
168	Kolmogorov-Smirnov 5% Critical Value						0.114	95% BCA Bootstrap UCL						33.85
169	Data not Gamma Distributed at 5% Significance Level						95% Chebyshev(Mean, Sd) UCL						44.76	
170								97.5% Chebyshev(Mean, Sd) UCL						53.56
171	Assuming Gamma Distribution						99% Chebyshev(Mean, Sd) UCL						70.84	
172	95% Approximate Gamma UCL (Use when n >= 40)						32.3							
173	95% Adjusted Gamma UCL (Use when n < 40)						32.5							
174														

Appendix L

	A	B	C	D	E	F	G	H	I	J	K	L		
1	General UCL Statistics for Full Data Sets													
187	Zinc													
188														
189	General Statistics													
190	Number of Valid Observations						67	Number of Distinct Observations						54
191	Number of Missing Values						2							
192														
193	Raw Statistics						Log-transformed Statistics							
194	Minimum						2.2	Minimum of Log Data						0.788
195	Maximum						630	Maximum of Log Data						6.446
196	Mean						61.33	Mean of log Data						3.511
197	Geometric Mean						33.49	SD of log Data						1.112
198	Median						35							
199	SD						94.3							
200	Std. Error of Mean						11.52							
201	Coefficient of Variation						1.538							
202	Skewness						4.329							
203														
204	Relevant UCL Statistics													
205	Normal Distribution Test						Lognormal Distribution Test							
206	Lilliefors Test Statistic						0.268	Lilliefors Test Statistic						0.0991
207	Lilliefors Critical Value						0.108	Lilliefors Critical Value						0.108
208	Data not Normal at 5% Significance Level						Data appear Lognormal at 5% Significance Level							
209														
210	Assuming Normal Distribution						Assuming Lognormal Distribution							
211	95% Student's-t UCL						80.55	95% H-UCL						84.6
212	95% UCLs (Adjusted for Skewness)						95% Chebyshev (MVUE) UCL						105.7	
213	95% Adjusted-CLT UCL (Chen-1995)						86.79	97.5% Chebyshev (MVUE) UCL						125
214	95% Modified-t UCL (Johnson-1978)						81.56	99% Chebyshev (MVUE) UCL						162.7
215														
216	Gamma Distribution Test						Data Distribution							
217	k star (bias corrected)						0.926	Data appear Lognormal at 5% Significance Level						
218	Theta Star						66.23							
219	MLE of Mean						61.33							
220	MLE of Standard Deviation						63.73							
221	nu star						124.1							
222	Approximate Chi Square Value (.05)						99.35	Nonparametric Statistics						
223	Adjusted Level of Significance						0.0464	95% CLT UCL						80.28
224	Adjusted Chi Square Value						98.86	95% Jackknife UCL						80.55
225							95% Standard Bootstrap UCL						80.1	
226	Anderson-Darling Test Statistic						1.404	95% Bootstrap-t UCL						98.19
227	Anderson-Darling 5% Critical Value						0.782	95% Hall's Bootstrap UCL						165.7
228	Kolmogorov-Smirnov Test Statistic						0.134	95% Percentile Bootstrap UCL						82.72
229	Kolmogorov-Smirnov 5% Critical Value						0.112	95% BCA Bootstrap UCL						88.58
230	Data not Gamma Distributed at 5% Significance Level						95% Chebyshev(Mean, Sd) UCL						111.5	
231							97.5% Chebyshev(Mean, Sd) UCL						133.3	
232	Assuming Gamma Distribution						99% Chebyshev(Mean, Sd) UCL						176	
233	95% Approximate Gamma UCL (Use when n >= 40)						76.59							
234	95% Adjusted Gamma UCL (Use when n < 40)						76.96							