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APPENDIX E-4

FUGITIVE DUST MODEL

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FUGITIVE DUST MODEL

ESTIMATION OF FUGITIVE DUST EXPOSURE POINT CONCENTRATIONS FROM ON-SITE DIRT BIKER ACTIVITY

1.00 INTRODUCTION

Exposure point concentrations (EPCs) for potential on-site dirt bike riders on Central Landfill property were calculated using Environmental Protection Agency (EPA) AP-42 emission factors and the Near Field Box dispersion model. The AP-42 emission factor for unpaved roads (Section 13.2.2) was used to determine emission rates from dirt bikers dust generation. These emission rates were used in a transport and dispersion model (Near Field Box) to estimate concentrations of individual compounds in ambient air on-site (EPCs).

2.00 EMISSION FACTOR

The derived emission rates from fugitive dust activities from dirt bikers was based on the highest concentrations of individual compounds detected in surficial soil (0-3 feet) based on previous Central Landfill soil sample collection. The USEPA AP-42 emission factors for unpaved roads was selected to quantify emission rates of fugitive dusts generated from dirt bikers. The emission rate is based on the following equations. Details regarding the modeling assumptions are provided in Table E.4-1 through E.4-3.

Emission Rate:

$$Q(10) = (a)\ (Et)\ (VMT)$$

where:

Q10	=	emission rate of individual compound (mg/hr)
(a)	=	mass fraction of individual compounds in soil (mg/hr)
(Et)	=	emission rate of particles 10 microns and smaller due to dirt bike travel over unpaved surfaces (lbs/VMT)
(VMT)	=	vehicle miles traveled per hour (miles)

where:

$$(Et) = k(5.9)\ [s/12]\ [S/30]\ [W/3]^{0.7}\ [w/4]^{0.5}\ [365-p/365]$$

where:

k	=	particle size multiplier (dimensionless)
s	=	silt content of road surface (%)
S	=	mean vehicle speed (miles per hour)
W	=	mean vehicle weight (tons)
w	=	mean number of wheels
P	=	number of days of at least .254 mm of precipitation per year

The predicted emission rate is presented Table E.4-4

The emission factor derived is based on mean vehicle weights and number of wheels greater than dirt bikes (i.e., trucks). However, based on discussions with personnel from Research Triangle Park (RTP) the developers of the unpaved road emission factor, the application of the unpaved road emission factor for dirt bikes is adequate and is a conservative estimate in the absence of a specific emission factor for dirt bikes at the time of this report.

3.00 DISPERSION MODEL

The Near Field Box model was used to estimate ambient air concentrations of compounds detected in soils on-site, based on compound-specific emission rates estimated by emission factors for dirt bike generating fugitive dusts, dimensions of the area, and average wind speed at the Site. The dimensions of the contaminant area (box) and the average annual wind speed at the Site, were used to set the downward height of the box and the average wind speed through the box, respectively.

TABLE E.4-1

MODELING OF FUGITIVE DUST EMISSION RATES FROM VEHICLE TRAFFIC AND AIR CONCENTRATIONS

Vehicle Traffic Fugitive Dust Emission Rate Calculations For Unpaved Roads

Emission rates of individual compounds in soil and debris were calculated by multiplying the mass fraction of the individual compounds in soil by the total fugitive dust emission rate. The total fugitive dust emission rate from motorcycle traffic was estimated to be equivalent to the fugitive dust generated by vehicle traffic on unpaved roads based on the AP-42 emission factors.

$$Q(10) = (a)\ (Et)\ (VMT)$$

Q (10)	=	emission rate of individual compound (mg/hr)
a	=	mass fraction of individual compounds in soil, debris (mg/kg)
Et	=	total fugitive dust emission rate (kg/hr)
VMT	=	vehicle miles traveled per hour (miles)

TABLE E.4-2

MODELING OF FUGITIVE DUST EMISSION RATES FROM VEHICLE TRAFFIC AND AIR CONCENTRATIONS

E10 - Fugitive Dust Generated by Motor Vehicle Activity

AP-42 EMISSION FACTOR - UNPAVED ROADS (U.S. EPA AP-42 1/95, Sec. 13.2.2)

This formula is used to estimate the emission rate of fugitive dusts into the air from motor vehicle traffic on unpaved roads based on the number of vehicle miles traveled at the site and the silt content of the soils, speed and weight of the vehicle, number of wheels, and precipitation. This equation provides conservative estimates of fugitive dust concentrations from unpaved roads. Actual dust emissions at the site are likely to be less than calculated by this model.

E t = k(5.9) x {(s/12)(S/30)((W/3)^0.7)} x {w/4}^0.5 x {365-p/365}

Et = emission rate of particles 10 microns and smaller due to motor vehicle travel over unpaved surfaces (lbs/VMT)

k = particle size multiplier (dimensionless)
k = 0.4

s = silt content of road surface (%)
s = 6.4 %

S = mean vehicle speed, miles per hour
S = 20

W = mean vehicle weight, tons
W = 0.3

w = mean number of wheels
w = 2

p = number of days of at least .254 mm of precip. per year
p = 120

conversion factor: 1 lbs. = 0.454 kg

Et = 2.9E-02 kg/VMT

VMT= 20 miles traveled

TABLE E.4-3

MODELING OF FUGITIVE DUST EMISSION RATES FOR UNPAVED ROADS AND AIR CONCENTRATIONS

Air Concentration Calculations

NEAR FIELD BOX MODEL **(Pasquill, 1975; Horst, 1979)**

This model is used to estimate air concentrations of compounds based on emission rates, dimensions of the site (box) and average wind speeds at the site. The model is accurate at short downwind distances (i.e., less than 100 meters) and is appropriate for estimation of on-site exposures.

Ca = Q10 / ((Hb) (Wb) (Um))

Ca = concentration of compounds in ambient air on site (mg/m^3)

Q10 = emission rate of compounds (mg/s)
where: Q10 = concentration (mg/hr) x hr/3600 s

Hb = downwind height of box (m)
Hb = 6.2 m (determined from length of box 100m (x) and model constraints)

Wb = width of box (m)
Wb = 1950 m (determined based on site area)

Um = average wind speed through the box (m/s)
Um = 0.22 (u10) ln(2.5 Hb)

u10 = wind speed at 10 m (m/s)
u10 = 5 m/s (annual wind speed for Providence, RI; National Climatic Data Center, observations from 1961 through 1990)

Um = 2.77 m/s

Ca (mg/m^3) = Q10 (mg/s) / Dispersion Factor (m^3/s), where

Dispersion Factor = (Hb) (Wb) (Um) m^3/s
(Hb) (Wb) (Um) = 3.35E+04 m^3/s

TABLE E.4-4

MODELING OF FUGITIVE DUST EMISSION RATES AND AIR CONCENTRATIONS FOR UNPAVED ROADS MOTORCYCLE RIDER CENTRAL LANDFILL

Compound	[a] Concentration In Soil (mg/kg)	(Et) Estimated x Emission Rate x (kg/VMT)	(VMT) Vehicle Miles Traveled (Miles)	(Q10) Compound = Emission Rate / (mg/s)	[Hb * Wb * Um] Dispersion Factor (m³/s)	=	(Ca) Predicted Concentration On-Site (mg/m³)
Semivolatile Organic Compounds							
Benzo(a)pyrene	0.222	2.9E-02	20	3.5E-05	3.35E+04		1.1E-09
Benzo(g,h,i)perylene	0.152	2.9E-02	20	2.4E-05	3.35E+04		7.2E-10
bis(2-Ethylhexyl)phthalate	11.026	2.9E-02	20	1.8E-03	3.35E+04		5.2E-08
Di-n-butylphthalate	0.169	2.9E-02	20	2.7E-05	3.35E+04		8.0E-10
Phenanthrene	0.241	2.9E-02	20	3.8E-05	3.35E+04		1.1E-09
Metals							
Aluminum, total	7611	2.9E-02	20	1.2E+00	3.35E+04		3.6E-05
Arsenic, total	5.545	2.9E-02	20	8.8E-04	3.35E+04		2.6E-08
Barium, total	40	2.9E-02	20	6.4E-03	3.35E+04		1.9E-07
Beryllium, total	1.535	2.9E-02	20	2.4E-04	3.35E+04		7.3E-09
Lead, total	85	2.9E-02	20	1.4E-02	3.35E+04		4.0E-07
Manganese, total	260	2.9E-02	20	4.1E-02	3.35E+04		1.2E-06
Zinc, total	425	2.9E-02	20	6.7E-02	3.35E+04		2.0E-06

Notes:

a = The 95% UCL of the arithmetic mean of each detected constituent in surficial soils was used to predict the exposure point concentration.

Et = Total fugitive dust emission rate, derived using published EPA AP-42 emission factors for unpaved roads. (U.S. EPA, 19

Q10 = Emission rate of individual compound, with a unit conversion factor of 3600 s/hr.

Hb * Wb * Um = Dispersion factor, derived using the Near Field Box Model (U.S. EPA, 1990)

Ca = Predicted Maximum Concentration in the modeled area (U.S.EPA, 1990), used as the exposure point concentration for evaluation of on-site exposures.

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APPENDIX E-5

ABSORPTION ADJUSTMENT FACTORS

Table 4. Recommended Dermal Absorption Factor from Soil

Compound	Dermal Absorption Factor	Reference
Arsenic	0.03	Wester, et al (1993a)
Cadmium	0.001	Wester, et al (1992a) USEPA (1992)
Chlordane	0.04	Wester, et al (1992b)
2,4-Dichlorophenoxyacetic acid	0.05	Wester, et al (1996)
DDT	0.03	Wester, et al (1990)
Dioxins		
TCDD <10% organic soil >10% organic soil	0.03 0.001	USEPA (1992)
PAHs Benzo(a)pyrene	0.13	Wester, et al (1990)
PCBs Aroclor 1254 and 1242 3,3'4,4'-Tetrachlorobiphenyl	 0.14 0.02	 Wester et al (1993b) USEPA (1992)
Pentachlorophenol	0.25	Wester et al (1993c)
Generic Defaults		
Semivolatile organic compounds	0.1	-
Inorganics	0.01	

ARSENIC CAS #: 7440382

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.0003 mg/kg/day (2)
Chronic Oral Reference Dose: 0.0003 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: 1.8 $(mg/kg/day)^{-1}$ (1f)
Inhalation Cancer Unit Risk: Not Volatile

Human studies support the assumption that the soluble salts of inorganic arsenic are almost completely absorbed by the oral route. Literature sources cite an absorption efficiency of 98% for arsenic in humans and laboratory animals (Vahter, 1983; EPA, 1984, 1985; Goyer, 1986).

Vahter, M. (1983) *Metabolism of Arsenic*. In: Fowler, B.A., ed. Biological and Environmental Effect of Arsenic. New York: Elsevier, pp. 171-198.

U.S. Environmental Protection Agency (U.S. EPA) (1985) Health Advisories for 52 Chemicals Which Have Been Detected in Drinking Water. PB86-118338.

U.S. Environmental Protection Agency (U.S. EPA) (1984) Health Assessment Document for Inorganic Arsenic. Final report. Research Triangle Park, NC: Environmental Protection Agency. EPA 600/8-83-021F.

Goyer, R.A. (1986) Casarett and Doull's Toxicology. (C.D. Klaassen, M.O. Amdur and J. Doull, Eds.) 3rd ed., pp. 582-635, MacMillan, New York.

No quantitative studies were located to evaluate the dermal absorption of arsenic compounds. However, clinical symptoms of arsenic poisoning have been reported in humans after accidents where the only route of exposure was through the skin, suggesting that dermal absorption does occur. An absorption efficiency of 3% may be considered a conservative upper-bound based on EP toxicity studies where the extraction of arsenic from soil (Ph 5, 24 hours) averaged 3%.

ARSENIC

The oral RfD for arsenic of 3E-04 mg/kg-day and the proposed oral unit risk factor of 5E-5 $(ug/L)^{-1}$ (backcalculated CSF of $(1.8\ mg/kg\text{-}day)^{-1}$) are based on epidemiological studies that characterized health effects in a large population of Taiwanese who consumed drinking water containing arsenic. The exact form of arsenic is unknown. For the purposes of development of the AAFs, it has been assumed that the arsenic was a soluble inorganic arsenic salt. The solubility of the various arsenic forms is reported to be a critical factor in their bioavailability (U.S. EPA, 1984).

Several investigators have estimated the extent of arsenic absorption following ingestion. Pomroy et al. (1980) administered radiolabeled arsenic acid in gelatin capsules followed by a glass of water. In one subject, approximately 11% was excreted in the feces with most of that (8%) excreted in 3 days. It is not possible to determine what fraction of this fecally excreted arsenic was unabsorbed and what fraction was absorbed and subsequently excreted in the feces. Thus, 89% represents a minimum estimate of absorption. These data are supported by the data from the other five subjects in the study. The average minimum absorption (assumes fecally excreted arsenic was unabsorbed) across all six subjects was 94%. This absorption estimate is supported by a study by Bettley and O'Shea (1975) whose data indicate a minimum absorption of 96%. These studies indicate that virtually all of an orally administered dose of arsenic can be absorbed from the gastrointestinal tract.

If site-specific data indicate that an insoluble form of arsenic is present, than the conservative AAFs calculated below may need to be modified. GZA utilized AAFs derived by the MADEP (1992) for all pathways, when available. However, MADEP did not derive an AAF for dermal contact with chemicals in water, thus GZA derived one.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral RfD and unit risk for arsenic are based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the RfD and unit risk to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (multiplying the RfD or dividing the CSF by the absorption). For arsenic, the AAF (dermal-water) is 1/(100%) = 1.

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Bettley and O'Shea. 1975. The absorption of arsenic and its relation to carcinoma. *British Journal of Dermatology*, 92:563-568.

Pomroy, C, S.M. Charbonneau, R.S. McCollough and G.K.H. Tam. 1980. Human retention studies with ^{74}As. *Toxicology and Applied Pharmacology*, 53:550-556.

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ARSENIC (INORGANIC)

Several studies in humans indicate that the soluble arsenates and arsenites (salts of the oxyacid forms of arsenic) are well absorbed across the gastrointestinal tract. ATSDR (1997) reports that in one study by Bettley and O'Shea, (1975) less than 5% of an oral dose given to humans was excreted in the feces indicating that absorption was at least 95% (the specific study conditions were not presented by ATSDR). ATSDR states that other studies in humans reported that urinary excretion (the primary route of elimination) accounted for 55-80% of daily oral intakes of arsenate or arsenite (Buchet et al 1981; Crecelius, 1977; Mappes, 1977; Tam et al., 1979). The MADEP (1992) utilizes an absorption efficiency of 98% for the soluble salts of inorganic arsenic, based on both U.S. EPA and other published literature sources. The solubility of the various arsenic forms is reported to be a critical factor in their bioavailability (U.S. EPA, 1984). Gastrointestinal absorption may be much lower if highly insoluble forms of arsenic are ingested such as arsenic triselinide, arsenic trisulfide, and lead arsenate (ATSDR, 1997).

Arsenic exhibits varying physiochemical properties depending on its valence state. In the environment, the transport and partitioning of arsenic in water is dependent upon the chemical form (oxidation state and counter ion) of the arsenic and on interactions with other materials present. Soluble forms move with the water in the environment. These forms are also more bioavailable when human exposure occurs. However, arsenic may be adsorbed from water onto sediments or soils, especially clays, iron oxides, aluminum hydroxides, manganese compounds and organic materials (ATSDR, 1997). Once adsorbed, arsenic is less soluble and less bioavailable.

Oral-Soil AAF

The relative Absorption Adjustment Factor (AAF) is used to account for differences in absorption of arsenic when exposure occurs via a route different than that used in the dose-response study (drinking water) and/or when the arsenic is present in a different environmental medium. The Texas Risk Reduction Program (1996) presented data from four studies on the oral bioavailability of arsenic from soil. The mean bioavailability reported in its Draft Document ranged from 8 to 24 percent in dogs, monkeys, swine and rabbits. Based upon these data, the Texas Natural Resource Conservation Commission (TNRCC) chose 20% because its the upper-end of the mean bioavailability determined in three models (monkey, dog and swine) reportedly having similar gastrointestinal systems relative to humans and because 20% is consistent with the mean bioavailabilty for soil arsenic obtained using the rabbit. (The TNRCC apparently assumed that the absorption of arsenic from the drinking water in the Taiwanese population was 100%, since they used the oral-soil value directly as the relative bioavailability factor.) The ATSDR, however, reports that the rabbit has a similar ratio of metabolites as humans, and suggests that this may be the best model for the toxicokinetics in humans.

GZA reviewed the two published studies cited by TNRCC (the dog study by Groen et al., 1994, and the rabbit study by Freeman et al., 1993; the other two were unpublished) and determined that the rabbit study was the most appropriate study from which to derive the AAF. The shortcomings of the dog study were that a total of only six animals were used, the arsenic-soil matrix consisted of arsenic in bog ore-containing soil from the Netherlands which is not representative of the type of soil found near the site, and the fact that the dog was not the recommended animal model. The bioavailability of the arsenic from the soil in the dog study (relative to intravenous administration of arsenic pentoxide (As_2O_5)) was 8%, the lowest value reported by TNRCC. It is likely that this low bioavailability value is reflective of the high adsorption potential of the soil matrix used.

In the rabbit study, the bioavailability of arsenic-containing soil obtained from an area near a smelter in Anaconda, Montana was determined relative to both intravenous (28%, reported by TNRCC as 24% based on normalized i.v. data) and oral gavage (48%) administration of sodium arsenate. The percent bioavailability value presented in this paper for the oral soil arsenic relative to the oral gavage can be used directly as the oral-soil AAF. This allows the relative bioavailability estimate to be based on a single species, thereby eliminating the uncertainties introduced by calculating a relative absorption factor using data obtained from different exposure matrices and animal species, as well as from different study protocols. Thus, although it is possible that the absolute bioavailability in the rabbit may differ somewhat from that in the human, the use of relative bioavailability data from within a single species allows one to evaluate the affect of the soil matrix itself. One would assume that this relative affect (decreased bioavailability due to adsorption to soil matrix) would be the same across species.

Freeman et al. (1993) exposed fasted prepubescent male and female SPF New Zealand White Rabbits (5/sex/treatment group) to a single oral administration of arsenic-containing soil (3900 ppm As) at three different dose levels (0.2, 0.5 and 1.0 g soil/kg body weight, corresponding to 0.78, 1.95 and 3.9 mg As/kg, respectively). The arsenic-containing soil was administered in a gelatin capsule. The test soil contained primarily enargite [Cu_3AsS_4] (48%), and goldfieldite [$Cu_7(Te,As,Sb)S_4$] (14%), with the remainder being present as As(V) oxides and solid solutions of arsenic with copper and iron. Some of the arsenic-bearing solid phases were encapsulated by insoluble silicate minerals, effectively preventing the release of arsenic into the gastrointestinal tract. Thus, the majority of the arsenic in the soil was present in forms that are less soluble than the forms typically found dissolved in water, which, as discussed above, is generally the case in environmental exposures. The particle size of the soil is reported to be similar to the size of ingested particles commonly found to adhere to children's hands.

Other exposure groups included untreated controls, an intravenous sodium arsenate group (1.95 mg As/kg) and a gavage aqueous sodium arsenate group (1.95 mg As/kg). Urine, cage rinse and feces were collected at 24-hour intervals for 5 days and analyzed for total arsenic. Using the

urinary excretion data, the average oral bioavailability of arsenic in soil relative to that of an aqueous solution of sodium arsenate administered by gavage was 48% indicating that the soluble form of arsenic used in the gavage exposures was about twice as bioavailable as the forms of arsenic present in the test soil. Thus, the AAF (oral-soil) is 0.48.

This relative bioavailability value can be used as the oral-soil AAF since it is based on the bioavailability of arsenic in soil relative to that following oral exposure to sodium arsenate in water (the dose response values for inorganic arsenic are based on drinking water exposures). Although some arsenic is excreted in the feces (an average of 8% following intravenous administration), the primary route of elimination is via the urine, and the amount excreted in the urine should be directly proportional to the bioavailability. The use of a relative bioavailability factor calculated from urinary excretion data should provide a reasonable estimate of the relative absorption, assuming that there are no significant differences in elimination of absorbed arsenic following intravenous and oral exposures.

Oral-Water AAF

The dose-response values were based on an epidemiological study involving drinking water exposures. Therefore no adjustment for relative absorption differences is necessary as the absorption following oral water exposures is assumed to be the same as in the dose-response study. The AAF (oral-water), therefore, is 1.

Oral-Diet AAF

Due to a lack of data on the bioavailability of inorganic arsenic present in food (data do indicate that methylated forms of arsenic present in fish are well absorbed), GZA assumed that the absorption would be the same as when it is present in drinking water. Thus, the AAF (oral-diet) equals 98%/98% = 1. This assumption is consistent with MADEP, 1992.

Dermal-Soil AAF

Due to a lack of quantitative data on the dermal absorption of arsenic from soil, the default assumption for inorganics of 1%, recommended by the Michigan Department of Environmental Quality's Environmental Response Division Staff (1995), was used in calculating the AAF. Based upon the assumed absorption utilized by the MADEP (1992), which is supported by information presented by ATSDR (1997), it was assumed that the gastrointestinal absorption of arsenic in the Taiwanese population was virtually complete (98%). Thus, the AAF (oral-soil) for the soluble forms of inorganic arsenic is 1%/98% = 0.01.

Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this process is an absorbed dose. The oral RfD is based on administered dose. An absorption adjustment factor is necessary to convert the RfD to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment was made to the RfD in the risk calculation. The Dose-Response Absorption Correction Factor (DRACF) for dermal-water equals the absorption in the dose-response study. The RfD was multiplied by the DRACF whereas the CSF (which is in inverse units of dose) was divided by the DRACF to obtain absorbed dose based values. Using an assumed gastrointestinal absorption of 98% in the dose-response study (based on MADEP, 1992 and ATSDR, 1997), GZA calculated the DRACF, for use with both the oral RfD and CSF, as 0.98.

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Michigan Department of Environmental Quality (MDEQ), Environmental Response Division (ERD) Staff. 1995. ERD Operational Momorandum #14 - Revision 2. Generic Remedial Action Plans Using Generic Industrial or Generic Commercial Cleanup Criteria and Other Requirements, June 6, 1995.

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TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.05 mg/kg/day (4)
Chronic Oral Reference Dose: 0.005 mg/kg/day (4)

Subchronic Inhalation Reference Concentration: 32 $\mu g/m^3$ (4)
Chronic Inhalation Reference Concentration: 9 $\mu g/m^3$ (3)

Oral Cancer Potency Factor: 0.029 $(mg/kg/day)^{-1}$ (1)
Inhalation Cancer Unit Risk: 0.0000083 $(\mu g/m^3)^{-1}$ (1)

The oral absorption efficiency of pure benzene is estimated to be essentially 100% based on rabbit data in which 100% of an oral dose was either metabolized or exhaled unchanged (Sabourin et al., 1986). The oral bioavailability of benzene was slightly but not significantly increased by adsorption to clay soil (Turkall, et al., 1988). Human (Parke and Williams, 1953) and animal studies suggest that virtually all (100%) of an oral dose of benzene is absorbed.

Parke, D.V. and Williams, R.T. (1953) *Studies in Detoxication. 49. The Metabolism of Benzene Containing [^{14}C] Benzene*. Biochem. J. 54:231-238.

Sabourin, P., Chen, B., Henderson, R. Lucier, G. and Birnbaum, L. (1986) *Effect of Dose on Absorption and Excretion of ^{14}C-Benzene Administered Orally or by Inhalation*. The Toxicologist. 6:163.

Turkall, R.M., Skowronski, G., Gerges, S., Von Hagen, S. and Abdel-Rahman, M.S. (1988) *Soil Adsorption Alters Kinetics and Bioavailability of Benzene in Orally Exposed Male Rats*. Arch. Environ. Contam. Toxicol. 17:159-164.

For dermal absorption, the controlling factor is contact time with the skin. One study (Susten et al., 1990) estimated the non-occluded dermal absorption of benzene in hairless mice to be approximately 1% of the applied dose in 4 hour (or 6% in 24 hours), with volatilization occurring rapidly. In a second study (Susten et al., 1985), the dermal absorption efficiency (non-occluded) of benzene was 1% of the applied dose in 2.5 hours (or 10% in 24 hours). In an occluded study (Lam and Bisgaard, 1989), the dermal absorption efficiency of 1,3-diaminobenzene was 100% of the applied dose in 24 hours for aqueous solutions and hydrogen peroxide-based solutions of this benzene analog. This suggests that the dermal absorption of benzene is highly dependent on whether an occluded or non-occluded study design is utilized. An average non-occluded dermal absorption efficiency (24 hour) of 10% was selected as a protective estimate from these studies while an occluded 24-hr dose would be absorbed 100%. The dermal bioavailability of benzene was slightly reduced by its adsorption to soil (Skowronski, et al., 1988). The

The oral and dermal subchronic reference dose for benzene was derived from an animal inhalation study. An absorption efficiency of 50% was used to calculate an absorbed dose. The RAFs for all scenarios are the absorption efficiencies of benzene by the route in question.

BENZENE RAFs Evaluation of Subchronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
1	0.08	1	1

BENZENE

The MADEP (1992) derived AAFs for evaluation of both the noncancer and cancer effects of benzene. However, MADEP did not derive dermal-water AAFs for this chemical. Thus, GZA has derived dermal-water AAFs for benzene, as described below.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral CSF for benzene is based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the CSF to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (multiplying the RfD, or dividing the CSF, by the percent absorption).

The oral CSF for benzene is based on an occupational study wherein human exposure was via inhalation. The U.S. EPA (1992) assumed that the fraction of the administered dose absorbed systemically via inhalation was the same as that via drinking water. Thus, no absorption adjustment was made to convert the inhalation dose to an ingestion dose. (This is in contrast to what MADEP indicated in the ShortForm. MADEP stated that EPA's CSF was based on an absorbed dose.) MADEP (1992) assumed that the oral absorption of benzene was complete (100%). Thus, GZA assumed that the absorption from the occupational study was 100%. Therefore, the AAF (dermal-water) for use with the CSF equals 1/100% = 1.

The oral RfD for benzene was derived by MADEP (1992), assuming 50% gastrointestinal absorption. Therefore, no further adjustment is necessary. Since this RfD based on absorbed dose, it can be used directly with the absorbed dose calculated for the dermal-water exposure scenario. For consistency, an AAF (oral-water) of 1 was included in the spreadsheets. However, this resulted in no adjustment of the dose.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

U.S. EPA. 1992. Integrated Risk Information System (IRIS).

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TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.04 mg/kg/day (2b)
Chronic Oral Reference Dose: 0.04 mg/kg/day (2f)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: 7.3 $(mg/kg/day)^{-1}$ (1g)
Inhalation Cancer Unit Risk: Not Volatile

No specific quantitative information found on oral or dermal route. Assume same as B[a]P.

The oral and dermal cancer potency value for benzo[a]anthracene is based on a dietary study in which B[a]P was administered to mice. In this study, an applied dose was used.

BENZO[a]ANTHRACENE RAFs Evaluation of Carcinogenicity			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{0.91} = 1$	$\frac{0.18}{0.91} = 0.2$	$\frac{0.91}{0.91} = 1$	$\frac{0.91}{0.91} = 1$

The oral and dermal chronic reference dose for benzo[a]anthracene is based on a naphthalene oral gavage study conducted in rats. In this study, an applied dose was used.

BENZO[a]ANTHRACENE RAFs Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{1} = 0.91$	$\frac{0.18}{1} = 0.18$	$\frac{0.91}{1} = 0.91$	$\frac{0.91}{1} = 0.91$

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.04 mg/kg/day (2b)
Chronic Oral Reference Dose: 0.04 mg/kg/day (2f)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: 7.3 $(mg/kg/day)^{-1}$ (1)
Inhalation Cancer Unit Risk: Not Volatile

The oral absorption of ^{14}C-labeled B[a]P, dissolved in peanut oil and administered by gavage, was studied in rats (Hecht et al., 1979). Absorption was determined by recovery of label in urine and feces. Unchanged B[a]P recovered in feces was estimated at 9% of the total dose, with all other fecal radioactivity (85% of applied dose) recovered as metabolites. This suggests an oral absorption efficiency of 91%.

Hecht, S.S., Grabowski, W. and Groth, K. (1979) *Analysis of Feces for B[a]P After Consumption of Charcoal-Broiled Beef by Rats and Humans*. Food Cosmet. Toxicol. 17:223-227.

The percutaneous absorption of ^{14}C-B[a]P was studied in vivo in Swiss Webster mice (Sanders et al., 1986) and in Sprague-Dawley rats (Yang et al., 1986). Absorption was determined by analyzing radioactivity in urine, feces and tissues, and by analysis of residual label at the site of application. Dermal absorption efficiency was measured as 40% (in mice) and 6% (in rats) in 24 hrs. The higher value of 40% is selected as a protective estimate for human dermal exposure to pure compound. In vitro estimates are lower, ranging from 0.1%-15% in humans and animals (Kao et al., 1985; Kao et al., 1988) and are not considered applicable to human exposure. The in vivo percutaneous absorption of soil-adsorbed B[a]P was determined in rats by Yang et al. (1989). The range of absorbed doses was 1.3% - 9.2% depending on the amount of soil applied. More efficient absorption occurred at lower soil application rates. Wester et al. (1990) confirms a low absorption for soil-associated B[a]P in the rhesus monkey with a range of 9% - 18%. The upper limit of 18% is selected as a protective estimate for human exposure to B[a]P contaminated soil.

Kao, J., Hall, J. and Helman, G. (1988) *In Vitro Percutanous Absorption in Mouse Skin: Influence of Skin Appendages*. Toxicol. Appl. Pharmacol. 94: 93-103.

Kao, J.K., Patterson, F.K., and Hall, J. (1985) *Skin Penetration and Metabolism of Topically Applied Chemicals in Six Mammalian Species, Including Man: An In Vitro Study With Benzo(a)pyrene and Testosterone*. Toxicol. Appl. Pharmacol. 81:502-518.

The oral and dermal chronic reference dose for benzo[a]pyrene is based on a naphthalene oral gavage study conducted in rats. In this study, an applied dose was used. The chronic inhalation reference concentration for benzo[a]pyrene is based on a naphthalene inhalation study in humans. In this study, an applied dose was used.

BENZO[a]PYRENE RAFs Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{1} = 0.91$	$\frac{0.18}{1} = 0.18$	$\frac{0.91}{1} = 0.91$	$\frac{0.91}{1} = 0.91$

The oral and dermal subchronic reference dose for benzo[a]pyrene is based on a naphthalene oral gavage study conducted in rats. In this study, an applied dose was used. The subchronic inhalation reference concentration for benzo[a]pyrene is based on a naphthalene inhalation study in humans. In this study, an applied dose was used.

BENZO[a]PYRENE RAFs Evaluation of Subchronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{1} = 0.91$	$\frac{0.18}{1} = 0.18$	$\frac{0.91}{1} = 0.91$	$\frac{0.91}{1} = 0.91$

BENZO[b]FLUORANTHENE CAS #: 205992

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.04 mg/kg/day (2b)
Chronic Oral Reference Dose: 0.04 mg/kg/day (2f)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: 7.3 $(mg/kg/day)^{-1}$ (1g)
Inhalation Cancer Unit Risk: Not Volatile

No specific quantitative information found on any route of exposure. Assume same as B[a]P.

The oral and dermal cancer potency value for benzo[b]fluoranthene is based on a dietary study in which B[a]P was administered to mice. In this study, an applied dose was used.

BENZO[b]FLUORANTHENE RAFs Evaluation of Carcinogenicity			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{0.91} = 1$	$\frac{0.18}{0.91} = 0.2$	$\frac{0.91}{0.91} = 1$	$\frac{0.91}{0.91} = 1$

The oral and dermal chronic reference dose for benzo[b]fluoranthene is based on a naphthalene oral gavage study conducted in rats. In this study, an applied dose was used.

BENZO[b]FLUORANTHENE RAFs Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{1} = 0.91$	$\frac{0.18}{1} = 0.18$	$\frac{0.91}{1} = 0.91$	$\frac{0.91}{1} = 0.91$

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.04 mg/kg/day (2b)
Chronic Oral Reference Dose: 0.04 mg/kg/day (2f)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: Not Volatile

No specific quantitative information found on any route of exposure. Assume same as B[a]P.

The oral and dermal chronic reference dose for benzo[g,h,i]perylene is based on a naphthalene oral gavage study conducted in rats. In this study, an applied dose was used.

BENZO[g,h,i]PERYLENE RAFs Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{1} = 0.91$	$\frac{0.18}{1} = 0.18$	$\frac{0.91}{1} = 0.91$	$\frac{0.91}{1} = 0.91$

The oral and dermal subchronic reference dose for benzo[g,h,i]perylene is based on a naphthalene oral gavage study conducted in rats. In this study, an applied dose was used.

BENZO[g,h,i]PERYLENE RAFs Evaluation of Subchronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{1} = 0.91$	$\frac{0.18}{1} = 0.18$	$\frac{0.91}{1} = 0.91$	$\frac{0.91}{1} = 0.91$

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.02 mg/kg/day (2)
Chronic Oral Reference Dose: 0.02 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: 0.014 $(mg/kg/day)^{-1}$ (1)
Inhalation Cancer Unit Risk: Not Volatile

^{14}C-DEHP appears to be efficiently absorbed from the gastrointestinal tract of the rat (Williams and Blanchfield, 1974). More than 90% of the radiolabel was excreted in urine. Fecal excretion was not quantified, suggesting oral absorption is probably close to 100%. A second study (Chadwick et al., 1982) demonstrated virtually complete absorption of ^{14}C-DEHP administered in the diet to F344 rats.

Chadwick, M., Branfman, A.K. and Silveira, D.M. (1982) Dose-Dependence of and Effect of Prior Exposure on the Metabolism of DEHP Administered in the Diet to Rats. Report to Chemical Manufacturers Association. Arthur D. Little, Inc.

Williams, D.T. and Blanchfield, B.J. (1974) *Retention, Excretion and Metabolism of DEHP Administered Orally to the Rat*. Bull. Environ. Contam. Toxicol. 11:371-387.

DEHP appears to be poorly absorbed through skin (Elsisi et al., 1985). Only 7% of an occluded applied dose of ^{14}C-DEHP was absorbed through shaved rat skin, as evidenced by the appearance of radiolabel in urine, feces and tissues. In a semi-occluded study (Elsisi et al., 1989), the shaved skin of F344 rats was exposed to ^{14}C-phthalates in ethanol and covered with a perforated plastic cap. Radioactivity was monitored in urine and feces as an index of excretion. Bis(2-ethylhexyl)phthalate was poorly absorbed with less than 2% of the applied dose recovered in biological material (98% recovered at the site of application). The value of 2% is selected as the most appropriate since the experimental protocol most closely represents the human exposure scenario.

Elsisi, A. E., Carter, D. E. and Sipes, I. G. (1975). *Dermal Absorption and Tissue Distribution of Phthalate Esters*. Toxicologist. 5:246.

Elsisi, E., Carter, D.E. and Sipes, I.G. (1989) *Dermal Absorption of Phthalate Diesters in Rats*. Fund. Appl. Toxicol. 12:70-77.

CADMIUM

CAS #: 7440439

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.001 mg/kg/day (2e)
Chronic Oral Reference Dose: 0.001 mg/kg/day (2d)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: Not Volatile

Human studies have measured the oral absorption efficiency of cadmium compounds with a reported range of 1% - 7% (McLellan et al., 1978; Shaikh and Smith, 1980). The range of reported oral absorption efficiencies in experimental animals is lower than in humans, from 0.5% - 3% (Engstrom and Nordberg, 1979; Moore et al., 1973; Friberg et al., 1974). Higher doses tend to be absorbed less efficiently as do doses administered in food or milk, when compared to aqueous doses. Iron deficiency has been observed to increase the oral absorption of cadmium in humans and animals (ATSDR, 1989). Therefore, the upper-bounds of 7% (humans) and 3% (animals) are selected as protective estimates.

Agency for Toxic Substances and Disease Registry (ATSDR) (1989) Toxicological Profile for Cadmium. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, pp. 46-49.

Engstrom, B. and Nordberg, G.F. (1979) *Dose-Dependence of Gastrointestinal Absorption and Biological Half-Time of Cadmium in Mice*. Toxicology 13:215-222.

Friberg, L., Piscator, M., Nordberg, G.F. and Kjellstrom, T. (1974) Cadmium in the Environment. 2nd ed. Boca Raton, FL: CRC Press.

Mclellan, J.S., Flanagan, P.R., Chamberlain, M.J. and Valberg, L.S. (1978) *Measurement of Dietary Cadmium Absorption in Humans*. J. Toxicol. Environ. Health 4:131-138.

Moore, W., Stara, J.F., Crocker, W.C., Malanchuk, M. and Ilitis, R. (1973) *Comparison of ^{115}Cd Retention in Rats Following Different Routes of Administration*. Environ. Res. 6:473-478.

Shaikh, Z.A. and Smith, J.C. (1980) *Metabolism of Orally Ingested Cadmium in Humans*. In: Holmstedt, B. et al., eds. Mechanisms of Toxicity and Hazard Evaluation. Amsterdam: Elsevier/North-Holland, pp. 569-574.

Cadmium is poorly absorbed by the dermal route (ATSDR, 1989). An upper-bound estimate of 1% is probably appropriate and protective for human exposure (see chromium).

Agency for Toxic Substances and Disease Registry (ATSDR) (1989) Toxicological Profile for Cadmium. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, pp. 46-49.

CADMIUM

The oral RfDs for cadmium of 1E-3 and 5E-4 mg/kg-day for food and water exposures, respectively, are based on a toxicokinetic model that evaluated a large quantity of both human and animal data. The RfDs are based on the highest level of cadmium in the human renal cortex not associated with significant proteinuria (i.e., the critical effect). To derive the RfD, it was assumed that 5% of cadmium was absorbed from water, while only 2.5% was absorbed from the diet. Although these RfDs account for matrix-specific differences in absorption, they are applicable to administered media-specific doses (U.S. EPA, 1992).

GZA evaluated the AAFs derived by the MADEP (1992) for noncancer effects and determined that they were not derived correctly. The MADEP only considered the RfD derived for dietary exposure and, thus, failed to consider that the U.S. EPA-derived RfDs already accounted for media-specific absorption differences. MADEP calculated an AAF for drinking water exposure, for use with the cadmium RfD intended for dietary exposure, assuming that absorption from the diet is the same as that from drinking water. This assumption is inconsistent with EPA's derivation of two RfDs. GZA derived AAFs for all pathways. However, the AAFs for oral exposure to soil, food, and water are numerically the same as those presented by MADEP. The difference is in their derivation, and in their intended use (e.g., oral-water AAF is used with RfD for water).

Derivation of Oral-Diet AAF

As discussed above, the oral RfD (food) for cadmium of 0.001 mg/kg-day is based on a toxicokinetic model with an assumed dietary absorption rate of 2.5%. Thus, no further adjustment is necessary when it is used for dietary exposures. Therefore, the AAF (oral-diet) is 1.0 (assumes equal absorption for basis of RfD, and for exposure). This AAF is only applicable when the AAF for food is used.

Derivation of Oral-Water AAF

As discussed above, the oral RfD (water) for cadmium of 0.0003 mg/kg-day is based on a toxicokinetic model with an assumed absorption rate of cadmium from water, of 5%. Thus, no further adjustment is necessary for drinking water exposures. Therefore, the AAF (oral-water) is 1.0 (assumes equal absorption for basis of RfD, and for exposure). This AAF is only applicable when the RfD for water is used.

Derivation of Oral-Soil AAF

GZA conservatively assumed that the absorption of cadmium from soil is equal to that from the diet. Thus the AAF (oral-soil) = 1.0. This AAF is for use with the RfD developed for dietary exposure.

Derivation of Dermal-Soil AAF

Dermal absorption of inorganics, especially from soil, is reported to be poor. Dermal absorption of cadmium is also expected to be poor, although no specific estimates were located. The MADEQE (1989) recommended a default dermal absorption (absolute) value of from 0.1% to 1% for inorganics. Thus, GZA calculated a dermal-soil relative absorption adjustment factor (AAF) of 0.1%/2.5% = 0.04 for the low end of the range and an AAF of 1%/2.5% = 0.4 for the high end of the recommended absorption range. To be conservative, GZA used the AAF (dermal-soil) of 0.4 in this risk assessment. This AAF is intended for use with the oral RfD for cadmium derived for food (1E-3 mg/kg-day).

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral RfD for cadmium is applicable to an administered dose, as discussed above. Therefore, an adjustment is necessary to convert the RfD to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (multiplying the RfD by the absorption). For cadmium, the AAF (dermal-water) is 1/(2.5%) = 40. This AAF is for use with the RfD derived for the diet. If the oral RfD derived for water is used, then the AAF (dermal-water) is 1/(5%) = 20. Care must be taken to ensure that the appropriate AAF is used with the corresponding RfD.

REFERENCES

MADEQE (Massachusetts Department of Environmental Quality Engineering). 1989. Guidance for Disposal Site Risk Characterization and Related Phase II Activities - In Support of the Massachusetts Contingency Plan. MADEQE Office of Research and Standards.

U.S. EPA. 1992. Integrated Risk Information System (IRIS).

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CARBON TETRACHLORIDE CAS #: 56235

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.007 mg/kg/day (2)
Chronic Oral Reference Dose: 0.0007 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: 430 $\mu g/m^3$ (3a)
Chronic Inhalation Reference Concentration: 430 $\mu g/m^3$ (3)

Oral Cancer Potency Factor: 0.13 $(mg/kg/day)^{-1}$ (1)
Inhalation Cancer Unit Risk: 0.000015 $(\mu g/m^3)^{-1}$ (1)

The oral absorption efficiency in animals is extensive. Studies have reported that 80-85% of an administered dose is recovered in expired air (Marchand et al., 1970; Paul and Rubinstein, 1963). This indicates that GI absorption is probably close to 100% since CCl_4 is metabolized with metabolites appearing in urine and feces.

Marchand, C., McLean, S. and Plaa, G.L. (1970) *The Effect of SKF525A on the Distribution of Carbon Tetrachloride in Rats*. J. Pharmacol. Exp. Therap. 714:232-238.

Paul, B.B. and Rubenstein, D. (1963) *Metabolism of Carbon Tetrachloride and Chloroform by the Rat*. J. Pharmacol. Exp. Therap. 141:141-148.

No studies were located containing quantitative information on the dermal absorption efficiency of carbon tetrachloride. Assume that the dermal absorption is similar to that of other volatile organics such as benzene whose dermal absorption efficiency has been estimated to be 10% of a non-occluded applied dose in 24 hours.

The oral and dermal cancer potency value for carbon tetrachloride is based on a gavage study in rodents. This toxicity value is based on applied dose.

CARBON TETRACHLORIDE RAFs Evaluation of Carcinogenicity			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{1}{1} = 1$	$\frac{0.1}{1} = 0.1$	$\frac{1}{1} = 1$	$\frac{1}{1} = 1$

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.2 mg/kg/day (2)
Chronic Oral Reference Dose: 0.02 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: 200 $\mu g/m^3$ (2)
Chronic Inhalation Reference Concentration: 20 $\mu g/m^3$ (2)

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: NC

The oral absorption efficiency of chlorobenzene has been reported to range from at least 18% in rats to at least 31% in humans (Lindsay-Smith et al., 1972; Ogata and Shimada, 1983). These estimates are probably low since the studies failed to quantitate exhaled compounds. A more conservative estimate may be 100%, based on its structural similarity to benzene.

Lindsay-Smith, J.R., Shaw, B.A.J. and Foulkes, D.M. (1972) *Mechanisms of Mammalian Hydroxylation: Some Novel Metabolites of Chlorobenzene*. Xenobiotica 2:215-226.

Ogata, M. and Shimada, Y. (1983) *Differences in Urinary Monochlorobenzene Metabolites Between Rats and Humans*. Int. Arch. Occup. Environ. Health 53:51-57.

No studies were located regarding the dermal absorption of chlorobenzene. Assume it to be similar to benzene (10% non-occluded, 24 hours).

The oral and dermal chronic reference dose for chlorobenzene is based on an oral (capsule) study in dogs. In this study, an applied dose was used.

CHLOROBENZENE Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{1}{1} = 1$	$\frac{0.1}{1} = 0.1$	$\frac{1}{1} = 1$	$\frac{1}{1} = 1$

CHLOROBENZENE

The MADEP (1992) assumed 100% gastrointestinal absorption of VOCs when developing AAFs. No consideration was given to possible matrix attenuation affects when the VOC was administered in the diet versus drinking water. GZA, therefore, assumed 100% gastrointestinal absorption of chlorobenzene in the dose-response study wherein it was administered to dogs in capsule form.

Derivation of Oral-Soil, Oral-Water, and Oral-Diet AAFs

The MADEP (1992) typically assumed 100% gastrointestinal absorption for the VOCs, when specific data were not available. The oral RfD for chlorobenzene is based on an oral study, thus, GZA assumed 100% absorption in this study. It was further assumed that the absorption was 100%, when administered in soil, water and the diet. Therefore, the AAF (oral-soil), the AAF (oral-water), and the AAF (oral-diet) are all equal to 100%/100% = 1.

Derivation of Dermal-Soil AAFs

The MADEQE (1989) recommended a default range of absorption estimates for VOCs in soil of 10% to 25%. GZA has conservatively assumed that the dermal absorption of chlorobenzene from soil is 25%. Thus, the AAF (dermal-soil) for this chemical equals (the absorption from soil)/(absorption in the dose-response study) which equals 25%/100% = 0.25.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral RfD for chlorobenzene is based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the RfD to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (multiplying the RfD by the percent absorption). For chlorobenzene the AAF (dermal-water) is 1/(100%) = 1.0.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

MADEQE (Massachusetts Department of Environmental Quality Engineering). 1989. Guidance for Disposal Site Risk Characterization and Related Phase II Activities - In Support of the Massachusetts Contingency Plan. MADEQE Office of Research and Standards.

U.S. EPA. 1992. Integrated Risk Information System (IRIS).

CHLOROFORM CAS#: 67663

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.01 mg/kg/day (2)
Chronic Oral Reference Dose: 0.01 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: 660 $\mu g/m^3$ (3a)
Chronic Inhalation Reference Concentration: 660 $\mu g/m^3$ (3)

Oral Cancer Potency Factor: 0.0061 $(mg/kg/day)^{-1}$ (1)
Inhalation Cancer Unit Risk: 0.000023 $(\mu g/m^3)^{-1}$ (1)

Oral absorption efficiency of chloroform in humans and animals is essentially 100% (Fry et al., 1972; Brown et al., 1974; Taylor et al., 1974).

Brown, D.M., Langley, P.F., Smith, D. and Taylor, D.C. (1974) *Metabolism of Chloroform. I. The Metabolism of ^{14}C-Chloroform by Different Species.* **Xenobiotica.** 4:151-163.

Fry, B.J., Taylor, T. and Hathaway, D.E. (1972) *Pulmonary Elimination of Chloroform and its Metabolites in Man.* **Arch. Int. Pharmacodyn.** 196:98-11.

Taylor, D.C., Brown, D.M., Kuble, R. and Langley, P.F. (1974) *Metabolism of Chloroform. II. A Sex Difference in the Metabolism of ^{14}C-Chloroform in Mice.* **Xenobiotica.** 4:165-174.

Dermal absorption of chloroform is rapid and quite large (329 umol/min/cm^2) across mouse skin (Tsurata, 1975). This suggests that an occluded dose would be 100% absorbed with a non-occluded dose being absorbed less efficiently (perhaps 10% as with benzene) due to rapid volatilization.

Tsurata, H. (1975) *Percutaneous Absorption of Organic Solvents. I. Comparative Study of the in Vivo Percutaneous Absorption of Chlorinated Solvents in Mice.* **Ind. Health.** 13:227-236.

CHLOROFORM

The MADEP (1992) assumed 100% gastrointestinal absorption of chloroform when developing AAFs. The MADEP, has not, however, derived dermal-water AAFs for this compound. Thus, GZA has derived a dermal-water AAF for chloroform, assuming 100% gastrointestinal absorption of chloroform in the oral drinking water study upon which the oral/dermal dose-response value is based.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral CSF for chloroform is based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the CSF to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (dividing the CSF by the percent absorption). For chloroform, the AAF (dermal-water) is 1/(100%) = 1.0.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

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CHRYSENE

CAS #: 218019

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.04 mg/kg/day (2b)
Chronic Oral Reference Dose: 0.04 mg/kg/day (2f)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: 7.3 $(mg/kg/day)^{-1}$ (1g)
Inhalation Cancer Unit Risk: Not Volatile

No specific quantitative information found on the absorption via the oral or dermal route. Assume same as B[a]P.

The oral and dermal cancer potency value for chrysene is based on a dietary study in which B[a]P was administered to mice. In this study, an applied dose was used.

CHRYSENE RAFs Evaluation of Carcinogenicity			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{0.91} = 1$	$\frac{0.18}{0.91} = 0.2$	$\frac{0.91}{0.91} = 1$	$\frac{0.91}{0.91} = 1$

The oral and dermal chronic reference dose for chrysene is based on a naphthalene oral gavage study conducted in rats. In this study, an applied dose was used.

CHRYSENE RAFs Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{1} = 0.91$	$\frac{0.18}{1} = 0.18$	$\frac{0.91}{1} = 0.91$	$\frac{0.91}{1} = 0.91$

1,1-DICHLOROETHANE CAS #: 75343

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 1 mg/kg/day (2)
Chronic Oral Reference Dose: 0.1 mg/kg/day (2)

Subchronic Inhalation Reference Concentration: 5000 $\mu g/m^3$ (2)
Chronic Inhalation Reference Concentration: 500 $\mu g/m^3$ (2)

Oral Cancer Potency Factor: Not Quantified
Inhalation Cancer Unit Risk: Not Quantified

No specific studies were located quantifying the respiratory absorption efficiency of 1,1-dichloroethane. The blood/air partitioning coefficient of 1,1-dichloroethane is approximately 4 times less than that of 1,2-dichloroethane suggesting less efficient pulmonary absorption of 1,1-dichloroethane than 1,2-dichloroethane. However, assume the 75% inhalation efficiency of 1,2-dichloroethylene is applicable.

No specific studies were located quantifying the oral absorption efficiency of 1,1-dichloroethane. Assume it to be the same as 1,2-dichloroethane (100%).

No specific studies were located quantifying the dermal absorption efficiency of 1,1-dichloroethane. Assume it to be similar to that of other volatile compounds (benzene) whose dermal absorption efficiency may reach 10% of a non-occluded applied dose in 24 hours.

The oral and dermal chronic reference dose for 1,1-dichloroethane is based on an inhalation study conducted in rats. In this study, an applied dose was used.

1,1-DCA RAFs Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{1}{0.75} = 1.3$	$\frac{0.1}{0.75} = 0.13$	$\frac{1}{0.75} = 1.3$	$\frac{1}{0.75} = 1.3$

1,1-DICHLOROETHANE

The MADEP (1992) derived AAFs for ingestion of soil, water and food, and for dermal exposure to soil. The MADEP, has not however, derived dermal-water AAFs for this compound. Thus, GZA has derived a dermal-water AAF for this chemical. MADEP assumed 75% absorption in the inhalation dose-response study used to derive the chronic oral RfD of 1.0E-01 mg/kg-day. GZA assumed the same absorption when deriving the dermal-water AAF.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral CSF for 1,1-dichloroethane is based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the CSF to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (dividing the CSF by the percent absorption). For 1,1-dichloroethane, the AAF (dermal-water) is 1/(75%) = 1.33.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

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1,1-DICHLOROETHYLENE CAS #: 75354

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.009 mg/kg/day (2)
Chronic Oral Reference Dose: 0.009 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: 5 $\mu g/m^3$ (3a)
Chronic Inhalation Reference Concentration: 5 $\mu g/m^3$ (3)

Oral Cancer Potency Factor: 0.6 $(mg/kg/day)^{-1}$ (1)
Inhalation Cancer Unit Risk: 0.00005 $(\mu g/m^3)^{-1}$ (1)

Since 1,1-dichloroethylene is a small organic molecule with chemical and physical properties similar to the lipid soluble anaesthetics, it is expected to penetrate the pulmonary epithelium rapidly and efficiently. An inhalation absorption efficiency of 98% is derived from metabolic excretion data of inhaled radiolabeled 1,1-dichloroethylene data in the rat (McKenna et al., 1977).

McKenna, M.J., Watanabe, P.G. and Gehring, P.J. (1977) *Pharmacokinetics of Vinylidene Chloride in the Rat.* Environ. Health Perspect. 21:99-105.

The oral absorption of 1,1-dichloroethylene in animals has been demonstrated to be rapid and essentially complete (100%). (Reichert et al., 1979; Jones and Hathaway, 1978; McKenna et al., 1978; Putcha et al., 1986).

McKenna, M.J., Zemple, J.A., Madrid, E.O., Braun, W.H. and Gehring, P.J. (1978) *Metabolism and Pharmacokinetic Profile of Vinylidene Chloride in Rats Following Oral Administration.* Toxicol. Appl. Pharmacol. 45:821-835.

Reichert, D., Werner, H.E. and Metzler, M. (1979) *Molecular Mechanism of 1,1-Dichloroethylene Toxicity: Excreted Metabolites Reveal Different Pathways of Reactive Intermediates.* Arch. Toxicol. 42:159-169.

Jones, P.K. and Hathaway, D.E. (1978) *Differences in Metabolism of Vinylidene Chloride Between Mice and Rats.* Br. J. Cancer. 37:411-417.

Putcha, L., Bruchner, J.V. and D'Souza, R. (1986) *Toxicokinetics and Bioavailability of Oral and Intravenous 1,1-Dichloroethylene.* Fund. Appl. Toxicol. 6:240-250.

No studies were located regarding the dermal absorption of 1,1-dichloroethylene. Due to its chemical and physical properties, dermal absorption is expected to be 100% of an occluded applied dose or 10% of a non-occluded applied dose (same as benzene).

1,1-DICHLOROETHYLENE

The MADEP (1992) derived AAFs for ingestion exposure to soil, water and food and for dermal exposure to soil, assuming 100% gastrointestinal absorption of 1,1-dichloroethylene in the oral drinking water study upon which the chronic oral RfD of 9.0E-03 mg/kg-day was based, and 98% absorption in the mouse inhalation study upon which the oral CSF of 6.0E-1 $(mg/kg\text{-}day)^{-1}$ was based. MADEP has not derived dermal-water AAFs for this compound. GZA, therefore, derived dermal-water AAFs for use with the oral RfD and CSF, using the same absorption assumptions as those used by the MADEP.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral RfD and CSF for 1,1-dichloroethene are based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the RfD and CSF to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (multiplying the RfD or dividing the CSF by the percent absorption). For 1,1-dichloroethene, the AAF (dermal-water) is 1/(100%) = 1.0 for use with the RfD, and 1/98% = 1.02 for use with the CSF.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

U.S. EPA. 1992. Integrated Risk Information System (IRIS).

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1,2-DICHLOROETHYLENE CAS #: 156605

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.2 mg/kg/day (2)
Chronic Oral Reference Dose: 0.02 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: 1100 $\mu g/m^3$ (3a)
Chronic Inhalation Reference Concentration: 1100 $\mu g/m^3$ (3)

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: NC

No studies were located addressing the oral or dermal absorption efficiency of 1,2-dichloroethylene. Assume behavior similar to 1,1-dichloroethylene and benzene (100% oral and 10% non-occluded dermal absorption efficiencies).

The oral and dermal chronic reference dose for 1,2-dichloroethylene is based on an oral drinking water study conducted in mice. In this study, an applied dose was used.

1,2-DCE RAFs Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{1}{1} = 1$	$\frac{0.1}{1} = 0.1$	$\frac{1}{1} = 1$	$\frac{1}{1} = 1$

The oral and dermal subchronic reference dose for 1,2-dichloroethylene is based on an oral drinking water study conducted in mice. In this study, an applied dose was used.

1,2-DCE RAFs Evaluation of Subchronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{1}{1} = 1$	$\frac{0.1}{1} = 0.1$	$\frac{1}{1} = 1$	$\frac{1}{1} = 1$

ETHYLBENZENE

CAS #: 100414

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 1 mg/kg/day (2)
Chronic Oral Reference Dose: 0.1 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: 1000 $\mu g/m^3$ (2)
Chronic Inhalation Reference Concentration: 1000 $\mu g/m^3$ (1)

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: NC

Animal studies indicate that ethylbenzene is quickly and efficiently absorbed by the oral route. Estimates range from 72%-92% in one study (El Masry et al., 1956) to 84% in a second study (Climie et al., 1983). A value of 100% is selected since these studies underestimated oral absorption by not controlling for respiratory volatilization following oral absorption.

Climie, I.J.G., Hutson, D.H. and Stoydin, G. (1983) *The Metabolism of Ethylbenzene Hydroperoxide in the Rat.* **Xenobiotica** 13:611-618.

El Masry, A.M., Smith, J.N. and Williams, R.T. (1956) *The Metabolism of Alkylbenzenes: n-Propylbenzene and n-Butylbenzene with Further Observations on Ethylbenzene.* **Biochem. J.** 64:50-56.

Absorption of pure liquid ethylbenzene and aqueous solutions containing ethylbenzene through human skin is rapid and substantial (20-30 $mg/cm^2/hr$) (Gromiec and Piotrowski, 1984). Occluded doses could potentially be 100% absorbed. The non-occluded dermal absorption of ethylbenzene has been measured to be 3.4% of an applied dose in 4 hours (Susten et al., 1990). This calculates to a 24 hour dermal absorption efficiency of 20%.

Susten, A.S., Niemeier, R.W. and Simon, S.D. (1990) *In Vivo Percutaneous Absorption Studies of Volatile Organic Solvents in Hairless Mice II. Toluene, Ethylbenzene and Aniline.* **J. Appl. Toxicol.** 10:217-225.

Gromiec, J.P. and Piotrowski, J.K. (1984). *Urinary Mandelic Acid as an Exposure Test for Ethylbenzene.* **Int. Arch. Occup. Environ. Health** 55:61-72.

ETHYLBENZENE

The MADEP (1992) assumed 100% gastrointestinal absorption of ethylbenzene when developing AAFs. GZA, therefore, assumed 100% gastrointestinal absorption of ethylbenzene in the dose-response study. The MADEP derived AAFs for ethylbenzene for the oral route, but not for the dermal-water AAFs exposure route. Thus, GZA has derived dermal-water AAFs for ethylbenzene.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral RfD for ethylbenzene is based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the RfD to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (multiplying the RfD by the percent absorption). For ethylbenzene, the AAF (dermal-water) is 1/(100%) = 1.0.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

U.S. EPA. 1992. Integrated Risk Information System (IRIS).

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TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.00075 mg/kg/day (4)
Chronic Oral Reference Dose: 0.00075 mg/kg/day (4)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: Not Quantified
Inhalation Cancer Unit Risk: Not Volatile

The oral absorption efficiency of lead compounds in adult experimental animals and adult humans ranges from 1% - 15% (EPA, 1986; Hammond, 1982; Chamberlain et al., 1978). Young humans and experimental animals absorb lead with higher efficiency, estimates ranging up to 50% (Hammond, 1982; Kostial et al., 1971, 1978; Forbes and Reina, 1972). The estimate of 50% is considered as being a conservative upper-bound for humans and experimental animals.

Chamberlain, A., Hard, C. and Little, M.J. (1978) Investigations into Lead from Motor Vehicles. Harwell, U.K.: United Kingdom Atomic Energy Authority. Rep. No. AERE-9198.

U.S. Environmental Protection Agency (EPA) (1986) Air Quality Criteria for Lead. June 1986 and Addendum, September, 1986. Research Triangle Park, NC: Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, EPA. EPA 600/8-83-018F.

Forbes, G.B. and Reina, J.C. (1972) *Effect of Age on Gastrointestinal Absorption (Fe, Sr, Pb) in the Rat*. J. Nutr. 102:647-652.

Hammond, P.B. (1982) *Metabolism of Lead*. In: Chisolm, J.J. and O'Hara, D.M., eds. Lead Absorption in Children: Management, Clinical and Environmental Aspects. Baltimore, MD: Urban and Schwarzenberg, pp. 11-20.

Kostial, K., Simonovic, J. and Pisonic, M. (1971) *Lead Absorption from the Intestine in Newborn Rats*. Nature 233:564-567.

The dermal absorption efficiency for lead as lead acetate has been reported to be 0.3% (12 hours) of an applied dose in humans, or 0.6% in 24 hours (Moore et al., 1980). Organic lead compounds are absorbed more rapidly and extensively than inorganic lead compounds.

Moore, M.R., Meredith, P.A., Watson, W.S., Sumner, D.J., Taylor, M.K. and Goldberg, A. (1980) *The Percutaneous Absorption of Lead-203 in Humans from Cosmetic Preparations Containing Lead Acetate, As Assessed by Whole-Body Counting and Other Techniques*. Food. Cosmet. Toxicol. 18:399-405.

LEAD

The oral dose-response value for lead of 7.5E-4 mg/kg-day was calculated from a Treatment Technology Action Level (TTAL) of 15 ppb set for drinking water at the tap. The gastrointestinal absorption of lead ranges from 8% (Hammond, 1992) to 15% (Chamberlain, et al., 1978) for adults. For children, absorption of lead from the diet has been reported to be higher, with estimates ranging from 40 to 50% (ATSDR, 1989). When calculating the dose-response value, the MADEP assumed a 50% absorption of lead from drinking water. Thus, this value is based on an absorbed dose.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral dose-response value for lead derived by the MADEP is also based on an absorbed dose. Therefore, this dose-response value can be used with the absorbed dose from dermal exposure to water with no further adjustment. Thus, an AAF of 1 was used, which results in no adjustment to the dose or to the dose-response value.

REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 1989. Toxicological Profile for Lead. ATSDR, Centers for Disease Control, Atlanta, GA.

Chamberlain, A.C., M.J. Heard, P. Little, D. Newton, A.C. Wells and R.D. Wiffen. 1978. Investigations into lead from motor vehicles. United Kingdom Atomic Energy Authority, Harwell, United Kingdom. Report No. AERE-R9198. As cited in ATSDR, 1989.

Hammond, B. 1982. Metabolism of Lead. In: J.J. Chisolm and D.M. O'Hara (eds). Lead Absorption in Children: Management, Clinical and Environmental Aspects. Urban and Schwarzenberg. Baltimore, MD. As cited in ATSDR, 1989.

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MERCURY CAS #: 7439976

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.0003 mg/kg/day (2)
Chronic Oral Reference Dose: 0.0003 mg/kg/day (2)

Subchronic Inhalation Reference Concentration: 0.3 $\mu g/m^3$ (2)
Chronic Inhalation Reference Concentration: 0.3 $\mu g/m^3$ (2)

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: NC

MERCURY (elemental)

Oral absorption of metallic mercury has been estimated to be, at most, 0.10% (Friberg and Nordberg, 1973). The oral absorption efficiency of 0.01% in humans and laboratory animals is most frequently cited in the literature (Owen, 1990). The value of 0.1% is suggested as a conservative upper limit for human exposure.

Friberg, L., Nordberg, F. (1973) *Inorganic Mercury - A Toxicological and Epidemiological Appraisal*. In: Miller, M.W., Clarkson, T.W., ed. Mercury, Mercurials and Mercaptans. Springfield, Illinois: Charles C. Thomas, pp. 5-22.

Owen, B.A. (1990) *Literature-Derived Absorption Coefficients for 39 Chemicals Via Oral and Inhalation Routes of Exposure*. Regul. Toxicol. Pharmacol. 11:237-252.

Hursh et al. (1989) report maximum systemic mercury as a fraction of initial amounts of mercury on the skin. The average percentage absorbed (from data obtained from 5 human volunteers) can be calculated as 40% of the free mercury deposited on the skin. Assuming that 10% of the mercury in soil could be extracted and available for dermal absorption (Landa, 1978), a dermal absorption factor of 0.04 is obtained.

Hursh, J.B., Clarkson, T.W., Miles, E.F., et al. (1989) *Percutaneous Absorption of Mercury Vapor by Man*. Arch. Environ. Health 44:120-127.

Landa, E.R. (1978) *The Retention of Metallic Mercury Vapor by Soils*. Geochem. Cosmochim. Acta. 42:1407-1411.

MERCURY

The oral RfD for inorganic mercury of 3E-4 mg/kg-day is based on three subchronic rat studies in which rats were dosed with mercuric chloride by either gavage or by subcutaneous injection. The U.S. EPA converted the subcutaneously injected dose to an oral dose by assuming 7% gastrointestinal absorption of mercuric chloride. The assumption of a 7% gastrointestinal absorption was based on a paper by Rahola et al. (1973). A later publication (Miettinen, 1973) by one of the authors of the study indicates that the gastrointestinal absorption in this study was 15%, not 7%. The MADEP (1992) also recommends a conservative estimate of gastrointestinal absorption of inorganic mercury of 15%. Thus, this value was assumed for the dose-response studies. The RfD is based on an administered dose of mercuric chloride in aqueous solution via gavage.

This RfD and accompanying AAFs are intended for use with water, soils and sediments containing unspeciated mercury, or in situations where speciation indicates the presence of inorganic mercury. This RfD and accompanying AAFs should not be used for risk assessments in which exposure to mercury taken up by fish is a concern. In such a case, the RfD for methyl mercury should be used and AAFs specific for methyl mercury should be derived.

Derivation of Oral-Water and Oral-Diet AAFs

The RfD for inorganic mercury is based on ingestion of aqueous solutions of mercuric chloride. GZA assumed that the gastrointestinal absorption of mercury from water and the diet is the same. Thus, the oral-water and oral-diet AAFs are both 1.

Derivation of Oral-Soil AAF

The MADEP (1992) estimated that ingestion of mercury in soil is absorbed at about one half the extent of mercury in water. Thus, GZA calculated an AAF (oral-soil) of 0.5.

Derivation of Dermal-Soil AAF

Absorption data specific to inorganic mercury are presented in the text, however. These data were used below to derive AAFs for inorganic mercury. The MADEP estimated that 6.5% of a dermal dose of inorganic mercury adsorbed to soil would be absorbed. MADEP also estimated that up to 15% of an oral dose of inorganic mercury could be absorbed from the gastrointestinal tract. From this estimate, GZA derived an AAF (dermal-soil) of 6.5%/15% = 0.43.

Derivation of Dermal-Water AAF

The methodology for estimating risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this process is an absorbed dose. The oral RfD is based on administered dose. In order to use

consistent dose-response criteria across all exposure pathways, the adjustment was made to the absorbed dermal dose in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to multiplying the RfD by the percent absorption in the applicable dose-response study. Using an assumed gastrointestinal absorption of 15% in the dose-response study, GZA calculated an AAF (dermal-water) of 1/15% = 6.7 for use with the oral RfD.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation of the Risk Assessment ShortForm-Residential Scenario.

Meittinen, J.K. 1973. Absorption and Elimination of Dietary Mercury (Hg^{2+}) and Methylmercury in Man. In: Miller, M.W. and T.W. Clarkson (eds). Mercury, Mercurials and Mercaptens. C.C. Thomas, Springfield.

Rahola, T., T. Hattula, T. Korolainen and J.K. Meittinen. 1973. Elimination of free and protein-bound ionic mercury 203 Hg^{2+} in man. *Annals of Clinical Research*, 5:214-219.

U.S. EPA. 1992. Integrated Risk Information System (IRIS).

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MERCURY (organic)

The oral absorption efficiency of methylmercury is reported as 95% (Aberg et al., 1969; Owen, 1990). Other forms of organic mercury may be orally absorbed less efficiently, with estimates ranging down to 80% (Fitzhugh et al., 1950).

Owen, B. A. (1990) *Literature-Derived Absorption Coefficients for 39 Chemicals Via Oral and Inhalation Routes of Exposure*. **Regul. Toxicol. Pharmacol.** 11:237-252.

Aberg, B., Ekman, R., Falk, U., et al. (1969) *Metabolism of Methylmercury (^{203}Hg) Compounds in Man: Excretion and Distribution*. **Arch. Environ. Health** 19:478-484.

Fitzhugh, O.G., Nelson, A.A., Laug, E.P., et al. (1950) *Chronic Oral Toxicities of Mercuri-phenyl and Mercuric Salts*. **Arch. Ind. Hyg. Occup. Med.** 2:433-442

Methyl mercury (in water) applied to the skin of guinea pigs at two dose levels resulted in 3.4% and 4.5% of the applied dose being absorbed (Skog and Wahlberg, 1964). Given the lipophilicity of organomercurials, an absorption value of 4.5% was chosen.

Skog, E. and Wahlberg, J.E. (1964) *A Comparative Investigation of the Percutaneous Absorption of Metal Compounds in the Guinea Pig by Means of the Radioactive Isotopes: ^{51}Cr, ^{58}Co, ^{65}Zn, ^{110m}Ag, ^{115m}Cd, ^{203}Hg*. **J. Invest. Dermatol.** 43:187-192.

METHYL ETHYL KETONE

CAS #: 78933

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.5 mg/kg/day (2)
Chronic Oral Reference Dose: 0.05 mg/kg/day (2)

Subchronic Inhalation Reference Concentration: 3000 $\mu g/m^3$ (2)
Chronic Inhalation Reference Concentration: 1000 $\mu g/m^3$ (1)

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: NC

No information was located to quantitatively determine the oral or dermal absorption efficiency of methyl ethyl ketone. Assume a 100% absorption efficiency by the oral route and a 10% dermal absorption efficiency (non-occluded, 24 hour).

The oral and dermal chronic reference dose for methyl ethyl ketone is based on an inhalation study conducted in rats. An absorption efficiency of 50% was used to calculate an absorbed dose. The RAFs to be used for all exposure pathways are the absorption efficiencies of methyl ethyl ketone by the route in question.

METHYL ETHYL KETONE RAFs Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
1	0.1	1	1

2-BUTANONE
(A.K.A. METHYL ETHYL KETONE)

The MADEP (1992) has derived AAFs for 2-butanone (MEK) for the oral route and for dermal exposure to soil, but has not derived dermal-water AAFs for this compound. Thus, GZA has derived dermal-water AAFs for MEK. The MADEP (1992) assumed 100% gastrointestinal absorption of MEK when developing other AAFs. GZA, therefore, assumed 100% gastrointestinal absorption of MEK in the dose-response study upon which the chronic RfD of 5.0E-2 mg/kg-day was based.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral RfD for MEK is based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the RfD to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (multiplying the RfD by the percent absorption). For MEK, the AAF (dermal-water) is 1/(100%) = 1.0.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

U.S. EPA. 1992. Integrated Risk Information System (IRIS).

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NAPHTHALENE and 2-METHYLNAPHTHALENE

CAS #: 91203
CAS #: 91576

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.04 mg/kg/day (2)
Chronic Oral Reference Dose: 0.04 mg/kg/day (2)

Subchronic Inhalation Reference Concentration: 71 $\mu g/m^3$ (3a)
Chronic Inhalation Reference Concentration: 71 $\mu g/m^3$ (3b)

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: NC

Oral absorption, based on fecal recovery of metabolites, has been demonstrated to be essentially 100% in the rat (Chang, 1943).

Chang, L H. (1943) *The Fecal Excretion of Polycyclic Hydrocarbons Following Their Administration to the Rat.* J. Biol. Chem. 151:93-102.

No information exists quantifying the dermal absorption efficiency of naphthalene. However, toxicity has been documented following exposure by this suggesting absorption has occurred. Assume 10% to represent the dermal absorption efficiency (same as non-occlude, 24 hour benzene value).

The oral and dermal chronic reference dose for naphthalene and 2-methylnaphthalene is based on an oral gavage study conducted in rats. In this study, an applied dose was used.

NAPHTHALENE & 2-METHYLNAPHTHALENE RAFs Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{1}{1} = 1$	$\frac{0.1}{1} = 0.1$	$\frac{1}{1} = 1$	$\frac{1}{1} = 1$

NICKEL

CAS #: 7440020

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.02 mg/kg/day (2)
Chronic Oral Reference Dose: 0.02 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: Not Volatile

Human and animal studies indicate that the oral absorption efficiency of nickel from food or water ranges from 1% - 10% (Christensen and Lagesson, 1981; Schroeder et al., 1974; Tedeschi and Sunderman, 1956; Ambrose et al., 1976; Nielsen et al., 1986; Ho and Furst, 1973). The upper-bound of 10% is selected as a protective estimate of the oral absorption efficiency of nickel.

Christensen, O.B. and Lagesson, V. (1981) *Nickel Concentrations of Blood and Urine After Oral Administration.* Ann. Clin. Lab. Sci. 11:119-125.

Schroeder, H.A., Mitchener, M. and Nason, A.P. (1974) *Life-Term Effects of Nickel in Rats: Survival, Tumors, Interactions with Trace Elements and Tissue Levels.* J. Nutr. 104:239-243.

Tedeschi, R.E. and Sunderman, F.W. (1957) *Nickel Poisoning V. The Metabolism of Nickel Under Normal Conditions and After Exposure to Nickel Carbonyl.* Arch. Ind. Health 16:486-488.

Ambrose, A.M., Larson, P.S., Borzelleca, J.R. and Hennigar, G.R. (1976) *Long Term Toxicologic Assessment of Nickel in Rats and Dogs.* J. Food. Sci. Technol. 13:181-187.

Nielsen, G.D., Andersen, O., Jensen, M. and Grandjean, P. (1986) *Gastrointestinal Nickel Absorption. A New Experimental Model Using the Gamma-Emitting Isotope ^{57}Ni.* In: The Sixth UOEH Int. Symp., 3rd COMTOX on Bio- and Toxicokinetics of Metals, Kitakyushu City, Japan, Int. Conf. Clin. Chem., Chem. Toxicol., July 27-31.

Ho., W. and Furst, A. (1973) *Nickel Excretion by Rats Following a Single Treatment.* Proc. West. Pharmacol. Soc. 16:245-248.

Aqueous solutions of various forms of nickel can penetrate occluded human skin with absorption efficiencies ranging from 55% - 77%, most absorption occurring in the first 24 hours (Norgaard, 1955). It is unclear whether the nickel was absorbed into the deep layers of the skin or into the bloodstream. Studies in guinea pigs (Lloyd, 1980) demonstrated that much of the nickel absorbed remained in the skin, primarily in the jighly keratinized areas, while approximately 0.005 - 0.51 % of the applied nickel chloride was recovered from the blood and urine. A more recent study on excised human skin (Fullerton et al., 1986) indicated that 3.5% of an applied dose of nickel chloride

NICKEL

The oral RfD value for nickel of 2E-2 mg/kg-day is based on a dietary study of nickel sulfate in rats. The RfD is based on administered dose. Human and animal studies indicate that the oral absorption of nickel from the diet or water ranges from 1% to 10% (Christiansen and Lagesson, 1981; Schroeder et al., 1974; Tedeschi and Sunderman, 1956; Ambrose et al., 1976; Ho and Furst, 1973; MADEP, 1992). MADEP chose the upperbound estimate of 10% as a protective estimate of the oral absorption efficiency of nickel. This is a conservative estimate of absorption of nickel from the diet, which is likely to be less than that from drinking water. However, in order to be consistent with MADEP, GZA assumed that the absorption of nickel from the diet in the dose-response study was 10%.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral RfD for nickel is based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the RfD to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (multiplying the RfD by the percent absorption). For nickel, the AAF (dermal-water) is 1/(10%) = 10.

REFERENCES

Ambrose, A.M., P.S. Larson, J.R. Borzelleca and G.R. Hennigar. 1976. Long term toxicological assessment of nickel in rats and dogs. *Journal of Food Science and Technology*, 13:181-187.

Christiansen, O.B. and V. Lagesson. 1981. Nickel concentrations of blood and urine after oral administration. *Annals of Clinical Laboratory Science*, 11:119-125.

Ho, W. and A. Furst. 1973. Nickel excretion by rats following a single treatment. *Proc. West Pharmacol. Soc.* 16:245-248.

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

Nielson, G.D., O. Anderson, M. Jensen and P. Grandjean. 1986. Gastrointestinal nickel absorption. A new experimental model using the gamma-emitting isotope ^{57}Ni. In: The Sixth UOEH Int. Symp., 3rd COMTOX on Bio- and Toxicokinetics of Metals. Kitakyushu City, Japan,

Int. Conf. Clin. Chem., Chem. Toxicol., July 27-31.

Schroeder, H.A., M. Mitchener, and A.P. Nason. 1974. Life-term effects of nickel in rats: Survival, tumors, interactions with trace elements and tissue levels. *Journal of Nutrition*, 104:239-243.

Tedeschi, R.E. and F.W. Sunderman. 1957. Nickel poisoning V. The metabolism of nickel under normal conditions and after exposure to nickel carbonyl. *Archives of Industrial Health*, 16:486-488.

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PHENANTHRENE CAS #: 85018

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.04 mg/kg/day (2b)
Chronic Oral Reference Dose: 0.04 mg/kg/day (2f)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: NC

No specific quantitative information found on the absorption via the oral or dermal route. Assume same as B[a]P.

The oral and dermal chronic reference dose for phenanthrene is based on a naphthalene oral gavage study in rats. In this study, an applied dose was used.

PHENANTHRENE RAFs Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{1} = 0.91$	$\frac{0.18}{1} = 0.18$	$\frac{0.91}{1} = 0.91$	$\frac{0.91}{1} = 0.91$

The oral and dermal subchronic reference dose for phenanthrene is based on a naphthalene oral gavage study conducted in rats. In this study, an applied dose was used.

PHENANTHRENE RAFs Evaluation of Subchronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{0.91}{1} = 0.91$	$\frac{0.18}{1} = 0.18$	$\frac{0.91}{1} = 0.91$	$\frac{0.91}{1} = 0.91$

TETRACHLOROETHYLENE

CAS #: 127184

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.1 mg/kg/day (2)
Chronic Oral Reference Dose: 0.01 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: 4600 $\mu g/m^3$ (3a)
Chronic Inhalation Reference Concentration: 4600 $\mu g/m^3$ (3)

Oral Cancer Potency Factor: 0.052 $(mg/kg/day)^{-1}$ (2h)
Inhalation Cancer Unit Risk: 0.00000058 $(\mu g/m^3)^{-1}$ (2h)

Results from several animal studies (Pegg et al., 1979; Schumann et al., 1980; Frantz and Wantanabe, 1983) indicate that tetrachloroethylene is rapidly and virtually completely absorbed following oral administration.

Frantz, S.W. and Wantanabe, P.G. (1983) *Tetrachloroethylene: Balance and Tissue Distribution in Male Sprague-Dawley Rats by Drinking Water Administration.* **Toxicol. Appl. Pharmacol.** 69:66-72.

Pegg, D.G., Zemple, J.A., Braun, W.H. and Watanabe, P.G. (1979) *Disposition of (^{14}C)Tetrachloroethylene Following Oral and Inhalation Exposure in Rats.* **Toxicol. Appl. Pharmacol.** 51:465-474.

Schumann, A.M., Quast, J.F., and Watanabe, P.G. (1980) *The Pharmacokinetics and Macromoleular Interactions of Perchloroethylene in Mice and Rats as Related to Oncogenicity.* **Toxicol. Appl. Pharmacol.** 55:207-219.

Dermal absorption of tetrachloroethylene appears to be poor (0.24 $mg/cm^2/hr$) (Tsuruta, 1975). Therefore, the absorption efficiency by the dermal route in a non-occluded exposure probably does not exceed 10% (see benzene).

Tsuruta, H. (1975) *Percutaneous absorption of organic solvents. I. Comparative study of +6 percutaneous absorption of chlorinated solvents in mice.* **Ind. Health** 13:227-236.

The oral and dermal cancer potency value for tetrachloroethylene is based on a gavage study in mice. This toxicity value is not based on absorbed dose.

TETRACHLOROETHYLENE RAFs Evaluation of Carcinogenicity			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{1}{1} = 1$	$\frac{0.1}{1} = 0.1$	$\frac{1}{1} = 1$	$\frac{1}{1} = 1$

THALLIUM CAS #: 7440280

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.0007 mg/kg/day (2)
Chronic Oral Reference Dose: 0.00007 mg/kg/day (2)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: Not Volatile

Limited human data suggest that most of an oral dose of thallium (applied as thallium nitrate) is absorbed from the gastrointestinal tract (Barclay et al., 1953). Animal studies suggest that thallium is completely absorbed when ingested. A single trace dose of thallium^{204} (as thallium nitrate) was administered orally to rats (Lie et al., 1960). The body burden of thallium^{204}, as percent dose, decreased with a single exponential function which extrapolated to 100% at zero time. It was concluded that thallium is completely absorbed from the gastrointestinal tract. A 100% oral absorption efficiency is therefore assumed for thallium compounds.

Barclay, R.K., Pencock, W.C., Kanofsy, D.A. (1953) *Distribution and Excretion of Radioactive Thallium in the Chick Embryo, Rat and Man.* **J. Pharmacol. Exp. Ther.** 107:178-187.

Lie, R., Thomas, R. and Scott, J. (1960) *The Distribution and Excretion of Thallium^{204} in the Rat, with Suggested MPC's and a Bio-Assay Procedure.* **Health Phys.** 2:334-340.

No quantitative studies were located regarding the dermal absorption of thallium in humans or animals. Assume a dermal absorption efficiency of 1% as a conservative upper-bound estimate (see chromium).

THALLIUM

The oral RfD for thallium of 7E-5 mg/kg-day is based on a rat study in which thallium sulfate was administered by gavage. The MADEP (1992) assumed that thallium was completely absorbed in this study.

Derivation of Dermal-Water AAF

The methodology for estimating risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this process is an absorbed dose. The oral RfD is based on administered dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment was made to the absorbed dermal dose in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to multiplying the RfD by the percent absorption in the applicable dose-response study. The AAF (dermal-water) for use with the RfD is 1/100% = 1.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation of the Risk Assessment ShortForm-Residential Scenario.

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TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 2 mg/kg/day (2)
Chronic Oral Reference Dose: 0.2 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: 2000 $\mu g/m^3$ (2)
Chronic Inhalation Reference Concentration: 400 $\mu g/m^3$ (1)

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: NC

Rabbit studies indicate that essentially 100% of an oral dose of toluene is either excreted as metabolites or exhaled unchanged, implying 100% absorption efficiency by the oral route (Smith et al., 1954; El Masry et al., 1956). Oral absorption efficiency of soil-adsorbed toluene was not changed from that of the pure compound even though absorption was delayed in time by the presence of sandy soil (Turkall et al., in press).

El Masry, A.M., Smith, J.N. and Williams, R.T. (1956) *Studies in Detoxication 69. The Metabolism of Alkylbenzenes: n-Propylbenzene and n-Butylbenzene with Further Observations on Ethylbenzene.* Biochem. J. 64:50-56.

Smith, J.N., Smithies, R.H. and Williams, R.T. (1954) *Studies in Detoxication 55. The Metabolism of Alkylbenzenes.* Biochem. J. 56:317-325.

Turkall, R.M., Skowronski, G.A. and Abdel-Rahman, M.S. (in press) *Differences in Kinetics of Pure and Soil-Adsorbed Toluene in Orally Exposed Male Rats.* Arch. Environ. Contam. Toxicol.

The dermal absorption of toluene has been measured to be approximately 2% of the applied dose in 4 hours, or 12% in 24 hours (Susten et al., 1990). This study allowed for volatilization of toluene, rapidly decreasing the actual applied dose. In a second study (Skowronski, et al., 1989), volatilization loss was minimized to less than 10% of the applied dose by occlusion. The dermal absorption efficiency was estimated to be approximately 90% with volatilization loss accounting for the remainder of the dose (essentially 100% dermal absorption). The estimate was based on recovery of radioactivity in urine, feces and expired air. The dermal absorption of toluene was unaffected by its adsorption to clay or sandy soils. The 24 hour non-occluded value (12%) is assumed to be the most appropriate and protective for human exposure.

Susten, A.S., Niemeier, R.W. and Simon, S.D. (1990) *In Vivo Percutaneous Absorption Studies of Volatile Organic Solvents in Hairless Mice II. Toluene, Ethylbenzene and Aniline.* J. Appl. Toxicol. 10:217-225.

Skowronski, G.A., Turkall, R.M. and Abdel-Rahman, M.S. (1989) *Effects of Soil on Percutaneous Absorption of Toluene in Male Rats.* J. Toxicol. Environ. Health 26:373-384.

TOLUENE

The MADEP (1992) assumed 100% gastrointestinal absorption of toluene when developing AAFs. GZA, therefore, assumed 100% gastrointestinal absorption of toluene in the dose-response study. The MADEP has derived AAFs for toluene for the oral route, but not for the dermal-water exposure route. Thus, GZA has derived dermal-water AAFs for toluene.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral RfD for toluene is based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the RfD to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (multiplying the RfD by the percent absorption). For toluene, the AAF (dermal-water) is 1/(100%) = 1.0.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

U.S. EPA. 1992. Integrated Risk Information System (IRIS).

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1,1,1-TRICHLOROETHANE

CAS #: 71556

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.9 mg/kg/day (2)
Chronic Oral Reference Dose: 0.09 mg/kg/day (2)

Subchronic Inhalation Reference Concentration: 10,000 $\mu g/m^3$ (2)
Chronic Inhalation Reference Concentration: 1000 $\mu g/m^3$ (2)

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: NC

No studies were located containing information to quantitate the oral absorption efficiency of 1,1,1-trichloroethane. However, it can be assumed to be rapidly and completely absorbed in a manner similar to other chlorinated volatiles.

No studies were located containing information to quantitate the dermal absorption efficiency of 1,1,1-trichloroethane. Assume a 10% absorption efficiency (non-occluded, 24 hour), comparable to other volatile compounds.

The oral and dermal chronic reference dose for 1,1,1-trichloroethane is based on an inhalation study conducted in guinea pigs. An absorption efficiency of 30% was used to calculate an absorbed dose. The RAFs to be used for all exposure pathways are the absorption efficiencies of 1,1,1-trichloroethane by the route in question.

1,1,1-TRICHLOROETHANE RAFs Evaluation of Chronic Exposures			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
1	0.1	1	1

1,1,1-TRICHLOROETHANE

The MADEP (1992) derived AAFs for ingestion exposure to soil, water and food and for dermal exposure to soil. MADEP has not derived dermal-water AAFs for any of the VOCs. The chronic oral RfD of 9.0E-02 mg/kg-day is based on an absorbed dose in an inhalation study, wherein U.S. EPA assumed 30% absorption. GZA derived a dermal-water AAF for this chemical as described below.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral RfD for 1,1,1-trichloroethane is based on an absorbed dose. Therefore, no adjustment is necessary to convert the RfD to be based on an absorbed dose. The oral RfD can be used directly with the absorbed dermal dose. A value of 1 was entered into the spreadsheets which resulted in no adjustment to the dose.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

U.S. EPA. 1992. Integrated Risk Information System (IRIS).

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TRICHLOROETHYLENE

CAS #: 79016

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.02 mg/kg/day (4)
Chronic Oral Reference Dose: 0.002 mg/kg/day (4)

Subchronic Inhalation Reference Concentration: 180 $\mu g/m^3$ (3a)
Chronic Inhalation Reference Concentration: 180 $\mu g/m^3$ (3)

Oral Cancer Potency Factor: 0.011 $(mg/kg/day)^{-1}$ (2h)
Inhalation Cancer Unit Risk: 0.0000017 $(\mu g/m^3)^{-1}$ (2h)

Oral absorption studies in experimental animals indicate that trichloroethylene is extensively absorbed by the oral route. Absorption efficiency was measured as 91% - 98% of an applied oral dose (Prout et al., 1985; Dekant et al., 1984) determined as radioactivity in expired air and urine. Radioactivity in the carcass was not determined. Absorption, therefore, is assumed to be complete.

Dekant, W., Metzler, M. and Henschler, D. (1984) *Novel Metabolites of Trichloroethylene Through Dechlorination Reactions in Rats, Mice and Humans*. **Biochem. Pharmacol.** 33:2021-2027.

Prout, M.S., Provan, W.M. and Green, T. (1985) *Species Differences in Response to Trichloroethylene*. **Toxicol. Appl. Pharmacol.** 79:389-400.

No studies were located regarding the dermal absorption efficiency of trichloroethylene. Assume 10% dermal absorption (non-occluded, 24 hour) based on physical and chemical properties similar to the other volatile compounds.

The oral and dermal cancer potency value for trichloroethylene is based on a gavage study in mice. This toxicity value is not based on absorbed dose.

TRICHLOROETHYLENE RAFs Evaluation of Carcinogenicity			
SOIL INGESTION	SOIL DERMAL	WATER INGESTION	VEGETABLE INGESTION
$\frac{1}{1} = 1$	$\frac{0.1}{1} = 0.1$	$\frac{1}{1} = 1$	$\frac{1}{1} = 1$

TRICHLOROETHYLENE

The MADEP (1992) assumed 100% gastrointestinal absorption of trichloroethylene when developing AAFs. The MADEP, has not, however, derived dermal-water AAFs for any of the VOCs. Thus, GZA has derived a dermal-water AAF for trichloroethylene, assuming 100% gastrointestinal absorption of trichloroethylene in the gavage study upon which the oral/dermal dose-response value is based.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral CSF for trichloroethylene is based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the CSF to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (dividing the CSF by the percent absorption). For trichloroethylene, the AAF (dermal-water) is 1/(100%) = 1.0.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

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TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.001 mg/kg/day (4)
Chronic Oral Reference Dose: 0.001 mg/kg/day (4)

Subchronic Inhalation Reference Concentration: 17 $\mu g/m^3$ (3a)
Chronic Inhalation Reference Concentration: 17 $\mu g/m^3$ (3)

Oral Cancer Potency Factor: 1.9 $(mg/kg/day)^{-1}$ (2)
Inhalation Cancer Unit Risk: 0.000084 $(\mu g/m^3)^{-1}$ (2)

In young human volunteers administered vinyl chloride monomer for 6 hours, an average retention of 42% was estimated. It was not reported whether steady state had been achieved. Maximum retention was achieved at 15 minutes and retention declined after 30 minutes after which it increased to a relatively constant value. The percentage retained seemed to be independent of the concentration inhaled. A range of reported inhalation absorption efficiencies is 40% - 98%, 64% being suggested as representative and protective (Owen, 1990)

Owen, B.A. (1990) *Literature-Derived Absorption Coefficients for 39 Chemicals Via Oral and Inhalation Routes of Exposure*. **Regul. Toxicol. Pharmacol.** 11:237-252.

Krajewski, J., Dobecki, M. and Gromiec, J. (1980) *Retention of Vinyl Chloride in the Human Lung*. **Br. J. Ind. Med.** 37:373-374.

Rats were administered single gavage doses of ^{14}C-vinyl chloride in corn oil and radioactivity levels excreted in expired air, urine and feces, as well as the amount remaining in the carcass, were measured at 72 hours. 0.47-2.39% of the administered dose was recovered in the feces indicating that absorption was nearly complete. Total recovery ranged from 82.3-91.3% suggesting a substantial loss of radioactivity. An oral absorption efficiency of 98% is assumed to be protective for human exposure.

Watanabe, P.G., McGowan, G.R. and Gehring, P.J. (1976) *Fate of [^{14}C] Vinyl Chloride After Single Oral Administration*. **Toxicol. Appl. Pharmacol.** 36:339-352.

Two rhesus monkeys exposed from the neck down to ^{14}C-vinyl chloride vapor were found to have dermal absorptions of 0.031% and 0.023%, respectively. The dermal absorption of neat vinyl chloride or soil contaminated with vinyl chloride would be expected to be greater due to increased time of dermal contact. Therefore, the dermal absorption efficiency is assumed to be similar to the 10% estimate derived for other volatile compounds (non-occluded, 24 hour).

VINYL CHLORIDE

The MADEP (1992) derived AAFs for vinyl chloride assuming that the oral CSF was based on an inhalation study. This is incorrect. The oral CSF of 1.9 $(mg/kg\text{-}day)^{-1}$ is based on a dietary study in rats (U.S. EPA, 1992). After a review of the literature, MADEP (1992) assumed that the oral absorption efficiency of vinyl chloride is 98%. GZA derived revised AAFs for use with the oral CSF, assuming that the CSF was based upon an oral study and that the absorption in this study was 98%. This is the absorption MADEP assumed in the noncancer dose-response study. However, it is not clear why this value was used, since this is the upper end of the range (40-98%) reported for inhalation studies. For oral studies, MADEP (1992) reported an absorption range of 82.3-91.3%. To be conservative, and to be consistent with MADEP's assumed absorption in the noncancer dose-response study, GZA assumed 98% absorption in the oral cancer dose-response study.

Derivation of Oral-Soil, Oral-Water and Oral-Diet AAFs

GZA assumed that the oral absorption of vinyl chloride in the oral cancer dose-response study was 98%. GZA also assumed that the oral absorption by humans of vinyl chloride in soil, water or the diet is also 98%. Thus, the AAFs (oral-soil, oral-water, and oral-diet) are all equal to 98%/98% = 1.00.

Derivation of Dermal-Soil AAF

The MADEP (1992) assumed that the dermal absorption of vinyl chloride in soil was about 10% of the applied dose. The AAF (dermal-soil), therefore, equals 10%/98% = 0.1.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral RfDs and CSFs for vinyl chloride are based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the RfDs and CSFs to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (multiplying the RfD, or dividing the CSF, by the percent absorption).

The oral CSF and oral RfD for vinyl chloride are based on an oral dietary study in rats. The MADEP (1992) and GZA assumed that the oral absorption from these studies was 98%. Therefore, for vinyl chloride, the AAF (dermal-water) is equal to 1/98% = 1.02.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

U.S. EPA, 1992. Health Effects Assessment Summary Tables (HEAST).

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TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 4 mg/kg/day (2)
Chronic Oral Reference Dose: 2 mg/kg/day (1)

Subchronic Inhalation Reference Concentration: 300 $\mu g/m^3$ (2)
Chronic Inhalation Reference Concentration: 300 $\mu g/m^3$ (2)

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: NC

The oral absorption efficiency of 100% is estimated from limited excretion data specifying that more than 98% of an oral dose of p-xylene was absorbed and excreted as metabolites in urine and expired air of the rabbit.

Bray, H.G., Humphric, B.G. and Thorpe, W.V. (1949) *Metabolism of Derivatives of Toluene 3. o-,m- and p-Xylenes.* Biochem. J. 45:241-244.

The dermal absorption efficiency of m-xylene, adsorbed to either sand or clay, has been demonstrated to be essentially 100% of an occluded dose when applied to the shaved skin of male rats (Skowronski et al., 1990). Absorption was rapid with 50% of the dose absorbed in less than 1 hour. Soil adsorption slightly delayed the dermal absorption of m-xylene relative to pure parent compound. The dermal absorption of soil adsorbed mixed xylene isomers is assumed to behave as m-xylene. No information exists on non-occluded dermal uptake, however, it would be assumed to be similar to that of its structural analog, toluene (12% in 24 hours).

Skowronski, G.A., Turkall, R.M., Kadry, A.R.M. and Abdel-Rahmen, M.S. (1990) *Effects of soil on the dermal bioavailability of m-xylene in male rats.*

XYLENE

The MADEP (1992) assumed 100% gastrointestinal absorption of xylene when developing AAFs. GZA, therefore, assumed 100% gastrointestinal absorption of xylene in the dose-response study. The MADEP has derived AAFs for xylene for the oral route, but has not derived dermal-water AAFs for any of the VOCs. Thus, GZA has derived dermal-water AAFs for xylene.

Derivation of Dermal-Water AAF

The methodology for estimating the risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The oral RfD for xylene is based on an administered (exposure) dose. Therefore, an adjustment is necessary to convert the RfD to be based on an absorbed dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment is made to the absorbed dermal dose in the exposure assessment, rather than to the dose-response criteria. This approach also enables all absorption adjustments (all pathways) to be performed in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to adjusting the dose-response criteria (multiplying the RfD by the percent absorption). For xylene, the AAF (dermal-water) is 1/(100%) = 1.0.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation for the Residential Short Form.

U.S. EPA. 1992. Integrated Risk Information System (IRIS).

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ZINC CAS #: 7440666

TOXICITY INFORMATION FROM THE RESIDENTIAL SHORTFORM:

Subchronic Oral Reference Dose: 0.2 mg/kg/day (2)
Chronic Oral Reference Dose: 0.2 mg/kg/day (2)

Subchronic Inhalation Reference Concentration: Not Volatile
Chronic Inhalation Reference Concentration: Not Volatile

Oral Cancer Potency Factor: NC
Inhalation Cancer Unit Risk: Not Volatile

The absorption of zinc in humans from the diet has been determined to range from 22% - 46% (Sandstrom et al., 1987) with the upper limit of the range suggested as an appropriate estimate. Zinc is more efficiently absorbed from drinking water with absorption estimates ranging up to 58% (Dinsmore et al., 1985; Farah et al., 1984; Valberg et al., 1985).

Dinsmore, W., Callender, M.E., McMaster, D., Todd, S.J. and Love, A.H.G. (1985) *Zinc Absorption in Alcoholics Using Zinc-65*. Digest. 32:238-242.

Farah, D.A., Hall, M.J., Mills, P.R. and Russell, R.I. (1984) *Effect of Wheat Bran on Zinc Absorption*. Human Nutr. Clin. Nutr. 38C:433-441.

Sandstrom, B., Davison, L., Kivisto, B., Hasselbland, C. and Cederbland, A. (1987) *The Effect of Vegetables and Beet Fibre on the Absorption of Zinc in Humans from Composite Meals*. Brit. J. Nutr. 58:49-57.

Valberg, L.S., Flanagan, P.R., Ghent, C.N. and Chemberlai, M.J. (1985) *Zinc Absorption and Leukocyte Zinc in Alcoholic and Nonalcoholic Cirrhosis*. Digest. Dis. Sci. 30:329-333.

No studies were located quantitatively describing the dermal absorption of zinc compounds. Assume a dermal absorption efficiency of 1% as a conservative upper-bound estimate (see chromium).

ZINC

The oral RfD for zinc of 2E-1 mg/kg-day is based on a human study with zinc sulfate in the diet. The MADEP (1992) assumed that the gastrointestinal absorption of zinc from the diet in the human subjects was 46%, the upper end of the range (22-46%) reported by Sandstrom et al. (1987).

Derivation of Dermal-Water AAF

The methodology for estimating risks posed by dermal exposure to chemicals of concern in water utilizes a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this process is an absorbed dose. The oral RfD is based on administered dose. In order to use consistent dose-response criteria across all exposure pathways, the adjustment was made to the absorbed dermal dose in the exposure assessment section. The AAF for dermal-water equals 1/(absorption in the dose-response study), which is mathematically equivalent to multiplying the RfD by the percent absorption in the applicable dose-response study. The AAF (dermal-water) for use with the RfD is 1/46% = 2.2.

REFERENCES

MADEP (Massachusetts Department of Environmental Protection). 1992. Documentation of the Risk Assessment ShortForm-Residential Scenario.

Sandstrom, B., L. Davidson, B. Kivisto, C. Hasselbland and A. Cederbland. 1987. The effect of vegetables and beet fibre on the absorption of zinc in humans from composite meals. *British Journal of Nutrition*, 58:49-57.

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