RECEIVED
JUL - 3 1991
N.Y.S. DEPT. OF ENVIRONMENTAL CONSERVATION
REGION 9

REMEDIAL INVESTIGATION

AT THE

FRONTIER CHEMICAL – PENDLETON SITE

PENDLETON (T), NIAGARA (C), NEW YORK

NYSDEC SITE NO. 9-32-043
WORK ASSIGNMENT NO. D002340-4

Prepared for:

NEW YORK STATE
DEPARTMENT OF ENVIRONMENTAL CONSERVATION
50 Wolf Road, Albany, New York

Thomas C. Jorling, Commissioner

DIVISION OF HAZARDOUS WASTE REMEDIATION

Michael J. O'Toole, Jr., P.E. – Director

URS Consultants, Inc.
282 Delaware Avenue
Buffalo, New York 14202

APPENDICES S-U

JUNE 1991

APPENDIX S

PATHWAY EXPOSURE EQUATIONS AND ASSUMPTIONS

PATHWAY # 1

INGESTION OF CHEMICALS IN SOIL BY TRESPASSERS.

$$\text{Intake (mg/kg-day)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AT}$$

Where:

CS = Chemical Concentration in Soil (mg/kg)
IR = Ingestion Rate (mg soil/day)
CF = Conversion Factor (10^{-6} kg/mg)
FI = Fraction Ingested from Contaminated Source (unitless)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Assumptions:

CS = UCL_{95} value for surface soils (mg/kg)
IR = 100 mg/day (value is that for age groups greater than 6 years old - EPA)
CF = 10^{-6} kg/mg
FI = 0.5 (assume that on days that the site is visited 1/2 of all soil ingested during the day is from the site)
EF = 50 days/year
ED = 30 years
BW = 70 kg
AT (non-carcinogen) = (30 years) (365 days/year) = 10950 days
AT (carcinogenic) = (70 years) (365 days/year) = 25550 days

$$\text{Intake (carcinogenic)} = \frac{(UCL_{95})(100)(10^{-6})(0.5)(50)(30)}{(70)(25550)} = (UCL_{95})(4.19 \times 10^{-08})$$

$$\text{Intake (non-carcinogenic)} = \frac{(UCL_{95})(100)(10^{-6})(0.5)(50)(30)}{(70)(10950)} = (UCL_{95})\ (9.78 \times 10^{-08})$$

PATHWAY # 2

DERMAL ABSORBTION OF CHEMICALS IN SOIL BY TRESPASSERS

$$\text{Absorbed Dose (mg/kg-day)} = \frac{CS \times CF \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

Where:

CS	=	Chemical Concentration in Soil (mg/kg)
CF	-	Conversion Factor (10^{-6} kg/mg)
SA	=	Skin Surface Area Available for Contact (cm^2/event)
AF	=	Soil to Skin Adherence Factor (mg/cm^2)
ABS	=	Absorption Factor (unitless)
EF	=	Exposure Frequency (events/year)
ED	=	Exposure Duration (years)
BW	=	Body Weight (kg)
AT	=	Averaging Time (period over which exposure is averaged - days)

Assumptions:

CS	=	UCL_{95} value for surface soils (mg/kg)
CF	=	Conversion Factor (10^{-6} kg/mg)
SA	=	$(0.23m^2 + 0.082m^2)\left(\frac{10{,}000 cm^2}{m^2}\right)$ = 3120 cm^2/event, assumes adults with arms and hands exposed
AF	=	1.45 mg/cm^2 (potting soil)
ABS	=	0.02 (Hawley, J.K., 1985)
EF	=	50 events/year
ED	=	30 years
BW	=	70 kg

AT (carcinogenic) = (70 years) (365 days/year) = 25550 days

AT (non-carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Absorbed Dose (Carcinogenic)} = \frac{(UCL_{95})\ (10^{-6})(3120)(1.45)(0.02)(50)(30)}{(70)(25550)} = (7.59 \times 10^{-8})\ (UCL_{95})$$

$$\text{Absorbed Dose (Non-carcinogenic)} = \frac{(UCL_{95})(10^{-6})(3120)(1.45)(0.02)(50)(30)}{(70)(10950)} = (1.77 \times 10^{-7})(UCL_{95})$$

PATHWAY # 3

DERMAL CONTACT WITH SHALLOW GROUNDWATER BY TRESPASSERS

$$\text{Absorbed Dose (mg/kg-day)} = \frac{CW \times SA \times PC \times ET \times EF \times ED \times CF}{BW \times AT}$$

Where:

CW	=	Chemical Concentration in Water (mg/kg)
SA	=	Skin Surface Area Available for Contact (cm^2/event)
PC	=	Chemical-specific Dermal Permeability Constant (cm/hr)
ET	=	Exposure Time (hours/day)
EF	=	Exposure Frequency (events/year)
ED	=	Exposure Duration (years)
CF	=	Conversion Factor
BW	=	Body Weight (kg)
AT	=	Averaging Time (period over which exposure is averaged - days)

Assumptions:

CW	=	UCL_{95} value for surface groundwater (mg/kg)
SA	=	3120 cm^2 (assumes adults with pants legs soaked with contaminated water)
PC	=	Chemical specific constant - values given in exposure assessment manual cm/hr - chemicals not listed were given the constant value of water (8×10^{-4})
ET	=	1 hour/day
EF	=	50 days/year but GW only exposed for 1/3 of year - therefore 50 is reduced to 17 days/year
ED	=	30 years
CF	=	Conversion factor = $\frac{1 \times 10^{-3} l}{cm^3}$
BW	=	70 kg

AT (carcinogenic) = (70 years) (365 days/year) = 25550 days

AT (non-carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Absorbed Dose (Carcinogenic)} = \frac{(UCL_{95})\ (PC)(3120)(1)(17)(30)(1 \times 10^{-3})}{(70)(25550)} = (8.90 \times 10^{-4})(PC)(UCL_{95})$$

$$\text{Absorbed Dose (Non-carcinogenic)} = \frac{(UCL_{95})\ (PC)(3120)(1)(17)(30)(1 \times 10^{-3})}{(70)(10950)} = (2.08 \times 10^{-3})(PC)(UL_{95})$$

PATHWAY #4

INHALATION OF AIRBORNE (VAPOR PHASE) CHEMICALS BY TRESPASSERS

$$\text{Intake (mg/kg-day)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AT}$$

Where:
CA = Contaminant Concentration in Air (mg/m^3)
IR = Inhalation Rate (m^3/hour)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged --days)

Assumptions:
CA = Exposure Point Concentration from model (mg/m^3)
IR = 20 m^3/day = 0.8333 m^3/hr
Average Adult Male
ET = 2 hours/day
EF = 50 days/year, but GW is only exposed for 1/3 of the year - therefore 50 is reduced to 17 days/year
ED = 30 years
BW = 70 kg
AT (carcinogenic) = (70 years) (365 days/year) = 25550 days
AT (non-carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Intake (carcinogenic)} = \frac{(CA)\ (0.8333)(2)(17)(30)}{(70)(25550)} = (4.75 \times 10^{-4})\ (CA)$$

$$\text{Intake (non-carcinogenic)} = \frac{(CA)\ (0.8333)(2)(17)(30)}{(70)(10950)} = (1.11 \times 10^{-3})(CA)$$

PATHWAY #5

INHALATION OF AIRBORNE (VAPOR PHASE) CHEMICALS BY RESIDENTS

$$\text{Intake (mg/kg-day)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AT}$$

Where:
CA = Contaminant Concentration in Air (mg/m^3)
IR = Inhalation Rate (m^3/hour)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged --days)

Assumptions:
CA = Exposure Point Concentration from model (mg/m^3)
IR = 20 m^3/day = 0.8333 m^3/hr
Average Adult Male
ET = 24 hours/day
EF = 365 days/year, but GW is only exposed for 1/3 of the year - therefore 365 is reduced to 122 days/year
ED = 30 years
BW = 70 kg
AT (carcinogenic) = (70 years) (365 days/year) = 25550 days
AT (non-carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Intake (carcinogenic)} = \frac{(CA)(0.8333)(24)(122)(30)}{(70)(25550)} = (0.041)\ (CA)$$

$$\text{Intake (non-carcinogenic)} = \frac{(CA)(0.8333)(24)(122)(30)}{(70)(10950)} = (0.096)\ (CA)$$

PATHWAY #6

INHALATION OF FUGITIVE DUST BY TRESPASSERS

$$\text{Intake (mg/kg-day)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AT}$$

Where:
CA = Contaminant Concentration in Air (mg/m^3)
IR = Inhalation Rate (m^3/hour)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged --days)

Assumptions:
CA = Airborne Concentration (mg/m^3) x 0.6361 (respirable fraction)
IR = 20 m^3/day = 0.8333 m^3/hr
Average Adult Male
ET = 2 hours/day
EF = 50 days/year
ED = 30 years (90th percentile at one residence, EPA, 1989)
BW = 70 kg
AT (carcinogenic) = (70 years) (365 days/year) = 25550 days
AT (non-carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Intake (carcinogenic)} = \frac{(\text{Airborne Concentration})(.6361)(.8333)(2)(50)(30)}{(70)(25550)} = (8.89 \times 10^{-4})\ (AC)$$

$$\text{Intake (non-carcinogenic)} = \frac{(\text{Airborne Concentration})(.6361)(.8333)(2)(50)(30)}{(70)(10950)} = (0.002)\ (AC)$$

PATHWAY #7

INHALATION OF FUGITIVE DUST BY RESIDENTS

$$\text{Intake (mg/kg-day)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AT}$$

Where:
CA = Contaminant Concentration in Air (mg/m^3)
IR = Inhalation Rate (m^3/hour)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged --days)

Assumptions:
CA = Airborne Concentration (mg/m^3) x 0.6361 (respirable fraction)
IR = 20 m^3/day = 0.8333 m^3/hr
Average Adult Male
ET = 24 hours/day
EF = 365 days/year
ED = 30 years (90th percentile at one residence, EPA, 1989)
BW = 70 kg
AT (carcinogenic) = (70 years) (365 days/year) = 25550 days
AT (non-carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Intake (carcinogenic)} = \frac{(\text{Airborne Concentration})(.6361)(.8333)(24)(365)(30)}{(70)(25550)} = (0.0778)\ (AC)$$

$$\text{Intake (non-carcinogenic)} = \frac{(\text{Airborne Concentration})(.6361)(.8333)(24)(365)(30)}{(70)(10950)} = (0.1817)\ (AC)$$

PATHWAY # 8

INGESTION OF CHEMICALS IN SOIL BY USERS

$$\text{Intake (mg/kg-day)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AT}$$

Where:

CS = Chemical Concentration in Soil (mg/kg)
IR = Ingestion Rate (mg soil/day)
CF = Conversion Factor (10^{-6} kg/mg)
FI = Fraction Ingested from Contaminated Source (unitless)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Assumption:

CS = UCL_{95} value for surface soils (mg/kg)
IR = 100 mg/day (value is that for age groups greater than 6 years old - EPA guidance)
CF = 10^{-6} kg/mg
FI = 0.5 (assume that on days that the site is visited 1/2 of all soil ingested during the day is from the site)
EF = 100 days/year
ED = 30 years
BW = 70 kg
AT (non-carcinogen) = (30 years) (365 days/year) = 10950 days
AT (carcinogenic) = (70 years) (365 days/year) = 25550 days

$$\text{Intake (carcinogenic)} = \frac{(UCL_{95})(100)(10^{-6})(0.5)(100)(30)}{(70)(25550)} = (UCL_{95})(8.39 \times 10^{-8})$$

$$\text{Intake (non-carcinogenic)} = \frac{(UCL_{95})(100)(10^{-6})(0.5)(100)(30)}{(70)(10950)} = (UCL_{95})\ (1.96 \times 10^{-07})$$

PATHWAY # 9

DERMAL ABSORBTION OF CHEMICALS IN SOIL BY USERS

$$\text{Absorbed Dose (mg/kg-day)} = \frac{CS \times CF \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

Where:

CS	=	Chemical Concentration in Soil (mg/kg)
CF	-	Conversion Factor (10^{-6} kg/mg)
SA	=	Skin Surface Area Available for Contact (cm^2/event)
AF	=	Soil to Skin Adherence Factor (mg/cm^2)
ABS	=	Absorption Factor (unitless)
EF	=	Exposure Frequency (events/year)
ED	=	Exposure Duration (years)
BW	=	Body Weight (kg)
AT	=	Averaging Time (period over which exposure is averaged - days)

Assumptions:

CS	=	UCL_{95} value for surface soils (mg/kg)
CF	=	10^{-6} kg/mg
SA	=	$(0.23m^2 + 0.082m^2 + 0.55\ m^2)\ \frac{10{,}000cm^2}{m^2} = 8620\ cm^2$/event, assumes adults with arms, hands and legs exposed.
AF	=	1.45 mg/cm^2 (potting soil)
ABS	=	0.02 (Hawley, J.K., 1985)
EF	=	100 events/year
ED	=	30 years
BW	=	70 kg

AT (carcinogenic) = (70 years) (365 days/year) = 25550 days
AT (non-carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Absorbed Dose (Carcinogenic)} = \frac{(UCL_{95})(10^{-6})(8620)(1.45)(0.02)(100)(30)}{(70)(25550)} = (UCL_{95})(4.19 \times 10^{-7})$$

$$\text{Absorbed Dose (Non-carcinogenic)} = \frac{(ULC_{95})(10^{-6})(8620)(1.45)(0.02)(100)(30)}{(70)(10950)} = (UCL_{95})(9.78 \times 10^{-07})$$

PATHWAY # 10

DERMAL CONTACT WITH SHALLOW GROUNDWATER BY USERS

$$\text{Absorbed Dose (mg/kg-day)} = \frac{CW \times SA \times PC \times ET \times EF \times ED \times CF}{BW \times AT}$$

Where:

CW = Chemical Concentration in Water (mg/kg)
SA = Skin Surface Area Available for Contact (cm^2/event)
PC = Chemical-specific Dermal Permeability Constant (cm/hr)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (events/year)
ED = Exposure Duration (years)
CF = Conversion Factor
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Assumptions:

CW = UCL_{95} value for shallow groundwater (mg/kg)
SA = 3120 cm^2 (assumes adults with pants legs soaked with contaminated water)
PC = Chemical specific constant - values given in exposure assessment manual
cm/hr - chemicals not listed were given the constant value of water (8×10^{-4})
ET = 1 hour/day
EF = 100 days/year but GW only exposed for 1/3 of year, therefore reduce to 33 days/year
ED = 30 years
CF = Conversion factor = $\frac{1 \times 10^{-3} l}{cm^3}$
BW = 70 kg
AT (carcinogenic) = (70 years) (365 days/year) = 25550 days
AT (non-carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Absorbed Dose (Carcinogenic)} = \frac{(UCL_{95})\ (PC)(3120)(1)(33)(30)(1 \times 10^{-3})}{(70)(25550)} = (1.73 \times 10^{-3})(PC)(UCL_{95})$$

$$\text{Absorbed Dose (Non-carcinogenic)} = \frac{(UCL_{95})\ (PC)(3120)(1)(33)(30)(1 \times 10^{-3})}{(70)(10950)} = (4.03 \times 10^{-3})(PC)(UCL_{95})$$

PATHWAY #11

INHALATION OF AIRBORNE (VAPOR PHASE) CHEMICALS BY USERS

$$\text{Intake (mg/kg-day)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AT}$$

Where:
CA = Contaminant Concentration in Air (mg/m^3)
IR = Inhalation Rate (m^3/hour)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged --days)

Assumptions:
CA = Exposure Point Concentration from model (mg/m^3)
IR = 20 m^3/day = 0.8333 m^3/hr
Average Adult Male
ET = 4 hours/day
EF = 100 days/year, but GW is only exposed for 1/3 of the year - therefore reduce 100 to 33 days/year
ED = 30 years
BW = 70 kg
AT (carcinogenic) = (70 years) (365 days/year) = 25550 days
AT (non-carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Intake (carcinogenic)} = \frac{(CA)(0.8333)(4)(33)(30)}{(70)(25550)} = (1.85 \times 10^{-3})(CA)$$

$$\text{Intake (non-carcinogenic)} = \frac{(CA)(0.8333)(4)(33)(30)}{(70)(10950)} = (4.31 \times 10^{-3})(CA)$$

PATHWAY #12

USER INHALATION OF FUGITIVE DUST

$$\text{Intake (mg/kg-day)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AT}$$

Where:
CA = Contaminant Concentration in Air (mg/m^3)
IR = Inhalation Rate (m^3/hour)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged --days)

Assumptions:
CA = Airborne Concentration (mg/m^3) x 0.6361 (respirable fraction)
IR = 20 m^3/day = 0.8333 m^3/hr
Average Adult Male
ET = 4 hours/day
EF = 100 days/year
ED = 30 years (90th percentile at one residence, EPA, 1989)
BW = 70 kg
AT (carcinogenic) = (70 years) (365 days/year) = 25550 days
AT (non-carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Intake (carcinogenic)} = \frac{(AC)(.6361)(.8333)(4)(100)(30)}{(70)(25550)} = (3.56 \times 10^{-3})\ (AC)$$

$$\text{Intake (non-carcinogenic)} = \frac{(AC)(.6361)(.8333)(4)(100)(30)}{(70)(10950)} = (8.30 \times 10^{-3})\ (AC)$$

PATHWAY # 13

INGESTION OF LAKEWATER BY USERS WHILE SWIMMING

$$\text{Intake (mg/kg-day)} = \frac{CW \times CR \times ET \times EF \times ED}{BW \times AT}$$

Where:

CW = Chemical Concentration in Water (mg/liter)
CR = Contact Rate (liters/hour)
ET = Exposure Time (hours/event)
EF = Exposure Frequency (days/years)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Assumptions:

CW = UCL_{95} value for lakewater (mg/l)
CR = 0.05 liters/hour
ET = 2 hours/day
EF = 7 days/year (national average, EPA, 1989)
ED = 30 years
BW = 70 kg
AT (carcinogenic) = (70 years) (365 days/year) = 25550 days
AT (non carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Intake (carcinogenic)} = \frac{(UCL_{95})(0.05)(2)(7)(30)}{(70)(25550)} = (1.17 \times 10^{-5})\ (UCL_{95})$$

$$\text{Intake (non-carcinogenic)} = \frac{(UCL_{95})(0.05)(2)(7)(30)}{(70)(10950)} = (2.74 \times 10^{-5})\ (UCL_{95})$$

PATHWAY # 14

DERMAL CONTACT WITH LAKEWATER BY USERS WHILE SWIMMING

$$\text{Absorbed Dose (mg/kg-day)} = \frac{CW \times SA \times PC \times ET \times EF \times ED \times CF}{BW \times AT}$$

Where:

CW	=	Chemical Concentration in Water (mg/kg)
SA	=	Skin Surface Area Available for Contact (cm^2)
PC	=	Chemical-specific Dermal Permeability Constant (cm/hr)
ET	=	Exposure Time (hours/day)
EF	=	Exposure Frequency (events/year)
ED	=	Exposure Duration (years)
CF	=	Conversion Factor
BW	=	Body Weight (kg)
AT	=	Averaging Time (period over which exposure is averaged - days)

Assumptions:

CW	=	UCL_{95} value for lakewater (mg/kg)
SA	=	19400 cm^2 (assumes adult male, whole body)
PC	=	Chemical specific constant (cm/hr) - values given in exposure assessment manual Chemicals not listed were given the constant value of water (8×10^{-4})
ET	=	2 hours/day
EF	=	7 days/year (national average, EPA, 1989)
ED	=	30 years
CF	=	$\frac{1 \times 10^{-3} \text{ liter}}{cm^3}$
BW	=	70 kg

AT (carcinogenic) = (70 years) (365 days/year) = 25550 days

AT (non-carcinogenic) = (30 years) (365 days/year) = 10950 days

$$\text{Absorbed Dose (Carcinogenic)} = \frac{(UCL_{95})(PC)(19400)(2)(7)(30)(1 \times 10^{-3})}{(70)(25550)} = (UCL_{95})(PC)(4.56 \times 10^{-3})$$

$$\text{Absorbed Dose (Non-carcinogenic)} = \frac{(UCL_{95})(PC)(19400)(2)(7)(30)(1 \times 10^{-3})}{(70)(10950)} = (UCL_{95})(PC)(1.06 \times 10^{-2})$$

APPENDIX T

VOLATILIZATION MODEL AND FUGITIVE DUST CALCULATIONS

VOLATILIZATION MODEL

- Example Calculation for Vinyl Chloride

Estimation of contaminants volatilizing from the groundwater, assuming the ground is continually saturated and that the amount of contamination present is constant.

Based on T. T. Shen, Estimation of Organic Emissions from Waste Lagoons, Journal of Air Pollution Control Association, Vol. 32, No. 1, p. 79, 1982.

$\underline{ERP_i = K_{OA}A\ (MF_i)M_i}$

Where:

ERP_i = Emission rate potential of contaminant i from saturated soil (mg/s)

K_{OA} = Gas-phase mass transfer coefficient of i (gmole/cm^2s)

A = Surface area (cm^2)

MF_i = Mole fraction of contaminant i

M_i = Molecular weight of contaminant i

1. $K_{OA} = (MW_{H2O}/M_i)^{0.335} (T/98)^{1.005} (K_{G,H2O})$

Where:

MW_{H2O} = Molecular weight of water = 18.02 g/gmol

M_i = Molecular weight of vinyl chloride = 62.50 g/gmol

T = Temperature = 68°F = 20°C = 293°K

$K_{G,H2O}$ - Gas phase mass transfer coefficient of water = 5.8×10^{-5} gmol/cm^2s (Hwang, 1982)

$K_{OA} = (18.02/62.50)^{0.335} (293/298)^{1.005} (5.8 \times 10^{-5}$ gmol/cm^2s)

$K_{OA} = (0.288^{0.335})(0.98^{1.005})(5.8 \times 10^{-5}$gmol/$cm^2$s)

$K_{OA} = 3.75 \times 10^{-5}$gmol/$cm^2$s

2. A = Area of landfill contributing to inhalation by individual (1.0×10^4 cm^2.

3. $MF_i = 18 \times 10^{-6} \frac{Ci}{Mi}$

Where:
C_i = Concentration of i in wet soil mg/kg = 2.4 ppm

(95th percentile upper control limit)

M_i = Molecular weight of contaminant i = 62.50 g/gmol

$MF_i = (18 \times 10^{-6}) \frac{(2.4)}{(62.50)} = 6.91 \times 10^{-7}$

$ERP_i = (K_{OA})\ (A)\ (MF_i)\ (Mi)$

$= (3.75 \times 10^{-5}\ gmol/cm^2s)(1.0 \times 10^4 cm^2)(6.91 \times 10^{-7})(62.50)$

$= 1.62 \times 10^{-5}$ mg/s

This is assumed to be the concentration in the air at ground level available for inhalation

EXPOSURE POINT CONCENTRATIONS FOR RESIDENTS (PATHWAY 5)

Based on Superfund Exposure Assessment Manual, EPA 540/1-88/001, April 1988

Estimation of ground level atmospheric concentrations of pollutants at selected points on a centerline of a plume directly downwind from a ground level source.

$$C_{(x)} = \frac{Q}{\pi\ T_y\ T_2\ \mu}$$

Where:
$C_{(x)}$ = Concentration of substance at distance x (0.1 km for residents)

Q = Release rate of substance = 1.62×10^{-5} mg/s (ERP i from volatilization calculation above)

π = 3.1416

T_y = Horizontal dispersion coefficient at x from Figure 3-5

(assuming D atmospheric stability) = 8m

T_2 = Vertical dispersion coefficient at x from Figure 3-6 = 4.4m

μ = mean wind speed = 5.5 m/s

$$C_{(x)} = \frac{1.62 \times 10^{-5} \text{ mg/s}}{\pi\ (8\text{m})(4.4\text{m})(5.5\text{ m/s})} = 2.67 \times 10^{-8} \text{ mg/m}^3$$

EXPOSURE POINT CONCENTRATIONS FOR ONSITE PEOPLE (PATHWAYS 4 + 11)

Actual concentrations of contaminants in the breathing zone can be calculated using a box model, assuming a box 1m wide and 2m high, with an average wind speed of 5.5 m/s

$$C = \frac{Q}{h.w.\mu}$$

Where:
C = Concentration in the breathing zone

Q = Release rate of substance = 1.62×10^{-5} mg/s (ERPi from volatilization calculation above)

h - height of box=2m

w = width of box=1m

μ = mean wind speed = 5.5 m/s

$$C = \frac{1.62 \times 10^{-5} \text{ mg/s}}{(2\text{m})\ (1\text{m})\ (5.5\text{ m/s})} = 1.48 \times 10^{-6} \text{ mg/m}^3$$

FUGITIVE DUST MODEL
(For Pathways # 6, 7 and 12)

Source: Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites: EPA 1985

The site material, based on site inspection, is largely crusted due to the high clay content of the soil, and therefore, a limited source must be considered for this fraction. However, several areas of the site, including several located in the highly contaminated process area, have a lower clay content and appear to be uncrusted.

A summary of grain size analyses is given below:

#	% Gravel	% Sand	% Silt	% Clay
SG-3	8.0	12.6	24.0	55.4
SG-4	15.8	33.9	25.6	24.7
SG-5	0.2	5.7	19.3	74.8
SG-6	0.7	3.6	20.4	75.3

Sample SG-4 may be considered an example of surface material in the "uncrusted" areas. More detailed grain size distribution for this sample is given below:

Mesh Size

4 mm - 16%

2 mm - 4%

1 mm - 2%

0.5 mm - 2%

0.25 mm - 5%

69% passes the 0.25 mm sieve, therefore, the mode of the sample is <0.25 mm.

Therefore, from Figure 3-4 in the manual - the threshold friction velocity is less than 5 cm/sec which is considerably less than the 75 cm/sec threshold velocity required for the limited source situation to apply for this fraction.

For the Frontier Site two Health Risk Situations are considered:

1) Current no-action scenario in which trespassers are exposed onsite and residents are exposed at their homes and yards along Beach Ridge and Townline Roads.

and

2) A recreational future use scenario in which users of the site are exposed onsite.

Scenario #1 - No Action Current Scenario

From the grain size distribution of surface materials onsite and by the observation of the crusted and uncrusted areas onsite - two equations apply:

1) Limited source for 75% of site
2) Unlimited source for 25% of site

Assuming a "limited reservoir" for 75% of the site -- an equation developed by Cowherd (1983) applies for this fraction and is given by

$$E_{10} = 0.83 \frac{(f)\ P(u^t)(1-v)}{(PE/50)^2}$$

where:

E_{10} = emission factor per unit area of contaminated surface (mg/m^2-hr)

f = frequency of disturbance per month

u^t = probable fastest mile of wind velocity between disturbances (m/sec)

$P(u^t)$ = erosion potential - quantity of erodible particles present on the surface prior to the onset of wind erosion (g/m^2)

V = fraction of contaminated surface covered by vegetated soil (equals 0 for bare soil)

PE = Thornwaite's Precipitation Evaporation Index used as a measure of average soil moisture content

Since vehicular traffic and disturbance is 0 for the current no-action scenario ⇒ f is 0 and E_{10} is 0 for 75% of the contaminated area.

For the remaining 25 % of the contaminated area of the site - consider the "unlimited source" situation.

The annual rate of wind erosion emissions with an "unlimited" erosion potential is estimated by a predictive emission factor equation developed by Cowherd:

$$E_{10} = 0.036\ (1-v)\ ([u]/ut)^3\ (F(x))$$

Where:

E_{10} = PM_{10} emission factor, i.e., annual average PM_{10} emission rate per unit area of contaminated surface (g/m^2-hr)

V = fraction of contaminated surface with vegetative cover (equals 0 for bare soil)

[u] = mean annual wind speed (m/sec)

x = 0.886 Ut/[u] = dimensionless ratio

F(x) = function plotted in graph in Cowherd (Figure 4-3)

ut = threshold value of wind speed at 7m (m/sec)

V = fraction vegetative cover is estimated to be approximately 50% or 0.5 for the contaminated area of the site.

[u] = mean annual wind speed is 5.5 m/sec for Buffalo, New York

$x = 0.886\ ut/[u]$

where:

[u] = 5.5 m/sec for Buffalo, New York

and $ut = (u)\ (1/0.4)\ L_N\ (Z/Zo)$

u = friction velocity

= 5 cm/sec or .05 m/sec - determined from grain size analysis and discussed above

Z = 700 cm (given) = height above surface

Zo = roughness height = 4 cm from Figure 3-6 in manual

$ut = (.05)\ \frac{(1)}{(\emptyset.4)}\ (5.16) = \emptyset.645$ m/s

$x = \frac{\emptyset.886\ (\emptyset.645)}{5.5} = \emptyset.104$ (dimensionless)

F(x) = 1.91 from Figure 4-3 in Manual

u(t) = Ø.645 (see above)

$E_{10} = 0.036\ (\emptyset.5)\left(\frac{5.5}{\emptyset.645}\right)^3\ (1.91)$

$E_{10} = 21.34\ g/m^2$ - hr

Contaminant emission rates (R_{10}) are determined from the emission factor (E_{10}) using equation 2-1 of the manual:

$R_{10} = E_{10}\ A$

where:

R_{10} = emission rate of contaminant as PM_{10}

= mass fraction of contaminant in PM_{10} emissions (UCL_{95} values will be used)

units check:

$$R_{10} = \frac{mg}{kg}\left(\frac{kg}{m^2.hr}\right)\frac{m^2}{} = \frac{mg}{hr}$$

Spreadsheet was used to calculate R_{10} for the contaminants of concern.

Inputs to spreadsheet as follows:

$R_{10} = E_{10}A = mg/hr$

= UCL_{95} values for each contaminant of concern in shallow soils in ppm

E_{10} = .Ø2134 $kg/m^2 \cdot hr$

A = 1/4 of contaminated area in m^2, contaminated area ~30,000 m^2

A = 1/4 CA = 7,500 m^2

R_{10} = 160.05 [UCL_{95} of each specific chemical]

Since the source is unlimited, no correction for decay in emission rate has been incorporated (i.e. the source will likely be continual for more than 70 years).

Check

1 m^3 of soil would decay @ a rate of .Ø2134 kg/hr or 186.91 kg/yr.

1 cu. foot of dry sand silt weights 90 lbs. or 40.9 kg
2.2 lbs/kg.

1 cu. foot = 0.02932 m^3, or

1444.21 kg or 7.7267 years of source

Based upon this reality check, we will assume Emission Rates that are an order of magnitude less than the above calculations ⇒ therefore, the emissions rate of dust (E_{10}) is reduced to .ØØ2134 kg/m^2 hr and R_{10} will now equal 16.005 [UCL_{95}].

Closest downwind resident ~ 400 ft. downwind

Climatic Region #4

Source size assume 100m x 100m - very close to 87m x 87m = 7569 m^3

$$X = Q_1 F_1$$

$$Q = \frac{R_{10}}{P_R}$$

Where:

X = respirable concentration (mass/volume) from spreadsheet

F = word erosion scaling factor from Appendix D in Manual

R_{10} = Annual Wind Erosion Rate = Emission Rate $(1/3.6 \times 10^{+6})$ g/s

Pr = for Climatic Region #4 = 0.288
SE direct = closest residences maximum f w/houses within 200m of site

for Trespassers use maximum value 3.425

for Residences use SE direction 3.265

X = values given in ug/m^3

EQUATION FOR CONVERSION OF R_{10} VALUES TO: $(X=Q_1, f_1)$

1) respirable concentrations for residents

$$x = \frac{(1/3.6 \times 10^{+6})\ (UCL_{95})}{0.288}\ (3.265) \quad \text{values in ug/m}^3$$

$$\frac{(2.778 \times 10^{-7})\ (UCL_{95})}{0.288}\ (3.265/1000) \quad \text{values in mg/m}^3$$

2) respirable concentrations for trespassers

$$x = \frac{(1/3.6 \times 10^{+6})\ (UCL_{95})}{.0288}\ (3.425)$$

$$\frac{(2.778 \times 10^{-7})\ (UCL_{95})}{.0288}\ (3.425/1000) \quad \text{values in mg/m}^3$$

FUGITIVE DUST CALCULATIONS IN THE FUTURE USE SCENARIO

Assume the respirable concentration for trespassers in the current no-action scenario is supplemented by a respirable concentration from dust generation by vehicular traffic on unpaved roads that is calculated by the following equation (USEPA, 1983)

$$E_{VT} = k(1.7)\ (s/12)\ (SP/48)\ (W/2.7)^{0.7}\ (W/4)^{0.5}\ (365\text{-}DP/365)$$

Where:

E_{VT} = emission factor for vehicular traffic, kg/kilometer traveled

k = Ø.45 = particle size multiplier for particles ≤10mm

s = silt content (of road surface material)(%)

SP = mean vehicle speed (kph)

W = mean vehicle weight (Mg)

w = mean vehicle number of wheels

Dp = number of days/year with at least 0.01 inches perciptiation

k = Ø.45

s = 22.3% (average of samples in contaminated area)

SP = 25 kph

W = 1.59 Mg

w = 4 wheels

Dp = 150 (from Figure 2-3 in exposure assessment manual)

E_{VT} = (Ø.765)(1.858)(Ø.52)(Ø.69)(1)(Ø.589) = Ø.3 kg/km traveled.

Emissions due to vehicular traffic = Ø.3 kg/kilometer traveled assume 500 feet of travel per visit

x 12 visits per day by users

= 6,000 vehicular feet per day
x 365 days/year

= 2,190,000 feet/year

= 414.77 miles/year x 1.6093 k/m = 667.5 kilometers/year x .3 kg =

200.25 kg/year = Ø.Ø228 kg/hr.

Reality Check

Assume road is 4 meters wide by 85 meters long

How long would it take to erode one cubic meter of soil from one square meter of road surface?

$$\frac{200.25 \text{ kg/year}}{340 \text{ sq. meter}} = Ø.5889 \text{ kg/sq. meter year}$$

$1m^3$ of dry soil = 1444.21 kg

1444.21 ÷ Ø.5889 = 2452 years

Reality check is OK

(E_{VF}) ( ) = R_{10}

for spreadsheet
R_{10} = Ø.Ø28[UCL_{95}]

$\frac{kg}{hr} \times \frac{mg}{kg} = R_{10}$

$\frac{mg}{hr} = R_{10}$

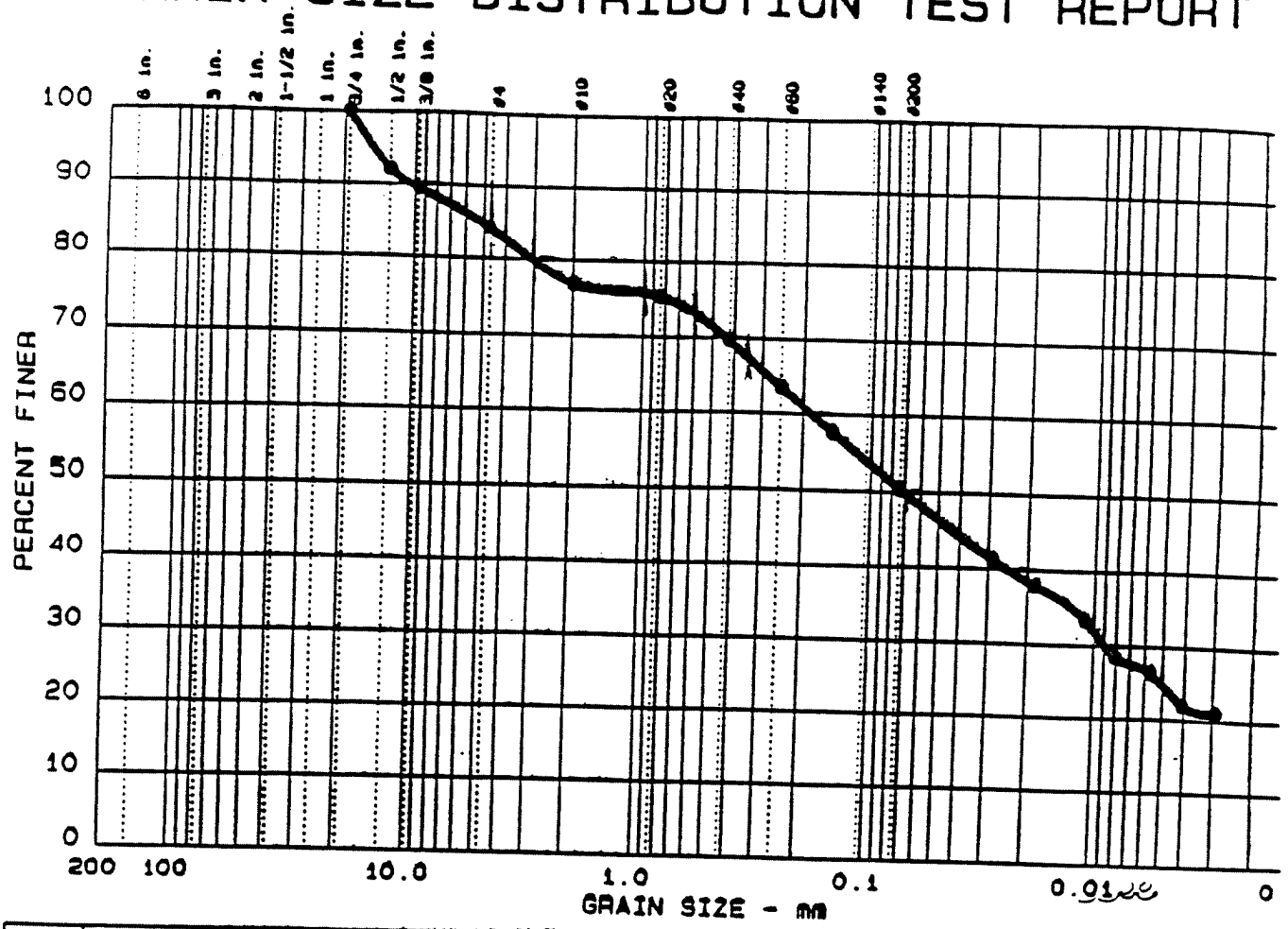
Since the value is additive with the non-vehicular windflow dust from the site - we will add the values

16.005 + Ø.Ø28

Therefore, R_{10} for the future use scenario will = 16.028 [UCL_{95}]

Airborne concentration is calculated as for the Trespasser Scenario by spreadsheet.

GRAIN SIZE DISTRIBUTION TEST REPORT



	Test	% +3"	% GRAVEL	% SAND	% SILT	% CLAY
●	20	0.0	15.8	33.9	25.6	24.7

	LL	PI	D_{85}	D_{60}	D_{50}	D_{30}	D_{15}	D_{10}	C_c	C_u
●	33.4	12.2	5.19	0.19	0.07	0.009				

MATERIAL DESCRIPTION	USCS	AASHTO
● BROWN SAND, Some Silt & Clay, little gravel	CL	

Project No.: 6008.004
Project: FRONTIER CHEMICAL
● Location: SG-4

Date: AUGUST 29, 1990

GRAIN SIZE DISTRIBUTION TEST REPORT

EMPIRE SOILS INVESTIGATIONS, INC

Remarks:

CLIENT: URS

WATER CONTENT: 13.7%

LAB NO. 426.028

Figure No. 1

APPENDIX U

TOXICOLOGICAL PROFILES

TECHNICAL PROFILE

ACENAPHTHENE

Acenaphthene, C_{12} H_{10}, is one of the polycyclic aromatic hydrocarbon (PAH) compounds. Because it is formed when gasoline, garbage, or any animal or plant material burns, it is usually found in smoke and soot. Acenaphthene is found in coal tar pitch used by industry as an adhesive. It is also used as a dye intermediate, insecticide and fungicide; and in the manufacture of some plastics. People may be exposed to acenaphthene from environmental sources such as air, water, and soil, and from cigarette smoke, gasoline exhaust condensates and overcooked food. Typical exposures are not usually to acenaphthene alone, but to a mixture of similar chemicals.

Classification

This substance/agent has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

Health Effects

Acenaphthene is a skin and mucous membrane irritant. If swallowed in large quantities it may cause vomiting.

TECHNICAL PROFILE

ACENAPHTHYLENE

Acenaphthylene, $C_{12}H_8$, is one of the polycyclic aromatic hydrocarbon (PAH) compounds. Because it is formed when gasoline, garbage, or any animal or plant material burns, it is usually found in smoke and soot. The chemical combines with dust particles in the air and is carried into water and soil and onto crops. Acenaphthylene is found in coal tar pitch used by industry as an adhesive.

People may be exposed to acenaphthylene from environmental sources such as air, water, and soil, and from cigarette smoke, and overcooked food. Typical exposures are not usually to acenaphthylene alone, but to mixture of similar chemicals.

Classification

Classification -- D; not classified as to human carcinogenicity -- Based on no human data and inadequate data from animal bioassays.

Health Effects

Intraperitoneal and intratracheal administration of naphthalene, acenaphthene, and acenaphthylene to rats produced monotypic effects in the form of vascular disorders, and degeneration in the internal organs and central nervous system. Inflammatory changes were also observed in the lungs; the degree was the same for all three substances. Splenic degeneration was noted among the unscheduled deaths in this study. It was concluded that chronic inhalation of acenaphthene and acenaphthylene had more pronounced toxic effects than naphthalene.

TECHNICAL PROFILE

ACETONE

Acetone, C_3H_6O, is a colorless liquid with a sweetish odor. It has a boiling point of 56.48°C and a density of 0.7972. It is used as a solvent for waxes, oils, resins, rubber, plastic, lacquers, varnishes, and rubber cement. It is used in the production of lubricating oils, pharmaceutical and pesticides.

Classification

This chemical is classed as a group D compound, not classifiable as to human carcinogenicity due to lack of data for humans and animals.

Health Effects

Acetone may irritate the eyes, skin, nose and throat. It's points of attack are the respiratory system and the skin. Inhalation may produce headache, fatigue, excitement, bronchial irritation, and in large amounts narcosis. Prolonged or repeated topical use may cause erythema and dryness.

TECHNICAL PROFILE

ALDRIN

Aldrin, $C_{12}H_8Cl_6$, is a colorless, crystalline solid with a melting point of 104°C. The technical grade is a tan to brown solid or powder. Aldrin was widely used as a pesticide from the 1950's to the early 1970's to control root worms, beetles, and termites. Most uses of aldrin were banned in 1975. It is no longer produced in or imported into the United States.

Classification

Aldrin has a weight of evidence classification of B2. It is a probable human carcinogen.

Health Effects

Aldrin is toxic by ingestion and inhalation, it is also absorbed by the skin. The acute effects of aldrin exposure include: headache, dizziness, irritability, appetite loss, nausea, muscle twitching, convulsions and loss of consciousness. An extremely high exposure may cause coma and death.

Chronic exposure has caused liver cancer in mice and thyroid cancer in rats. While inadequate evidence exists to evaluate whether aldrin is carcinogenic to humans the EPA considers it a probable carcinogen based on sufficient animal evidence.

Dogs and rats have developed liver and kidney degeneration. Oral administration to pregnant mice and rats has increased the number of fetal deaths and congenital abnormalities, and caused growth retardation in the offspring. In humans aldrin is a neurotoxin and has been shown to cause electroencephalogram abnormalities following short or long term exposures (NIOSH, 1988 and ATSDR Toxicological Profile for Aldrin, May 1989).

TECHNICAL PROFILE

ALUMINUM

Aluminum is a common element of the natural environment comprising up to 10 percent of the content of soil and stone. Aluminum is generally not toxic to animals. Solubility increases at low pH and in acidic waters, such as bogs, aluminum concentrations can reach levels toxic to fish.

Aluminum has many uses including: corrosion-resistant chemical equipment, uses in the electrical industry, photoengraving plates, in points and protective coatings, as a catalyst, as rocket fuel, and as an ingredient of incendiary mixtures.

Classification

The U.S. EPA classifies aluminum as a class E compound, a non-carcinogen.

Health Effects

Aluminum salts may cause dermatitis, eczema, conjunctivitis, and mucous membrane irritation. Inhalation of fine aluminum powder has been reported as a cause of pulmonary fibrosis. Aluminum may also be implicated in Alzheimer's disease (Sax).

TECHNICAL PROFILE

ANTHRACENE

Anthracene, $C_6H_4(CH)_2C_6H_4$, is one of the polycyclic aromatic hydrocarbon (PAH) compounds. It is a yellow crystal with blue fluorescence, which is insoluble in water, soluble in alcohol and ether. Because it is formed when gasoline, garbage, or any animal or plant material burns, it is usually found in smoke and soot. This chemical combines with dust particles in the air and is carried into water and soil and onto crops. Anthracene is found in coal tar pitch used by industry as an adhesive. It is used in dyes, calico printing, as a component of smoke screens and as scintillation counter crystals.

People may be exposed to anthracene from environmental sources such as air, water, and soil, and from cigarette smoke and overcooked food. Typical exposures are not usually to anthracene alone, but to a mixture of similar chemicals.

Classification

USEPA weight-of-evidence classification--D, not classifiable as to human carcinogenicity on the basis that no human data and inadequate data from animal bioassays exists.

Health Effects

Anthracene is a skin irritant and an allergen. It is an experimental equivocal tumorigenic agent and has experimental neoplastic effects.

TECHNICAL PROFILE

AROCLOR-1260, AROCLOR-1254, (PCBs)

Polychlorinated biphenyls (PCBs), $C_{12}H_{10-x}Cl_x$, are complex mixtures containing isomers of chlorobiphenyls with different chlorine content. There are 209 possible compounds obtainable by substituting chlorine for hydrogen on different positions of the biphenyl ring system. It should also be noted that PCB commercial mixtures have been shown to contain other classes of chlorinated derivatives. PCBs are used in electrical capacitors, electrical transformers, vacuum pumps, and gas-transmission pumps (Merck, 1983). They are also used in hydraulic fluids, plasticizers, adhesives, fire retardants, wax extenders, inks, lubricants, and oils.

Classification

PCB and its compounds have been classified in Group B2 by the U.S. EPA: a probable human carcinogen based an hepatocellular carcinomas in animal studies and inadequate yet suggestive evidence of excess risk of liver cancer in humans by ingestion and inhalation or dermal contact.

Health Effects

PCBs can be inhaled, ingested, and absorbed through the skin. Toxic effects in humans include chloracne, pigmentation of skin and nails, excessive eye discharge, swelling of eyelids, distinctive hair follicles, and gastrointestinal disturbances (Merck, 1983). Generally, toxic effects are dependent on the degree of chlorination, the higher degree of chlorination, the stronger the effects.

Acute and chronic exposure can cause liver damage. Signs and symptoms include edema, jaundice, vomiting, anorexia, nausea, fatigue, and abdominal pains. Studies of accidental oral intake indicate that chlorinated biphenyls are embryotoxic, causing stillbirths and increased eye discharges in infants born to women exposed during pregnancy (Sittig, 1985).

TECHNICAL PROFILE

ARSENIC

Arsenic, As, is present as an impurity in many metal ores and is produced as a by-product in the smelting of these ores, particularly copper. It is labelled as a poison and is used in a variety of industries: agricultural, insecticides, herbicides, pharmaceuticals, pigment production, and manufacturing of glass.

Classification

This substance and certain arsenic compounds have been listed as carcinogens. The weight of evidence classification as to human carcinogenicity is listed in Group A based on observation of increased lung cancer mortality in populations exposed primarily through inhalation and an increased skin cancer incidence in several populations consuming drinking water with high arsenic concentrations.

Health Effects

Arsenic can be inhaled or ingested through dust and fumes. Acute toxic effects are generally seen following ingestion of the compound. Symptoms may develop within one-half to four hours following ingestion and are characterized by constriction of the throat followed by dysphagia, epigastric pain, vomiting, and watery diarrhea. If large amounts are ingested, shock may develop due to severe fluid loss, and death may ensue within 24 hours. Exfoliative dermatitis and peripheral neuritis may develop. Acute cases due to inhalation are rare. Chronic arsenic poisoning due to ingestion is also rare. It can, however, be inhaled resulting in symptoms of weight loss, nausea, eruption of the skin, loss of hair, and peripheral neuritis. Horizontal white lines (striations) on the fingernails and toenails are commonly seen in chronic arsenic poisoning. Liver damage from chronic poisoning is still debated. Arsenic does have a depressant effect upon bone marrow, with evidence of also causing lung and skin cancer.

TECHNICAL PROFILE

BARIUM

Barium, Ba, an alkaline earth metal, is a silver-white, slightly lustrous, somewhat malleable metal. It is produced by the reduction of barium oxide. The primary sources are the minerals barite ($BaSo_4$) and witherite ($BaSo_3$). Barium may ignite spontaneously in air in the presence of moisture involving hydrogen. Most barium compounds are soluble in water, although the chemical itself is not. Metallic barium is used as a carrier for radium and for the removal of residual gas in vacuum tubes and in alloys with nickel, lead, calcium, magnesium, sodium, and lithium. Barium compounds are used in several manufacturing operations: X-ray diagnostic work, glassmaking, papermaking, and animal and vegetable oil refining. They are used in brick and tile, pyrotechnics, and the electronics industries. They are found in lubricants, pesticides, glazes, textiles dyes and finishes, pharmaceuticals, rodenticides, a stabilizer and mold lubricant in the rubber plastics industries, an extender in paints, a loader for paper, soap, rubber and linoleum, and a fire extinguisher for uranium or plutonium fires.

Classification

This chemical has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

Health Effects

The soluble barium salts, such as chloride and sulfide, are poisonous when ingested (Sax/Lewis, 1987). The insoluble sulfate used in radiography is not acutely toxic. Few cases of systemic poisoning have been reported. Barium compounds, when ingested or given orally, exert a profound effect on all muscles and especially smooth muscles, markedly increasing their contractility. The heart rate is slowed and may stop in systole. Other effects are increased intestinal peristalsis, vascular constriction, bladder contraction, and increased voluntary muscle tension. The inhalation of barium sulfate dust may lead to deposition in the lungs in sufficient quantities to produce "baritosis", a benign pneumoconiosis. Barium and its compounds may affect the heart, lungs, central nervous system, skin, eyes, and respiratory system (Sittig, 1987).

TECHNICAL PROFILE

BENZENE

The hydrocarbon benzene, C_6H_6, is a clear, volatile colorless liquid with a characteristic odor. Uses of benzene include: a constituent in motor fuels, as a solvent, imprinting, as a chemical intermediate, and in the manufacture of detergents, explosives, pharmaceuticals and dye stuffs.

Classification

Benzene is recognized as a human carcinogen (IARC, 1982; IRIS, 1991). A weight of evidence of A, positive human carcinogen has been established based on studies of increased incidence of nonlymphocytic leukemia from occupational exposure and increased incidence of neoplasia in rats and mice exposed by inhalation and gavage (IRIS, 1991).

Health Effects

Poisoning occurs most commonly via inhalation of the vapor, though benzene can be ingested and penetrate the skin and poison in that manner. Exposure to benzene can cause irritation to the skin, eyes, and upper respiratory tract; erythema, vesiculation, and dry, scaly dermatitis can result from defatting of the skin; pulmonary edema and hemorrhage can result if the liquid gets taken into the lung. Acute benzene exposure will cause central nervous system depression, headache, dizziness, nausea, convulsions and possibly even coma and death.

Benzene is a recognized leukemogen. In several studies occupational exposure has been shown to be the cause of increased incidences of nonlymphocytic leukemia. Benzene is a myelotoxic agent therefore, chronic benzene exposure may result in hypo or hyperplasia of the bone marrow, which will in turn cause changes in the peripheral blood. Anemia, leucopenia, macrocytosis, reticulocytosis, thrombocytopenia, and prolonged bleeding time may result. Other effects of chronic benzene exposure are: fatigue, headache, dizziness, nausea, loss of appetite, weight loss, weakness, pallor, nosebleeds, bleeding gums, menorrhagia, petechiae and purpura. Chronic benzene poisoning exhibits great variation in symptoms between individuals.

TECHNICAL PROFILE

BENZO(A)ANTHRACENE

Benzo[a]anthracene is one of the polycyclic aromatic hydrocarbon (PAH) compounds. Because it is formed when gasoline, garbage, or any animal or plant material burns, it is usually found in smoke and soot. This chemical combines with dust particles in the air and is carried into water and onto soil and crops. Benzo[a]anthracene is found in coal tar pitch used by industry as an adhesive.

People may be exposed to benzo[a]anthracene from environmental sources such as air, water, and soil, and from cigarette smoke and overcooked food. Typical exposures are not usually to benzo[a]anthracene alone, but to a mixture of similar chemicals.

Classification

The weight-of-evidence classification for benzo[a]anthracene is B2-- probable human carcinogen based on no human data and sufficient data from animal bioassays.

Health Effects

Benzo[a]anthracene produced tumors in mice exposed by gavage, intraperitoneal, subcutaneous or intramuscular injection, and topical application. Benzo[a]anthracene produced mutations in bacteria and in mammalian cells, and transformed mammalian cells in culture.

Although there are no human data that specifically link exposure to benzo[a]anthracene to human cancers, benzo[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke.

TECHNICAL PROFILE

BENZO(B)FLUORANTHENE

Benzo(b)fluoranthene (B(b)F), in its pure form, is a colorless crystalline solid at room temperature and has a molecular weight of 252.32 g/mole. It has a vapor pressure of 5×10^{-7} and an octanol water coefficient of $1/15 \times 10^{6}$, and is therefore expected to have poor mobility in the environment. B(b)F is a polycyclic aromatic hydrocarbon that is formed during combustion of fossil fuels and organic material. It is found environmentally in mixtures with other PAH compounds including B(a)P.

Classification

The USEPA weight of evidence classification for B(b)F is B2- probable human carcinogen. Sufficient evidence of carcinogenicity in animals exists, in the absence of positive human data.

Health Effects

There are no data available to assess significant exposure levels of B(b)F alone for humans. Reports of adverse health effects such as carcinogenicity by the inhalation and dermal routes of exposure do exist for mixtures that include B(b)F thus providing some information to qualitatively assess the role of B(b)F as a human carcinogen.

No information has been found about specific levels of B(b)F that have caused harmful effects in humans after ingestion, inhalation, or dermal contact. The carcinogenicity of B(b)F has not been adequately studied, there are no reports directly correlating human B(b)F exposure and tumor development, although humans are likely to be exposed by all routes. There are a number of reports associating human cancer with exposure to mixtures of PAHs that include B(b)F. B(b)F is a skin carcinogen in animals following dermal application, and a lung carcinogen following intratracheal instillation. It is likely that B(b)F could cause cancer in humans as well.

TECHNICAL PROFILE

BENZO(K)FLUORANTHENE

Benzo[k]fluoranthene is one of the polycyclic aromatic hydrocarbon (PAH) compounds. Because it is formed when gasoline, garbage, or any animal or plant material burns, it is usually found in smoke and soot. This chemical combines with dust particles in the air and is carried into water and onto soil and crops. Benzo[k]fluoranthene is found in coal tar pitch used by industry as an adhesive.

People may be exposed to benzo[k]fluoranthene from environmental sources such as air, water, and soil, and from cigarette smoke and overcooked food. Typical exposures are not usually to benzo[k]fluoranthene alone, but to a mixture of similar chemicals.

Classification

The USEPA weight-of-evidence classification for benzo[k]fluoranthene is B2, a probable human carcinogen on the basis that no human data and sufficient data from animal bioassays exists.

Health Effects

Benzo[k]fluoranthene produced tumors after lung implantation in mice and when administered with a promoting agent in skin-painting studies. Equivocal results have been found in a lung adenoma assay in mice. Benzo[k]fluoranthene is mutagenic in bacteria. Although there are no human data that specifically link exposure to benzo[k]fluoranthene to human cancers, benzo[k]fluoranthene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke.

TECHNICAL PROFILE

BENZO(G,H,I)PERYLENE

Benzo[g,h,i]perylene is one of the polycyclic aromatic hydrocarbon (PAH) compounds. Because it is formed when gasoline, garbage, or any animal or plant material burns, it is usually found in smoke and soot. This chemical combines with dust particles in the air and is carried into water and onto soil and crops. Benzo[g,h,i]perylene is found in coal tar pitch used by industry as an adhesive.

People may be exposed to benzo[g,h,i]perylene from environmental sources such as air, water, and soil, and from cigarette smoke and overcooked food. Typical exposures are not usually to benzo[g,h,i]perylene alone, but to a mixture of similar chemicals.

Classification

USEPA weight-of-evidence classification-- D; not classifiable as to human carcinogenicity based upon no human data and inadequate animal data from lung implant, skin-painting and subcutaneous injection bioassays.

Health Effects

Benzo[g,h,i]perylene appeared to increase lung epidermoid tumors when administered with trioctanonin in a lung implant study. In a lifetime implant study, 3-month-old female Osborne-Mendel rats received a lung implant of benzo[g,h,i]perylene. Epidermoid carcinomas in the lung and thorax were observed. The apparent increased incidence of tumors was not statistically significant and no distant tumors were seen.

TECHNICAL PROFILE

BENZO(A)PRYRENE

Benzo(a)pyrene (B(a)P), $C_{20}H_{12}$, is a polycyclic aromatic hydrocarbon (PAH) compound. It is formed when any organic material burns and is usually found in smoke and soot as a combustion by-product. B(a)P is found in coal tar pitch used by industry, and is found in creosote.

Classification

B(a)P weight of evidence is B2 because of sufficient evidence of carcinogenicity in experimental animals, but inadequate evidence of cancer in humans from epidemiologic studies.

Health Effects

Short term and intermediate oral exposure to very high levels of B(a)P resulted in death in experimental animals fed B(a)P in the diet. The induction of cancer is the key endpoint of toxicity following chronic exposures to lower doses of B(a)P in the diet. Lethal effects from high doses of B(a)P were caused by bone marrow depression. There is no information available for the potential of human carcinogenicity following oral B(a)P exposure. Studies with experimental animals have produced leukemia and tumors of the forestomach and lung following intermediate exposures in mice.

No short term or intermediate inhalation exposure effects are available for B(a)P. The induction of cancer is the key long term effect. B(a)P is a moderately potent experimental carcinogen in many species by many routes of exposure. There are no reports directly correlating human B(a)P exposure and tumor development, although humans are likely to be exposed by all routes. There are a number of reports associating human cancer and exposure to mixtures of PAHs that include B(a)P. In view of these observations and it's well established carcinogenic activity in laboratory animals, it is reasonable to conclude that B(a)P would be expected to be carcinogenic to humans by all routes of exposure.

TECHNICAL PROFILE

BENZOIC ACID

Benzoic Acid, $C_7H_6O_2$, is a benzene carboxylic acid. It is a crystalline solid, with a melting point of 122.4°C. Benzoic acid is used in preserving foods, fats, juices, dyes, and as a standard in chemical analysis.

Classification

Benzoic acid is not classified as a human carcinogen. The weight of evidence for classification as to human carcinogenicity is classified as Group D: not classifiable as to human carcinogenicity based on no human data and inadequate data from animal bioassays.

Health Effects

Benzoic acid is a mild irritant to the skin, eyes, and mucous membranes. It is a poison by vapor inhalation. Benzoic acid and sodium benzoate have been tested for mutagenicity or genotoxicity with no positive results reported (Litton Bionetics, 1975). Sodium benzoate appeared to have no maternal toxicity, fetal toxicity, or teratogenic potency in mice, rats, hamsters, or rabbits when administered (FDRL, 1972). In the stomach, both benzoic acid and sodium benzoate exist in the ionized form of a benzoate. Both benzoic acid and sodium benzoate are absorbed rapidly and completely by the gastrointestinal tract. Therefore, exposure to sodium benzoate is essentially equivalent to exposure to benzoic acid.

TECHNICAL PROFILE

BENZYL ALCOHOL

Benzyl alcohol, $C_6H_5CH_2OH$, is a constituent of jasmine, hyacinth, and ylang-ylang oils, Peru and tolu balsams. In pure form, it is a liquid with a slight aromatic odor, and sharp, burning taste. It has a boiling point of 206°C and is somewhat soluble in water. Benzyl alcohol is used in perfumes and flavors; as a photographic developer for color movie films; for dyeing nylon filament, textiles and sheet plastics; as a solvent; as an intermediate for benzyl esters and ethers; and in cosmetics, ointments, ball point pen inks and stencil inks.

Classification

Benzyl alcohol is not classified for carcinogenicity by U.S. EPA.

Health Effects

Benzyl alcohol is an eye and skin irritant.

TECHNICAL PROFILE

2-BUTANONE

2-Butanone, C_4H_8O, otherwise known as methyl ethyl ketone (MEK), is a colorless liquid with a fragrent, mintlike moderately sharp odor. It is used as a solvent in coating industries, in the manufacturing of synthetic resins, in cements and adhesives, and in the dewaxing of lubricating oils.

Classification

The weight of evidence classification by the U.S. EPA categorizes MEK in Group D, which does not list MEK as a human carcinogen. No data presently exist to evaluate this classification, evidence for carcinogenicity in humans and animals is inadequate.

Health Effects

MEK may affect the central nervous system and the lungs. It may be inhaled, absorbed through the skin, ingested, or contact the eyes and skin. Headaches, dizziness, or vomiting may develop. It is moderately toxic by ingestion. MEK irritates the eyes at concentration in the range of 350 ppm. No other adverse effects have been observed.

TECHNICAL PROFILE

BUTYLBENZYLPHTHALATE

Butylbenzylphthalate, $C_4H_9OOCC_6H_4OOC_7H_7$, is a clear, oily liquid with a slight odor. It is also known as benzylbutylphthalate on BBP. It has a melting point of less than -35°C, boiling point of 370°C and density of 1.116. Butylbenzylphthalate is used as a plasticizer for polyvinyl and cellulosic resins and as an organic intermediate.

Classification

Butylbenzylphthalate has a weight of evidence of C. It is considered a possible human carcinogen based on a significant increase in mononuclear cell leukemia in female rats. There is no data on human carcinogenity.

Health Effects

Oral administration of butylbenzylphthalate to rats resulted in decreased body weight gain, small testes, testicular lesions, and decreased hemoglobin, hematocrit and red blood cell count. Liver and kidney effects were also reported. No information on human health effects was found.

TECHNICAL PROFILE

CADMIUM

Cadmium, Cd, is a metallic element. It is naturally occurring in zinc, copper and lead ores. Since cadmium is very corrosion resistant it is used as protective coating for iron, steel and copper. Cadmium is used in alkaline batteries, as a stabilizer for polyvinyl chloride plastics, in nickel plating, and in the manufacture of semiconductors, photocells, and jewelery. Cadmium compounds are used as pesticides, polymerization catalysts, pigments, paints and in the photographic industry.

Classification

The USEPA classifies cadmium as having a weight of evidence of B1, being a probable carcinogen. Cadmium is also a teratogen and an experimental carcinogen.

Health Effects

The substance may attack the respiratory system, lungs, kidney, prostate, and blood. Cadmium compounds are well absorbed by inhalation but poorly absorbed from the intestinal tract. Skin absorption appears negligible. After being absorbed cadmium has a very long half life. It is stored in the kidneys and liver.

Acute health effects are usually delayed a few hours after exposure. There is irritiation of the upper respiratory tract; possibly followed by coughing, chest pain, sweating and chills. 8 to 24 hours after exposure severe pulmonary irritiation may develop with shortness of breath and general weakness. Breath may become shorter as pulmonary edema develops. There is an approximately 15% mortality rate in acute cases. Survivors may have emphysema and corpulmonale.

The chronic effects of cadmium poisoning are kidney damage and mild hypochronic anemia. In experimental animals chronic effects have included liver damage, central nervous sytem damage, testicular atrophy, teratogenic effects (rodents), decrease in red blood cell count, sarcomata, and testicular neoplasms.

TECHNICAL PROFILE

CARBON DISULFIDE

Carbon disulfide, CS_2, is a mobile, clear, or faintly yellow liquid. Pure distillates have a sweet pleasing ethereal odor. Reagent and commercial grades are foul-smelling. It is used in the manufacture of soil disinfectants, vacuum tubes, and for cleaning and extractions, especially in metal treatment and plating. It is a fumigant for commodities, a corrosion inhibitor, and a polymerization inhibitor for vinyl chlorides.

Classification

This chemical has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential. No weight of evidence is classified.

Health Effects

Poisoning usually occurs from inhalation but also may be caused by ingestion and skin absorption. Acute toxicity exhibits euphoria, restlessness, mucous membrane irritations, nausea, vomiting, unconsciousness, and terminal convulsions. Chronic toxicity exhibits marked psychic disturbances ranging from extreme irritability to mania with hallucinations, tremors, auditory and visual disturbances, weight loss, and blood dyscrasis. Dermal contact with concentrated solutions may cause burning, pain, erythema, and exfoliation.

TECHNICAL PROFILE

4-CHLOROANILINE

4-Chloroaniline, $ClC_6H_4NH_2$, an aromatic amino compound, is also known as p-aminochlorobenzene, and 4-chlorophenylamine. It is a white or pale yellow solid with a characteristic sweet odor and a melting point of 69.5°C. It is soluble in hot water and organic solvents. 4-Chloroaniline is used as a dye intermediate, in pharmaceuticals and in agricultural chemicals.

Classification

4-Chloroaniline, has not been evaluated by the US EPA for evidence of human carcinogenic potential.

Health Effects

4-Chloroaniline is absorbed through the intact skin. It may cause methemoglobinemia (increased methemoglobin in the blood caused by a decrease in the amount of NADH which reduces methemoglobin to hemoglobin or by an increase in the oxidation of hemoglobin). The major manifestation of methemoglobinemia is cyanosis. Anoxia may result in severe cases of methemoglobinemia. Oral administration (by gavage) of 4-chloroaniline to rats has caused proliferative lesions of the spleen and renal tubular degeneration.

TECHNICAL PROFILE

CHLOROBENZENE

Chlorobenzene, C_6H_5Cl, otherwise known as benzene chloride, is a clear, colorless liquid used in the manufacture of aniline, phenols, and as an intermediate of dyes and pesticides.

Classification

The weight of evidence for carcinogenicity by the U.S. EPA is presently being evaluated. This does not imply that chlorobenzene is necessarily a carcinogen.

Health Effects

Chlorobenzene can be inhaled, ingested, or irritate the eyes and skin. It may affect the respiratory and central nervous systems, and the liver. It may cause drowsiness, incoherence, skin irritation, and liver damage. Little is known of the effects of repeated exposures at lower concentrations, but it may also cause kidney damage. Histopathologic changes have been observed in the liver in animal studies (Monsanto, 1967).

TECHNICAL PROFILE

BIS(2-CHLOROETHOXY)METHANE

Bis(2-chloroethoxy)methane, $C_5H_{10}Cl_2O_2$, is a chloroalkyl ether which is also known as dichloroethyl formal, bis(beta-chloroethyl)formal and BCEXM. It is a liquid with a boiling point of 217.5°C and density of 1.23. The chloroalkyl ethers are used in organic synthesis, textile treatment, polymer and insecticide manufacturing, as degreasing agents and solvents and in the preparation of ion exchange resins.

Classification

Bis(2-chloroethoxy)methane has a weight of evidence of D, it is not classifiable as to human carcinogenity.

Health Effects

There is little specific data on the health effects of BCEXM (Sittig, 1985). It is listed as a skin and eye irritant and a high toxic hazard by oral, inhalation and dermal routes. (Sax)

TECHNICAL PROFILE

BIS(2-CHLOROETHYL)ETHER

Bis(2-Chloroethyl)ether, $ClC_2H_4OC_2H_4Cl$, is also known as dichloroethyl ether and BCEE. It is a colorless liquid with a strong unpleasant odor. Bis(2-chloroethyl)ether is used as a general solvent, in textile scouring and cleansing, in wetting and penetrating compounds, in organic synthesis, in varnishes, lacquers and finish removers, in dry cleaning and as a soil fumigant.

Classification

Bis(2-Chloroethyl)ether has a B2 weight of evidence, based on positive carcinogenicity results in two strains of mice and evidence of mutagenicity. No human carcinogenicity data is available.

Health Effects

BCEE is irritating to the eyes, nose, skin and respiratory passages. It may cause vomiting, retching and coughing. There may be a delayed response of several days resulting in lung lesions (the concentration needed to damage the respiratory tract is easily detected by odor). In high concentrations it can be a central nervous system depressant. BCEE may be fatal by inhalation, ingestion or absorption through the skin.

TECHNICAL PROFILE

CHLOROFORM

Chloroform, $CHCl_3$ a trihalomethane, also known as trichloromethane, is a colorless, heavy, volatile liquid with a characteristic odor and sweet taste. It is used as a solvent, in fluorocarbon refrigerants, fluorocarbon plastics, fumigants, insecticides and analytical chemistry. It was formerly used as an anesthetic.

Classification

Chloroform has a weight of evidence of B2, a probable human carcinogen. This is based on increased incidence of several tumor types in rats and mice. There is inadequate human carcinogenicity data.

Health Effects

Acute health effects may include: vomiting, dizziness, salivation, nausea, fatigue, headache, eye and skin irritation, coma, and rapid death caused by cardiac arrest.

Effects of chronic exposure include loss of appetite, hallucinations, moodiness, physical and mental slugginess, enlargement of the liver, and kidney damage. Chloroform exposure seems to affect alcoholics sooner and more severely.

TECHNICAL PROFILE

CHROMIUM

Chromium is a naturally occurring element that is found in soil and in volcanic dusts and gasses. It is found in the environment in three major states, chromium (0), chromium (III), and chromium (VI). Chromium (III) occurs naturally in the environment, while chromium (0) and chromium (VI) are generally produced by industrial processes. Chromium (0) is the metallic form and is used in steel making and for electroplating. Other chromium compounds are made by the chemical industry for use as pigments, and in leather tanning, rubber making, wood treatment, and water treatment.

Classification

The USEPA weight-of-evidence classification for hexavalent chromium is A, a human carcinogen by the inhalation route. Results of epidemiologic studies are consistent across investigators and locations. Dose-response relationships for lung tumors have been established. Evidence for other chromium compounds (trivalent and metallic) is inconclusive.

Health Effects

The three forms of chromium have different effects on health. Hexavalent chromium is irritiating. Acute effects may include: ulcers of the skin, irritation of the nasal mucosa, perforation of the septum and gastrointestinal irritation. Kidney and liver damage, and inflammation and ulceration of the gastrointestinal tract are also possible, as are chronic effects.

Trivalent chromium is an essential nutrient. The minimum daily requirement for optional health is not known, but it is estimated that a daily ingestion of 50-200 micrograms/day is safe and adequate. Chromium (III) may be harmful at very high doses.

The health effects of metallic chromium (chromium (0)) are not well characterized.

Epidemiologic studies of chromate production facilities in the United States, Great Britain, Japan, and West Germany have established an association between chromium exposure and lung cancer. Most of these studies did not attempt to determine whether Cr III or Cr VI compounds were the etiologic agents. Three studies of the chrome pigment industry, one in Norway, one in England, and the third in the Netherlands and Germany also found an association between occupational chromium exposure (predominantly to Cr VI) and lung cancer. Hexavalent chromium compounds were carcinogenic in animal assays producing the following tumor types: intramuscular injection site tumors, intraplural implant site tumors for various chromium VI compounds, intrabronchial implantation site tumors for various Cr VI compounds, and subcutaneous injection site sarcomas.

TECHNICAL PROFILE

CHRYSENE

Chrysene is one of the polycyclic aromatic hydrocarbon (PAH) compounds. Because it is formed when gasoline, garbage, or any animal or plant material burns, it is usually found in smoke and soot. This chemical combines with dust particles in the air and is carried into water and onto soil and crops. Chrysene is found in coal tar pitch used by industry as an adhesive.

People may be exposed to chrysene from environmental sources such as air, water, and from tobacco smoke and overcooked food. Typical exposures are not usually to chrysene alone, but to mixtures of similar compounds.

Classification

The USEPA weight-of-evidence classification for chrysene is B2, a probable human carcinogen on the basis that no human data and sufficient data from animal bioassays exists.

Health Effects

Chrysene produced carcinomas and malignant lymphoma in mice after intraperitoneal injection and skin carcinomas in mice following dermal exposure. In mouse skin painting assays chrysene tested positive in both initiation and complete carcinogen studies. Chrysene produced chromosomal abnormalities in hamsters and mouse germ cells after gavage exposure, positive responses in bacterial gene mutation assays and transformed mammalian cells exposed in culture. It was shown to be a complete carcinogen. Chrysene has produced positive results for initiating activity in several mouse strains when applied in combination with various promoting agents producing skin papillomas and carcinomas.

Although there are no human data that specifically link exposure to chrysene to human cancers, chrysene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke.

TECHNICAL PROFILE

CYANIDE (NOS)

Cyanide (NOS) is used primarily in the extraction of ores, electroplating, metal treatment, and various manufacturing processes. Cyanide is commonly found in certain rat and pest poisons, silver and metal polishes, photographic solutions, and fumigating products.

Classification

The weight of evidence for classification as to human carcinogenicity is Group D, it is not classifiable as to human carcinogenicity. Pertinent data regarding human and animal carcinogenicity have not been located.

Health Effects

Cyanides are extremely poisonous. Death can occur within seconds of inhalation or ingestion. Death is due to respiratory arrest of central origin. Cyanide is readily absorbed from all routes, including the skin, and by inhalation. Alkali salts are toxic when ingested. Death may occur with absorption of even small amounts of cyanide compounds and can occur within minutes or hours depending on route of exposure. Inhalation of toxic fumes represents a potentially rapid fatal type of exposure.

Symptoms of cyanide poisoning include: salivation, nausea, anxiety, confusion, vertigo, giddiness, lower jaw stiffness, convulsions, paralysis, coma, cardiac arrhythmias and transient respiratory stimulation followed by respiratory failure.

TECHNICAL PROFILE

DIBENZOFURAN

Dibenzofuran is formed as a by-product in the manufacture of chlorinated herbicides, and is produced during the combustion of PCBs. It is not found in a pure form, but as a component of mixtures of dioxins and furans produced in a similar fashion.

Classification

The USEPA weight-of-evidence classification of dibenzofuran is D, not classifiable as to human carcinogenicity based upon no human data and no animal data for dibenzofuran alone.

Health Effects

There is no data on the possible carcinogenicity of dibenzofuran alone in humans. Studies have evaluated exposure to a mixture of polychlorinated biphenyls (PCBs), polychlorinated dibenzofurans (PCDFs) and polychlorinated quinones (PCQs) by consumption of contaminated rice oil. However, these studies have limited value because they do not assess dibenzofuran or correlate exposure with cancer risk. Additionally, because of the multiple exposures, the extent to which the various components contributed to the increase in cancer mortality cannot be determined.

No animal carcinogenicity data on dibenzofuran is currently available. The U.S. EPA (1986) noted that the biological activity of PCDFs varies greatly, so that risk assessment of dibenzofuran by analogy to any of these more widely studied compounds would not be recommended.

TECHNICAL PROFILE

DI-N-BUTYLPHTHALATE

Di-n-butylphthalate, $C_{16}H_{22}O_4$, is a colorless, oily liquid with a weak aromatic odor. It is used in plasticizing vinyl acetate emulsion systems, in plasticizing cellulose esters, and as an insect repellent.

Classification

USEPA weight of evidence classification for DBP is group D, on the basis that no pertinent data are available to evaluate DBP for carcinogenicity in humans and animals.

Health Effects

DBP can irritate the nasal passage, upper respiratory system, and stomach. No medical conditions have been found for this substance.

TECHNICAL PROFILE

1,2 DICHLOROBENZENE

1,2-Dichlorobenzene, $C_6H_4Cl_2$, also known as 1,2-DCB, is a colorless to pale yellow liquid that is used as a process solvent in the manufacturing of toluene diisocyanate and as an intermediate in the synthesis of dye stuffs, herbicides, and degreasers.

Classification

This chemical is among those substances being evaluated by the USEPA for evidence of human carcinogenic potential. This does not imply that this chemical is necessarily a carcinogen.

Health Effects

Human exposure to 1,2-DCB is reported to cause hemolytic anemia and liver necrosis. Dichlorobenzenes in general are toxic to non-human mammals, birds, and aquatic organisms and impart an offensive taste and odor to water (Sittig, 1985). Persons with pre-existing pathology (hepatic, renal, and central nervous system) or metabolic disorders and are who taking certain drugs (hormones or other metabolically active), might be considered risks from exposure to 1,2-DCB. Irritation of eyes and nose, liver and kidney damage, and skin blisters may appear upon 1,2-DCB exposure.

TECHNICAL PROFILE

1,3-DICHLOROBENZENE

1,3-Dichlorobenzene is one of a group of chlorinated benzene compounds. These compounds are rarely found in their pure form, but usually occur as mixtures. They are by-products of the manufacture of dyes and pesticides.

Classification

The USEPA weight of evidence classification for 1,3-dichlorobenzene is D, not classifiable as to human carcinogenicity on the basis of no human data, no animal data and limited genetic data.

Health Effects

Although little is known about the toxicity of health effects of 1,3-dichlorobenzene alone, it is thought that the effects are generally similar to those of chlorobenzene. (see chlorobenzene profile).

TECHNICAL PROFILE

1,4-DICHLOROBENZENE

In pure form, 1,4-dichlorobenzene is a white crystalline material that is volatile at room temperatures with a characteristic penetrating odor. It is used as an insecticidal fumigant, popular for protecting clothes against moths.

Classification

Not classified for carcinogenicity by USEPA.

Health Effects

Vapors may cause irritation to skin, throat, and eyes. Prolonged exposure to high concentrations may cause weakness, dizziness, and loss of weight. Liver injury may develop. (MERCK, 1989)

TECHNICAL PROFILE

1,1-DICHLOROETHANE

1,1-Dichloroethane, CH_3CHCl_2, is also known as ethylidene dichloride and ethylidene chloride. It is a colorless , neutral, flammable liquid with an aromatic odor and saccharin taste. 1,1-Dichloroethane is used as a chemical intermediate, has limited use as a solvent, and was formerly used as an anesthetic.

Classification

1,1-Dichloroethane has a weight of evidence of C, it is a possible human carcinogen. This is based on no human data and limited evidence of carcinogenicity in rats and mice.

Health Effects

1,1-Dichloroethane causes central nervous system depression, skin irritation, drowsiness, unconsciousness and liver and kidney damage.

Female rats have shown an increased incidence of mammary gland adenocarcinomas and hemangiosarcomas. Mice have shown an increased incidence of hepatocellular carcinomas and benign uterine polyps.

TECHNICAL PROFILE

1,2-DICHLOROETHANE

1,2-dichloroethane, $ClCH_2CH_2Cl$, is also known as ethylene dichloride. It is a colorless flammable liquid which has a pleasant odor and sweetish taste. 1,2-dichloroethane is used in the manufacture of ethylene glycol, diaminoethylene, polyvinyl chloride, nylon, rayon and various plastics. It is a solvent, a degreaser, and an extracting agent. It is also used as an antiknock agent in gasoline, a fumigant, in dry cleaning, in photography, xerography, water softening, and in the production of adhesives, cosmetics, pharmaceuticals and varnishes.

Classification

1,2-Dichloroethane has a weight of evidence of B2. It is a probable human carcinogen. There is no human data. Several tumor types were induced in rats and mice treated by gavage. Lung papillomas developed in mice after topical application.

Health Effects

Acute exposure to 1,2-dichloroethane may cause nausea, vomiting, eye damage, confusion, dizziness and pulmonary edema. Death has resulted from respiratory and circulatory failure. Chronic exposure can cause dry, scaly, fissured dermatitis; neurological changes; loss of appetite and other gastrointestinal problems; irritation of the mucous membranes; and liver and kidney damage.

TECHNICAL PROFILE

1,2-DICHLOROETHENE (TOTAL)

1,2-Dichloroethene, $C_2H_2Cl_2$, exists in 2 isomers: 60% cis and 40% trans. It is also known as acetylene dichloride. At room temperature it is a flammable, colorless liquid with a pleasant odor. It decomposes slowly when exposed to air, light and moisture. 1,2-dichloroethene's uses include: use as a general solvent for organic materials; dye extraction; in the manufacture of lacquers, perfumes, thermoplastics and pharmaceuticals; as a refrigerant and in the extraction of rubber.

Classification

The trans and cis isomers have not been evaluated for evidence of human carcinogenic potential.

Health Effects

1,2-dichloroethene can cause dermatitis and irritation of the mucous membranes. In high concentration it is a narcotic and causes central nervous system depression. Acute symptoms include dizziness, nausea, vomiting and central nervous system intoxication similar to alcohol intoxication. Transient renal effects have been observed.

TECHNICAL PROFILE

2,4-DIMETHYLPHENOL

2,4-Dimethylphenol, $(CH_3)_2C_6H_3OH$, is a white crystalline solid. 2,4-dimethylphenol is a naturally occurring substituted phenol derived from the cresol fraction of petroleum or coal tars. It's uses include: disinfectants, solvents, pharmaceuticals, biocides, rubber chemicals, additives to lubricants and gasolines, and as a constituent in a wide range of commercial products for industry and agriculture.

Classification

2,4-dimethylphenol has not been evaluated by the USEPA for human carcinogenic potential. Animal studies suggest that it may be a topical carcinogen.

Health Effects

Toxic by ingestion and dermal absorption (HAwley, G., 1981).

TECHNICAL PROFILE

DIMETHYLPHTHALATE

Dimethylphthalate, $C_6H_4(COOCH_3)_2$, is a colorless oily liquid with a slightly aromatic odor. It is also known as phthalic acid and dimethyl ester. Dimethylphthalate's uses include: plasticizer for nitrocellulose and cellulose acetate, resins, rubber, solid rocket fuels, lacquers, plastics, coating agents, safety glass, molding powders and insect repellant.

Classification

The weight of evidence for dimethylphthalate is D, not classifiable.

Health Effects

Health effects include irritation of the nasal passages and upper respiratory system, stomach irritation, eye pain, gastrointestinal irritation, central nervous system depression, coma and hypotension.

TECHNICAL PROFILE

DI-N-OCTYLPHTHALATE

Di-n-octylphthalate, $C_6H_4(COOC_8H_{17})_2$, is a liquid which is also known as DOP. It is used as a plasticizer in the manufacture of plastics products.

Classification

Not classified for carcinogenicity by USEPA.

Health Effects

Di-n-octylphthalate is an eye and skin irritant. As a group, the phthalic acid esters are oily liquids used as intermediates in manufacturing or as lubricants. They are generally toxic in high concentrations and some are considered to be carcinogenic.

TECHNICAL PROFILE

ETHYLBENZENE

Ethylbenzene, C_8H_{10}, is a colorless liquid with a pungent aromatic odor. It is used in the manufacture of cellulose acetate, styrene, and synthetic rubber.

Classification

The U.S. EPA weight of evidence classification is Group D: not classifiable as a human carcinogen based on the lack of animal bioassays and human studies.

Health Effects

Ethylbenzene is moderately toxic by irritation to skin, eyes, and mucous membranes, and by ingestion and inhalation routes (Sax/Lewis, 1987). The liquid is an irritant to the skin and mucous membranes. A concentration of 0.1 percent of vapor in the air is an irritant to human eyes, and a concentration of 0.2 percent is extremely irritating at first, then causes dizziness, irritation of the nose and throat, and a sense of constriction in the chest. Exposure to 1 percent concentration has been reported as causing anoxia, loss of consciousness, tremor of the extremities, and finally death through respiratory failure. Pathological findings were congestion of the brain and lungs, with edema. Ethylbenzene is an experimental teratogen.

TECHNICAL PROFILE

BIS(2-ETHYLHEXYL)PHTHALATE

Bis(2-ethylhexy)phthalate, $C_6H_4(COOCH_2C_2H_5CH_2CH_2CH_2CH_3)_2$, is as colorless oily liquid with almost no odor. It is also known a BEHP. BEHP is produced by the reaction of 2-ethylhexyl alcohol and phthalic anhydride. It is used as a plasticizer for resin and in the manufacture of organic pump fluids.

Classification

The USEPA weight of evidence has classified BEHP in Group B2: a probable human carcinogen. This is based on studies where orally administered BEHP produced significant dose-related increases in liver tumor responses in rats and mice of both sexes.

Health Effects

BEHP can be inhaled, ingested, and be a skin and eye irritant. It may affect the upper respiratory and gastrointestinal systems. Symptoms may include irritation of the eyes and mucous membranes; nausea; and diarrhea (Sittig, 1985).

TECHNICAL PROFILE

FLUORANTHENE

Fluoranthene is one of the polycyclic aromatic hydrocarbon (PAH) compounds. Because it is formed when gasoline, garbage, or any animal or plant material burns, it is usually found in smoke and soot. This chemical combines with dust particles in the air and is carried into water and onto soil and crops. Fluoranthene is found in coal tar pitch used by industry as an adhesive.

People may be exposed to fluoranthene from environmental sources such as air, water, and from tobacco smoke and overcooked food. Typical exposures are not usually to fluoranthene alone, but to mixtures of similar compounds.

Classification

The USEPA weight-of-evidence classification for fluoranthene is D, not classifiable as to human carcinogenicity on the basis of no human data and inadequate data from animal bioassays.

Health Effects

Although fluoranthene has not exhibited the properties of a mutagen or primary carcinogen, there is concern about its toxicity. This concern is based on the fact that it is widespread in the environment and that it belongs to the PAH group which includes numerous potent carcinogens.

In a 13 week mouse oral subchronic toxicity study where mice were gavaged with a range of doses of fluoranthene, all treated mice exhibited nephropathy, increased salivation, increased liver enzyme levels and increased liver weights in a dose-dependent manner. Microscopic liver lesions (indicated by pigmentation) were observed in 65 and 87.5% of the mid- and high-dose mice, respectively.

TECHNICAL PROFILE

FLUORENE

Fluorene is one of the polycyclic aromatic hydrocarbon (PAH) compounds. Because it is formed when fossil fuels, garbage, or any other plant or animal material is burned, it is usually found in smoke and soot. This chemical combines with dust particles in the air and is carried into water and onto soil and crops. Fluorene is found in coal tar pitch used by industry as an adhesive.

Although there is no human data that specifically links exposure to fluorene with human cancers, it is a component of mixtures that have been associated with human cancer. These include coal tar, soot, coke oven emissions, over-cooked food and tobacco smoke.

Classification

USEPA weight-of-evidence classification for fluorene is D, not classifiable as to human carcinogenicity based upon no human data and inadequate data from animal bioassays.

Health Effects

Mice were exposed to fluorene suspended in corn oil by gavage for 13 weeks. Increase salivation, hypoactivity, and urine-wet abdomens in males were observed in all treated animals. The percentage of mice exhibiting hypoactivity was dose-related. Labored respiration, ptosis (drooping eyelids), and unkempt appearance were also observed. A significant decrease in red blood cell count, packed cell volume, and hemoglobin concentration was observed. Increased total serum bilirubin levels were also observed. A dose-related increase in relative liver weight was observed in treated mice. A significant increase in absolute and relative spleen and kidney weight was observed in mice exposed to fluorene. Increases in the absolute and relative liver and spleen weights in high-dose males and females were accompanied by histopathological increases in the amounts of hemosiderin in the spleen and in the Kupffer cells of the liver. No other histopathological lesions were observed.

TECHNICAL PROFILE

2-HEXANONE

2-Hexanone, $CH_3COC_4H_9$, is also known as n-methylbutylketone, MBK, or n-butylmethylketone. It is a flammable colorless liquid with an odor which is similar to, but more pungent than, acetone. It is slightly soluble in water and soluble in alcohol and ether.
2-Hexanone is used as an industrial solvent.

Classification

2-Hexanone has not been evaluated by USEPA for evidence of human carcinogenic potential.

Health Effects

2-Hexanone may cause irritation of the eyes and mucous membranes. High vapor concentration can cause respiratory irritation, central nervous system depression, narcosis, coma, and cardiorespiratory failure. The effects of chronic inhalation at low dose levels include degenerative axonal changes, primarily in the peripheral nerves and long spinal cord tracts; and depression of the circulating white blood cell count. High dose levels can cause atrophy of the epithelium of testicular germ cells.

Toxic neuropathy in workers was reported when 2-hexanone was substituted for methyl isobutyl ketone. The exposed workers experienced distal symmetrical sensory changes and weaknesses (the legs being most severely affected), and unexplained weight loss. The time between the ketone substitution and the onset of neuropathy was approximately 7 months. Similar cases with painters, cabinet finishers and a screen cleaner have been documented. In all of these cases exposure was probably a combination of dermal and inhalation routes. (Clayton, 1982) Study has shown that 2-hexanone is readily absorbed by inhalation, gastrointestinal and percutaneous routes. A slow excretion rate may allow the accumulation of neurotoxic metabolites within the body.

TECHNICAL PROFILE

INDENO(1,2,3-C,D)PYRENE

Indeno(1,2,3-c,d)pyrene is one of the polycyclic aromatic hydrocarbon (PAH) compounds. Because it is formed when fossil fuels, garbage, or any other plant or animal material is burned, it is usually found in smoke and soot. This chemical combines with dust particles in the air and is carried into water and onto soil and crops. Indeno(1,2,3-c,d)pyrene is found in coal tar pitch used by industry as an adhesive.

Classification

The USEPA weight-of-evidence classification for indeno(1,2,3-c,d)pyrene is B2, probable human carcinogen based upon no human data and sufficient data from animal bioassays.

Health Effects

Probable human carcinogen. There is animal data that specifically links exposure to indeno(1,2,3-c,d)pyrene with cancers. It produced tumors in mice following lung implants, subcutaneous injection and dermal exposure. Indeno(1,2,3-c,d)pyrene is a component of mixtures that have been associated with human cancer. These include coal tar, soot, coke oven emissions, over cooked food and tobacco smoke.

TECHNICAL PROFILE

IRON

Iron has four naturally occurring valences (+2, +3, rarely +4, +6), and four stable isotopes. After oxygen, iron is the most commonly used element in manufacturing. Iron and its compounds have numerous uses.

Classification

Not classified for carcinogenicity by the USEPA.

Health Effects

Because of the large number of compounds, the range of toxicity by oral exposure is from non-toxic to highly toxic (MERCK, 1989). Iron is essential to most plant and animals in the naturally occurring valences (+2, +3). Inhalation of dusts can cause irritation of mucous membranes.

TECHNICAL PROFILE

ISOPHORONE

Isophorone is a colorless or pale liquid with a sharp peppermint like odor. This cyclic ketone is also known as trimethylcyclohexanone and isoacetophorone. Isophorone is used in solvent mixtures for finishes, polyvinyl and cellulose resins, herbicides, pesticides, and printing inks.

Classification

Isophorone has been given a weight-of-evidence classification of C, a possible human carcinogen.

Health Effects

Isophorone is irritating to the eyes, skin, nose and throat. It may cause dizziness, fatigue, headache, and dermatitis.

TECHNICAL PROFILE

METHYLENE CHLORIDE

Methylene chloride, CH_2Cl_2, is a nonflammable, colorless liquid with a pleasant aromatic odor. It is also known as dichloromethane. It is used as a solvent for degreasing and cleaning fluids and as a solvent for food processing.

Classification

The weight of evidence classification given to methylen chloride is B2, recognizing this substance as a probable human carcinogen. Human carcinogenicity data is inadequate.

Health Effects

Methylene chloride can irritate the eyes, nose, and throat. It can be inhaled as a vapor, absorbed through the skin, or ingested. Methylene chloride is a mild narcotic. Effects from intoxication include headache, giddiness, stupor, irritability, numbness, and tingling in the limbs. Irritation to the eyes and upper respiratory passages occurs at higher dosages. In severe cases, observers have noted toxic encephalopathy with hallucinations, pulmonary edema, coma, and death. Cardiac arrhythmias have been produced in animals but have not been common in human experience. Exposure to this agent may cause elevated carboxy hemoglobin levels which may be significant in smokers, workers with anemia or heart disease, and those exposed to carbon monoxide.

TECHNICAL PROFILE

2-METHYLNAPHTHALENE

2-Methylnaphthalene is a polycyclic aromatic hydrocarbon (PAH) compound. It is formed when fossil fuels, garbage, or any plant or animal material is burned, and, therefore, it is usually found in smoke and soot. 2-Methylnaphthalene is found in cigarette smoke, power plant emissions, and coal tar pitch.

Classification

Although not classified by USEPA, 2-methylnaphthalene is structurally similar to non-carcinogenic PAH compounds.

Health Effects

2-Methylnaphthalene is a component of PAH mixtures that occur in coal tar, tobacco smoke, and emissions from power plants and foundries. Although specific information on 2-methylnaphthalene is not available, these mixtures have toxicological effects including death, cancer, and reproductive failure.

TECHNICAL PROFILE

4-METHYL-2-PENTANONE

4-Methyl-2-pentanone, $(CH_3)_2CHCH_2COCH_3$, is also known as methyl isobutyl ketone and MIBK. It is a colorless, flammable liquid with a pleasant odor. It is used as a solvent for paints, varnishes and nitrocellulose lacquers; in the manufacture of methyl amyl alcohol; in extraction processes; in organic synthesis and as a denaturant for alcohol.

Classification

MIBK has not been evaluated by the USEPA for evidence of human carcinogenic potential.

Health Effects

MIBK exposure may cause irritation of eye and mucous membranes, dermatitis, headaches, narcosis, coma, and death.

TECHNICAL PROFILE

2-METHYLPHENOL

2-Methylphenol, $C_6H_4OHCH_3$, is also known as o-cresol. It is a white crystal with a phenol like odor. 2-Methylphenol's uses include: disinfectant; phenolic resins; ore floatation; textile scouring agent; organic intermediate; manufacture of salicylaldehyde, coumarin and herbicides; surfactant; and synthetic food flavors.

Classification

O-cresol has a weight-of-evidence of C. It is a possible human carcinogen. This is based on an increased incidence of skin papillomas in mice. There is inadequate human carcinogenicity data.

Health Effects

Acute exposure by all absorption routes may cause muscular weakness, gastroenteric disturbances, severe depression, collapse, paralysis, and death. 2-Methylphenol is very corrosive on tissues and will cause burns and dermatitis. Edema of the lungs; and kidney, liver, pancreas and spleen injury may also occur. Chronic exposure may cause digestive disturbances, liver and kidney damage, skin eruptions, nervous disorders, and vertigo.

TECHNICAL PROFILE

4-METHYLPHENOL

4-Methylphenol, $CH_3C_6H_4OH$, is also known as p-cresol. It is crystalline with a phenol like odor. Its uses include: disinfectant; phenolic resins; ore floatation; textile scouring agent; organic intermediate; manufacture of salicylaldehyde, coumarin and herbicides; surfactant; and synthetic food flavors.

Classification

P-Cresol has a weight of evidence of C. It is considered a possible human carcinogen.

Health Effects

4-Methylphenol is a toxic germicide. Cresols are poisonous, and 8 grams or more taken orally can produce rapid circulatory collapse and death. Chronic poisoning from oral or percutaneous absorption may produce digestive disturbances, nervous disorders, and vertigo. Cresol is moderately toxic by ingestion and inhalation. The main hazard accompanying its use lies in severe chemical burns and dermatitis.

TECHNICAL PROFILE

NAPHTHALENE

Naphthalene is one of the polycyclic aromatic hydrocarbon (PAH) compounds. Because it is formed when gasoline, garbage, or any animal or plant material burns, it is usually found in smoke and soot. This chemical combines with dust particles in the air and is carried into water and onto soil and crops. Naphthalene is found in coal tar pitch used by industry as an adhesive. It may be used as insecticide.

People may be exposed to naphthalene from environmental sources such as air, water, and soil, and from cigarette smoke and overcooked food. Typical exposures are not usually to naphthalene alone, but to a mixture of similar chemicals.

Classification

USEPA weight-of-evidence classification - D; not classifiable as to human carcinogenicity on the basis that no human data and inadequate data from animal bioassays exist.

Health Effects

Naphthalene is a primary irritant. It will cause erythema and dermatitis upon repeated contact. It is an allergen and may cause dermatitis in hypersensitive persons. Eye contact with naphthalene dust has caused irritation and cataracts. Ingestion or inhalation of high vapor concentrations may cause intravascular hemolysis. Eye irritation, headache, confusion, excitement, malaise, profuse sweating, nausea, vomiting, abdominal pain and bladder irritation are the initial symptoms. Progressive jaundice, hematuria, hemoglobinuria, a blockage of the renal tubules and acute renal shutdown may occur. Blood effects include: red cell fragmentation, icterus, severe anemia, leukocytosis and decreases in hemoglobin, hematocrit and red blood cell count. Liver damage is another effect of naphthalene.

TECHNICAL PROFILE

NICKEL

Nickel, Ni, is a lustrous, hard ferromagnetic material. It is used for nickel plating, for various alloys, for coins, storage batteries, magnets, lightening rod tips, machinery parts, and as a catalyst for the hydrogenation of oils and other organic substances. Probably the largest use of nickel is in the manufacture of Manel metal, stainless steels, nickel-chrome resistance wires, and in alloys for electronic and space applications (Merck, 1987).

Classification

The U.S. EPA weight of evidence classification is Group A: stating nickel to be a human carcinogen. This classification is based on human data in which exposure to nickel refinery dust caused lung and nasal tumors in refinery workers, and on animal data in which carcinomas were produced in rats by inhalation and injection.

Sufficient human carcinogenicity data exists. Nickel refinery dust from pyromethallurgical sulfide nickel matte refineries is considered a human carcinogen when inhaled. Evidence of carcinogenicity includes a consistency of findings across different countries in several epidemiologic studies (Clydach, Wales; Copper Cliff, Ontario; Port Colborne, Ontario; Kristiansand Norway and Huntington, West Virginia). Specific tumor sites (lung and nose), high relative risks, particularly for nasal cancer and dose response relationships by length of exposure have been examined. Excess risks are greatest in the dustier areas of the respective refineries.

Health Effects

Nickel and most of its salts are generally considered not to cause acute systemic poisoning. However, ingestion of large doses of nickel compounds have been shown to cause intestinal disorders, convulsions, and asphyxia. Many nickel compounds are experimental carcinogens and some are human carcinogens by inhalation. All nickel contaminated dusts are regarded as carcinogenic by inhalation. The most common effect resulting from exposure to nickel compounds is the development of the "nickel itch". This form of dermatitis occurs chiefly in persons doing

nickel-plating. There is marked variation in individual susceptibility to the dermatitis. It occurs more frequently under conditions of high temperature and humidity, when the skin is moist and chiefly affects the hands and arms. Nickel carbonyl is highly irritating to the lungs and also can produce asphyxia by decomposing with the formation of carbon monoxide. These compounds are common air contaminants (Sax/Lewis, 1987).

TECHNICAL PROFILE

NITROBENZENE

Nitrobenzene, $C_6H_5NO_2$, exists as greenish yellow crystals or as a yellow, oily liquid. It is also known as nitrobenzol or essence of mirbane. The uses of nitrobenzene include: aniline manufacture, solvent for cellulose ethers, modifying esterification of cellulose acetate, paint solvents, leather dressings, metal polishes and shoe polishes.

Classification

USEPA Weight-of-evidence is D, not classifiable as to human carcinogenicity.

Health Effects

The effects of nitrobenzene may be delayed several hours. Nitrobenzene may irritate the eyes. It affects the central nervous system causing fatigue, headache, vertigo, vomiting and general weakness. Severe depression, unconsciousness, coma, and death due to respiratory failure are possible. Nitrobenzene is a powerful methemoglobin former and will cause cyanosis. Chromic nitrobenzene exposure may cause spleen and liver damage, jaundice, and hemolytic icterus. Anemia and Heinz bodies have been observed in the red blood cells. Consumption of alcohol may increase the toxic effects of nitrobenzene.

TECHNICAL PROFILE

N-NITROSODIPHENYLAMINE

N-Nitrosodiphenylamine, $(C_6H_5)_2NNO$, is also known as diphenylnitrosamine. It is greenish crystals which have a melting point of 145°C. N-nitrosodiphenylamine is used as a rubber accelerator. N-nitrosodiphenylamine is structurally related to the carcinogenic nitrosamines.

Human exposure to nitrosamines results from contact with mixtures containing these compounds (e.g., cutting oils, tobacco products). Because of potential confounding by the other substances in these mixtures, data are of limited use in the evaluation of carcinogenicity of individual nitrosamines.

Classification

The USEPA weight-of-evidence classification for n-nitrosodiphenylamine is B2, a probable human carcinogen based upon the increased incidence of bladder tumors in male and female rats, and reticulum cell sarcomas in mice, and its structural relationship to carcinogenic nitrosamines.

Health Effects

N-Nitrosodiphenylamine is an eye irritant. It is a probable human carcinogen.

TECHNICAL PROFILE

PHENANTHRENE

Phenanthrene, $C_{14}H_{10}$, is one of the polycyclic aromatic hydrocarbon (PAH) compounds. It is a colorless, shining crystalline solid. Because it is formed when gasoline, garbage, or any animal or plant material burns, it is usually found in smoke and soot. This chemical combines with dust particles in the air and is carried into water and onto soil and crops. Phenanthrene is found in coal tar pitch used by industry as an adhesive. It is used in dyestuffs, explosives, drug synthesis, and biochemical research.

People may be exposed to phenanthrene from environmental sources such as air, water, and soil, and from cigarette smoke and overcooked food. Typical exposures are not usually to phenanthrene alone, but to a mixture of similar chemicals.

Classification

The USEPA weight-of-evidence classification for phenanthrene is D, not classifiable as to human carcinogenicity on the basis that no human data and inadequate data from a single gavage study in rats and skin painting and injection studies in mice exists.

Health Effects

Phenanthrene is a skin photosensitizer.

TECHNICAL PROFILE

PHENOL

Phenol, C_6H_5OH, is also known as carboxylic acid and hydroxybenzene. When pure it is a white or colorless solid, but it is usually used as a liquid. It has a strong, sweet odor. Phenol has many uses including: phenolic resins; epoxy resins; 2,4-D; selective solvent for lubricating oils; germicidal paints; pharmaceuticals; laboratory reagent; dyes and indicators; slimicide; general disinfectant; wood preservatives; and fertilizer.

Classification

Phenol is classed as a Group D chemical, not classifiable as to human carcinogenicity. This is based on no human data and inadequate animal data.

Health Effects

The health effects of phenol include discoloration, eczema, inflammation, necrosis, sloughing and gangrene of the skin. Oral ingestion may cause the mucous membranes in the throat and esophagus to show swelling, corrosions and necrosis along with hemorrhage and serious infiltration of the surrounding area. Hyperemia and infarcts of the lungs, bronchiopneumonia, purulent bronchitis, and hyperplasia of the peribronchial tissues could be caused by a severe intoxication. Myocardial degeneration and necrosis are also possible.

Symptoms of acute phenol poisoning may include: headache, dizziness, muscular weakness, dimness of vision, ringing ears, irregular rapid breathing, weak pulse and dyspnea. If enough phenol is absorbed loss of consciousness, collapse and death could occur. (Sax)

Effects of severe chronic exposure are systemic disorders such as digestive disturbances (vomiting, difficulty swallowing, ptyalism, diarrhea, and anorexia), and nervous disorders (with headache, fainting, vertigo, and mental disturbances). Kidney, liver, spleen and pancreas damage are also characteristic. (Sax, Clayton)

TECHNICAL PROFILE

PHENOLS (TOTAL)

Phenols are a class of aromatic organic compounds in which one or more hydroxy groups are attached directly to the benzene ring. this group includes phenol, pyrocatechol, resorcinol, hydroquinone, quinone, pyrogallol, o,m, and p-cresol, creosote, pentachlorophenol, other chlorophenols, bromo and iodophenols, o-phenylphenol, d-tert-butylmethylphenol, p-tert-butylphenol and dodecylthiophenol. Phenols are used in a large variety of organic compounds, just a few of which are: explosives, fertilizers, wood preservatives, paints, rubber, synthetic resins and pharmaceuticals.

Classification

Several phenols have been found to cause papillomas and carcinomas in mice. There is no specific evidence of human cancer attributed to phenolic compounds. Phenol is classed as a group D chemical. Phenols, as a group, are not classified.

Health Effects

In general the health effects of the phenol group are similar to those of phenol itself. (See phenol profile.) The following are effects of some phenolic compounds which differ from the effects of phenol:

Pyrocatechol - large doses can cause depression of the central nervous system and a prolonged blood pressure rise. It is more toxic than phenol except by inhalation.

Resorcinol - intoxication symptoms similar to those of phenol but the antipyretic action is more marked.

Hydroquinone - more toxic than phenol. Methemoglobin formation is marked, therefore oxygen carrying capacity of the blood is greatly reduced and anoxia may result.

Quinone - Asphyxia is probably important in terminal cases, due to pulmonary damage caused by excretion of quinone into the alveoli, and the not well known effects of quinone on hemoglobin.

Pyrogallol - its strong reducing action gives it a tremendous affinity for oxygen in the blood possibly causing anoxia.

Pentachlorophenol - when absorbed by animals in sufficient quantity it causes accelerated respiration and blood pressure, hyperpyrexia, hyperglycemia, glycosuria and hyperperistalsis.

TECHNICAL PROFILE

POTASSIUM

Potassium, K, is an alkali metal. It is widely found in the environment. It occurs in all soils. Potassium is used in the preparation of potassium peroxide, in heat exchange alloys, as a laboratory reagent, and as a component of fertilizer.

Classification

Potassium is not classified by the USEPA for human carcinogenicity.

Health Effects

The toxicity of potassium compounds is almost always that of the anion. Potassium is a dangerous fire and explosion hazard. Potassium metal may explode violently when cut or handled. It can ignite spontaneously in moist air. Burning potassium is difficult to extinguish.

TECHNICAL PROFILE

PYRENE

Pyrene, $C_{16}H_{10}$, is a condensed ring hydrocarbon. It is a colorless solid which is derived from coal tar. Pyrene is used for biochemical research.

Classification

The USEPA weight-of-evidence classification for pyrene is D, not classifiable as to human carcinogenicity on the basis of no human data and inadequate data from animal bioassays.

Health Effects

Pyrene is absorbed by the skin and is a skin irritant. Workers exposed to 3 to 5 mg/m^3 of pyrene exhibited some teratogenic effects. Pyrene is a polycyclic aromatic hydrocarbon (PAH). The acute toxicity of pure PAHs appears low when administered orally or dermally to rats or mice. Human exposure to PAHs is almost exclusively via the gastrointestinal and respiratory tracts, and approximately 99 percent is ingested in the diet. Despite the high concentrations of pyrene to which humans may be exposed through food, there is currently little information available to implicate diet-derived PAHs as the cause of serious health effects.

TECHNICAL PROFILE

SELENIUM

Selenium, Se, is a non-metallic element. It is naturally occurring in rock and soils but is unevenly distributed. Selenium is found as any one of a number of compounds. It is used in electronics, xerographic plates, photocells, magnetic computer cores, solar batteries, rubber accelerators, catalysts and as a trace element in animal feeds.

Classification

There are no data to support USEPA classification of carcinogenicity for selenium.

Health Effects

Selenium is an essential element to life. The normal intake of selenium is 150 mg/day. Higher amounts may cause brittle hair, deformed nails, and loss of feeling and control in arms and legs. Some selenium compounds can cause dermatitis; irritate the upper respiratory tract, eyes, and the mucous membrane of the stomach. Liver damage has been seen in livestock grazing on high selenium soils.

TECHNICAL PROFILE

TETRACHLOROETHENE

Tetrachloroethene, C_2Cl_4, is a colorless liquid with an ether like odor. It is also known as perchloroethylene. Tetrachloroethene is a commercially important chlorinated hydrocarbon solvent and chemical intermediate. It has been widely used as a dry-cleaning agent, textile processing solvent, heat transfer medium, in the manufacture of fluorocarbons, and for vapor degreasing in metal cleaning operations.

Classification

The USEPA weight-of-evidence for tetrachloroethene is B2, a probable human carcinogen. It has been shown to cause mononuclear cell leukemia and kidney tumors in rats, and hepatocellular adenomas and carcinomas in mice. There is inadequate data for humans.

Health Effects

Tetrachloroethene is rapidly absorbed following oral and inhalation exposure, while absorption following dermal exposure is poor. It is an eye and nose irritant. Repeated contact may cause a dry, scaly and fissured dermatitis. Acute exposure to tetrachloroethene may cause central nervous system depression, hepatic injury and anesthetic death.

Animal experiments have produced cardiac arrhythmias and renal injury. Malaise, dizziness, headache, increased perspiration, fatigue, staggering gait and slowing of mental ability are symptoms of overexposure. The principal target organs of tetrachloroethene are the central nervous system, liver, and kidneys.

TECHNICAL PROFILE

TOLUENE

Toluene, C_7H_8, is also known as methylbenzene. It is a colorless liquid, with a sweet, pungent, benzene-like odor. It is derived from coal tar. Toluene is used in the manufacture of benzene, as chemical feed, as a solvent for paints and coatings, and as a fuel component.

Classification

The U.S. EPA's weight of evidence classification as to human carcinogenicity is Group D: not classified as a carcinogen based on no human data and inadequate animal data.

Health Effects

Toluene is a poison by intraperitoneal routes. It is moderately toxic by inhalation and subcutaneous routes. It is known to be a skin, eye, and respiratory tract irritant. Toluene affects the central nervous system, the kidney, the liver, and skin. Inhalation of 200 ppm of toluene for eight hours may cause impairment of coordination. With higher concentrations, these effects are increased. Acute exposure to toluene includes symptoms of headache, dizziness, fatigue, drowsiness, and lack of coordination. Chronic effects are anemia and leukopenia, with biopsies showing bone marrow hypoplasia.

TECHNICAL PROFILE

1,2,4-TRICHLOROBENZENE

1,2,4-Trichlorobenzene, $C_6H_3Cl_3$, is a colorless liquid with a pleasant aroma. Its major use is as a dye carrier. It is also used as a solvent in chemical manufacturing, as a dielectric fluid, heat transfer medium, degreaser, lubricant and in insecticides.

Classification

1,2,4-Trichlorobenzene has a weight-of-evidence of D, not classifiable as to human carcinogenicity.

Health Effects

Chlorinated benzenes irritate the skin, conjunctiva, and mucous membrane of the upper respiratory tract. Skin burns may be caused by prolonged or repeated exposure. Acute exposure to chlorinated benzenes may cause drowsiness, incoordination, and unconsciousness. Liver damage has occurred in animal exposures. Animal experiments have indicated that chronic exposure to chlorinated benzenes may result in liver, kidney, and lung damage.

TECHNICAL PROFILE

1,1,1-TRICHLOROETHANE

1,1,1-Trichloroethane, $C_2H_3Cl_3$, is a colorless liquid with a sweet odor. It is also known as 1,1,1-TCE and methyl chloroform. 1,1,1-TCE has found wide use as a substitute for carbon tetrachloride. It is used as a dry cleaning agent, vapor degreasing agent, in textile processing, for cleaning precision instruments, as a propellant and as a pesticide.

Classification

Weight-of-evidence classification by the USEPA is Group C, a possible human carcinogen. Documented evidence of carcinogenicity in animals is available. No evidence in humans is available.

Health Effects

Acute health effects of 1,1,1-TCE may include: eye irritation, mild conjunctivitis, dizziness, incoordination, drowsiness, increased reaction time, unconsciousness, and death. It acts as a narcotic and depresses the central nervous system. Repeated skin contact may cause a dry, scaly, and fissured dermatitis. 1,1,1-TCE may be injurious to the liver and kidneys.

TECHNICAL PROFILE

TRICHLOROETHENE

Trichloroethene C_2HCl_3, a nonflammable mobile liquid, has a characteristic odor resembling chloroform. It is primarily used as a solvent in vapor degreasing. It is also used for extracting caffeine from coffee, as a dry cleaning agent, and as a chemical intermediate in the production of pesticides, waxes, gums resins, tars, paints, and varnishes.

Classification

The EPA has classified trichloroethene in Group B2: sufficient evidence in animals and inadequate evidence in humans. It has been found to induce hepatocellular carcinomas in tests on mice.

Health Effects

Trichloroethene is a poison by inhalation, intravenous and subcutaneous routes. It is moderately toxic by ingestion. Mutagenic data exist. It is an experimental teratogen, carcinogen, and tumorigen. It is a strong skin and eye irritant. Inhalation of high concentrations cause narcosis and anesthesia. A form of addiction has been observed in exposed workers. Prolonged inhalation of moderate concentrations causes headache and drowsiness. Fatalities following severe, acute exposure have been attributed to ventricular fibrillation resulting in cardiac failure. There may also be damage to the liver and other organs from chronic exposure.

TECHNICAL PROFILE

VINYL CHLORIDE

Vinyl chloride, C_2H_3Cl, is an unsaturated, halogenated, aliphatic hydrocarbon. It is also known as monochloroethene and ethylmonochloride. It is a flammable, colorless gas at room temperature with a faintly sweet odor. It is usually handled as a cooled gas. Vinyl chloride is used as a vinyl monomer in the manufacture of polyvinyl chloride and other resins. It is also used as a solvent.

Classification

Vinyl chloride has a weight-of-evidence of A, it is a known human carcinogen by both inhalation and oral routes.

Health Effects

Acute health effects of vinyl chloride include: dizziness, light headedness, nausea, unconsciousness, and irritation of the eyes and skin. Death has been reported from severe vinyl chloride exposures.

Chronic health effects include: thickening of the skin, contact and allergic dermatitis, fatigue, coughing and sneezing, abdominal pain, gastrointestinal bleeding, nausea, vomiting, indigestion, diarrhea, jaundice, weight loss, anorexia, and a cold, tingling sensation of the hands and feet. Increased blood pressure, decreased blood platelet counts, higher liver enzyme counts, restricted blood flow, bone degeneration, liver and spleen enlargement, nervous system disturbances, central nervous system depression, decreased respiratory function and emphysema also occur.

Vinyl chloride is a known human carcinogen by both inhalation and oral routes. It causes angiosarcoma. Workers with a history of exposure have also developed cancers of the lung, brain, skin, nervous system, gall bladder, mouth and pharynx. An increase in fetal mortality among vinyl chloride workers' wives has also been observed. (NIOSH, 1988) NIOSH has reported that chronic inhalation or oral administration of vinyl chloride has caused cancers of the liver, kidney, central nervous system, skin, and mammary and ear duct glands in mice, rats and hamsters.

TECHNICAL PROFILE

XYLENES (TOTAL)

Xylene C_8H_{10}, commonly known as dimethylbenzene, is used as a solvent, a raw material for production of benzoic acid, phthalic anhydride, isophthalic, and terephthalic acids, as well as their dimethyl esters used in the manufacture of polyester fibers; dyes and other organics. It is also used for sterilizing catgut. Xylene exists in three isomeric forms: ortho meta, and para-xylene.

Classification

The weight of evidence classification for human carcinogenicity is Class D: not classifiable as to human carcinogenicity. No human data is available and animal data is inadequate.

Health Effects

Xylene is a poison by ingestion and inhalation. It may affect the central nervous system, eyes, gastrointestinal tract, blood, liver, kidneys, and skin. Xylene vapors may cause irritation to the eyes, nose, and throat. Repeated or prolonged skin contact with xylene may cause drying and defatting of the skin, which may lead to dermatitis. Liquid xylene is irritating to the eyes and mucous membranes, and aspiration may cause chemical pneumonitis, pulmonary edema, and hemorrhage. Repeated exposure of the eyes to high concentrations of xylene vapor may cause reversible eye damage.

Acute exposure to xylene vapor may cause central nervous system depression and minor reversible effects on liver and kidneys. At high concentrations xylene vapor may cause dizziness, staggering, drowsiness, and unconsciousness. At extremely high concentrations, breathing xylene vapors may cause pulmonary edema, anorexia, nausea, vomiting, and abdominal pain.

TECHNICAL PROFILE

ZINC

Zinc, Zn, is a metal with many uses in industry. It can be found in pure form or mixed with other metals to form alloys such as brass, or chemical salts such as zinc chloride. Zinc compounds are found naturally in air, soil and water, and are present in most foods.

Classification

The USEPA weight-of-evidence classification for zinc is D, not classifiable as to human carcinogenicity on the basis of inadequate evidence in humans and animals.

Health Effects

Zinc is an essential element needed by the body in low doses. It can be harmful to the body if too much is taken in. The effects of zinc compounds are variable but generally of low toxicity.

REFERENCES

Agency for Toxic Substances and Disease Registry, May 1989, Toxicological Profile for Aldrin/Dieldrin, ATSDR/TP-88/01.

Agency for Toxic Substances and Disease Registry, July 1989, Toxicological Profile for Chromium, ATSDR/TP-88/10.

Agency for Toxic Substances and Disease Registry, Dec. 1989, Toxicological Profile for bis (12 Chloroethyl) ether, ATSDR/TP/02.

Agency for Toxic Substances and Disease Registry, Dec. 1989, Toxicological Profile for 1,2-Dichloroethane, ATSDR/TP-89/10.

Agency for Toxic Substances and Disease Registry, Dec. 1989, Toxicological Profile for Isophorone, ATSDR/TP-89/15.

Agency for Toxic Substances and Disease Registry, Dec. 1989, Toxicological Profile for Phenol, ATSDR/TP-89/20.

Bishop, W.E., Maki, A.W., 1980. A critical comparison of two bioconcentration test methods. Aquatic Toxicology, ASTM STP 707, J.G. Eaton, P.R. Parrish, A.C. Hendricks, eds. American Society for Testing and Materials. Philadelphia, Pennsylvania.

Buckingham, J., 1984. Dictionary of Organic Compounds. Chapman and Hall. New York, New York.

Callahan, M.A., M.W. Slimak, N.W. Gabel, I.P. May, C.F. Fowler, J.R. Freed, P. Jennings, R.L. Durfee, F.C. Whitmore, B. Maestri, W.R. Mabey, B.R. Holt, C. Gould, 1979.Water Related Environmental Fate of 129 Priority Pollutants. Volume I: Introduction and Technical Background, Metals and Inorganics, Pesticides, and PCBs. U.S. EPA. Washington, C.D. EPA-440/4079-029a.

Callahan, M.A., M.W. Slimak, N.W. Gabel, I.P. May, C.F. Fowler, J.R. Freed, P. Jennings, R.L. Durfee, F.C. Whitmore, B. Maestri, W.R. Mabey, B.R. Holt, C. Gould, 1979. Water Related Environmental Fate of 129 Priority Pollutants. Volume II: Halogenated Aliphatic Hydrocarbons, Halogenated Ethers, Monocyclic

Aromatics, Phthalate Esters, PAHs, nitrosamines, and Miscellaneous Compounds. U.S. EPA. Washington, D.C. EPA-440/4-79-029b.

Clayton, George and Florence, eds. 1982. Pattys Industrial Hygiene and Toxicology 3rd ed. Volumes 2A, 2B, and 2C. John Wiley and Sons, Inc., New York.

Hawley, Gessner G., ed., 1981. The condensed Chemical Dictionary, 10th ed., Van Nostrand Reinhold Company, New York.

Hilsenhoff, W.L., 1988. Rapid Field Assessment of Organic Pollution with Family Level Biotic Index. J. Am. Ornithol. Soc. 7(1) 65 - 68.

Kenaga, E.E., C.A.I. Goring, 1980. Relationship Between Water Solubility, Soil Sorption, Octanol-water Partitioning, and Concentration of Chemicals in Biota. Aquatic Toxicology. ASTM STP 707. J.B. Eaton, P.R. Parrish, A.C. Hendricks, eds. American Society for Testing and Materials. Philadelphia, Pennsylvania.

Mabey, W.R., J.H. Smith, R.T. Podoll, H. Johnson, T. Mill, W. Chou, J. Gates, I.W. Partridge, D. Vandenberg, 1982. Aquatic Fate and Process Data for Organic Priority Pollutants. U.S. EPA. 440/4-81-014. Washington, D.C.

National Institute for Occupational Safety and Health, 1988. Occupational Safety and Health Guidelines, DHHS (NIOSH) Publication #88 - 118. Supplement 1 - OHG, U.S. Dept. of Health and Human Services, Public Health Service, Centers for Disease Control, NIOSH, Cincinnati, Ohio.

Sax, I.N., R.J. Lewis, Sr., 1987. Hazardous Chemicals Desk Reference. Van Nostrand Reinhold Company, Inc. New York.

Sax, I.N., 1984. Dangerous Properties of Industrial Materials, 6th Ed. Van Nostrand Reinhold Company, Inc. New York.

Sittig, M., 1985. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 2nd Ed. Noyes Publications. Park Ridge, New Jersey.

U.S. Environmental Protection Agency. (Continuously updated.) Integrated Risk Information System (IRIS).

USEPA, 1985. Chemical Emergency Preparedness Program: Chemical Profiles. U.S. EPA, Washington, D.C.

USEPA, 1986. Superfund Public Health Evaluation Manual. U.S. EPA Office of Emergency and Remedial Response, Office of Solid Waste and Emergency Response. Washington, D.C. EPA 540/1-86/060.

USEPA, 1988. Superfund Exposure Assessment Manual. U.S. Environmental Protection Agency, Office of Remedial Response, Washington, D.C. EPA/540/1-88/001.

USEPA, 1989. Risk Assessment Guidance for Superfund. Human Health Evaluation Manual, Part A, Interim Final. U.S. EPA. Office of Emergency and Remedial Response. OSWER Directive 9285.7-01a. Washington, D.C.

Warren-Hicks, W., B.R. Parkhurst, S.S. Baker, Jr., 1989. Ecological Assessment of Hazardous Waste Sites: A Field and Laboratory Reference. US. EPA, Environmental Research Laboratory. Corvallis, OR. EPA/600/3-89/013.

Weast, R.C., M.J. Astle, CRC Handbook of Data on Organic Compounds. CRC Press, Inc., Boca Raton, Florida.

Windholz, M., 1983. The Merck Index, 10th Ed. Merck and Co., Inc. Rahway, New Jersey.