

Appendix F.7

Toxicity Profiles

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F.7.1 ARSENIC

F.7.1.1 PHARMACOKINETICS

Several studies confirm that soluble inorganic arsenic compounds and organic arsenic compounds are almost completely (>90 percent) absorbed from the GI tract in both animals and humans (Ishinishi et al. 1986). The absorption efficiency of insoluble inorganic arsenic compounds depends on particle size and stomach pH. Initial distribution of absorbed arsenic is to the liver, kidneys, and lungs, followed by redistribution to hair, nails, teeth, bone, and skin, which are considered tissues of accumulation. Arsenic has a long half-life in the blood of rats, compared with other animals and humans, because of firm binding to the hemoglobin in erythrocytes.

Metabolism of inorganic arsenic includes reversible oxidation-reduction so that both arsenite (valence of 3) and arsenate (valence of 5) are present in the urine of animals treated with arsenic of either valence (Ishinishi et al. 1986). Arsenite is subsequently oxidized and methylated by a saturable mechanism to form mono- or dimethylarsenate; the latter is the predominant metabolite in the urine of animals or humans. Organic arsenic compounds (arsenilic acid, cacodylic acid) are not readily converted to inorganic arsenic. Excretion of organic or inorganic arsenic is largely via the urine, but considerable species variation exists. Continuously exposed humans appear to excrete 60 to 70 percent of their daily intake of arsenate or arsenite via the urine.

F.7.1.2 NONCANCER TOXICITY

A lethal dose of arsenic trioxide in humans is 70 to 180 mg. (approximately 50 to 140 mg arsenic; Ishinishi et al. 1986). Acute oral exposure of humans to high doses of arsenic produces liver swelling, skin lesions, disturbed heart function, and neurological effects. The only noncancer effects in humans clearly attributable to chronic oral exposure to arsenic are dermal hyperpigmentation and keratosis, as revealed by studies of several hundred Chinese exposed to naturally occurring arsenic in well water (Tseng 1977; Tseng et al. 1968; EPA 1998b). Similar effects were observed in persons exposed to high levels of arsenic in water in Utah and the northern part of Mexico (Cebrian et al. 1983; Southwick et al. 1983). Occupational (predominantly inhalation) exposure is also associated with neurological deficits, anemia, and cardiovascular effects (Ishinishi et al. 1986), but concomitant exposure to other chemicals cannot be ruled out. The EPA (1998b) derived an RfD of 0.3 ug/kg/day for chronic oral exposure, based on an NOAEL of 0.8 ug/kg/day for skin lesions from Chinese data. The principal target organ for arsenic appears to be the skin. The nervous system and cardiovascular systems appear to be less significant target organs. Inorganic arsenic may be an essential nutrient, exerting beneficial effects on growth, health, and feed conversion efficiency (Underwood 1977).

F.7.1.3 CARCINOGENICITY

Inorganic arsenic is clearly a carcinogen in humans. Inhalation exposure is associated with increased risk of lung cancer in persons employed as smelter workers, in arsenical pesticide applicators, and in a population residing near a pesticide manufacturing plant (EPA 1998b). Oral exposure to high levels in well water is associated with increased risk of skin cancer (Tseng 1977; EPA 1998b). Extensive animal testing with various forms of arsenic given by many routes of exposure to several species, however, has not demonstrated the carcinogenicity of arsenic (International Agency for Research on Cancer [IARC 1980]). The EPA (1998b) classifies inorganic arsenic in cancer weight-of-evidence Group A (human carcinogen), and recommends an oral unit risk of 0.00005 ug/L in drinking water, based on the incidence of skin cancer in the Tseng (1977) study. The EPA presents a chronic oral slope factor of 1.5 per mg/kg/day based on the same information. The EPA (1998b) notes that the uncertainties associated with the oral unit risk are considerably less than those for most carcinogens, so that the unit risk might be reduced in order of magnitude. An inhalation unit risk of 0.0043 per mg/m³ was derived for inorganic arsenic from the incidence of lung cancer in occupationally exposed men (EPA 1998b), equivalent to 15.1 per mg/kg/day, was derived from the same data assuming an inhalation rate of 20 m³/day and a body weight of 70 kg for humans.

F.7.2 ASBESTOS

F.7.2.1 NONCANCER TOXICITY

Data not available at this time.

F.7.2.2 CARCINOGENICITY

This section provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to the following sections for information on long-term toxic effects other than carcinogenicity.

Weight-Of-Evidence Classification

Classification -- A; human carcinogen

Basis -- Observation of increased mortality and incidence of lung cancer, mesotheliomas and gastrointestinal cancer in occupationally exposed workers are consistent across investigators and study populations. Animal studies by inhalation in two strains of rats showed similar findings for lung cancer and mesotheliomas. Animal evidence for carcinogenicity via ingestion is limited (male rats fed intermediate-range chrysotile fibers; i.e., >10 um length, developed benign polyps), and epidemiologic data in this regard are inadequate.

Human Carcinogenicity Data

Sufficient. Numerous epidemiologic studies have reported an increased incidence of deaths due to cancer, primarily lung cancer and mesotheliomas associated with exposure to inhaled asbestos. Among 170 asbestos insulation workers in North Ireland followed for up to 26 years, an increased incidence of death was seen due to all cancers (SMR=390), cancers of the lower respiratory tract and pleura (SMR=1760) (Elmes and Simpson, 1971) and mesothelioma (7 cases). Exposure was not quantified.

Selikoff (1976) reported 59 cases of lung cancer and 31 cases of mesothelioma among 1249 asbestos insulation workers followed prospectively for 11 years. Exposure was not quantified. A retrospective cohort mortality study (Selikoff et al., 1979) of 17,800 U.S. and Canadian asbestos insulation workers for a 10-year period using best available information (autopsy, surgical, clinical) reported an increased incidence of cancer at all sites (319.7 expected vs. 995 observed, SMR=311) and cancer of the lung (105.6 expected vs. 486 observed, SMR=460). A modest increase in deaths from gastrointestinal cancer was reported along with 175 deaths from mesothelioma (none expected). Years of exposure ranged from less than 10 to greater than or equal to 45. Levels of exposure were not quantified. In other epidemiologic studies, the increase for lung and pleural cancers has ranged from a low of 1.9 times the expected rate, in asbestos factory workers in England (Peto et al., 1977), to a high of 28 times the expected rate, in female asbestos textile workers in England (Newhouse et al., 1972). Other occupational studies have demonstrated asbestos exposure-related increases in lung cancer and mesothelioma in several industries including textile manufacturing, friction products manufacture, asbestos cement products, and in the mining and milling of asbestos. The studies used for the inhalation quantitative estimate of risk are listed in the table in Section II.C.2.

A case-control study (Newhouse and Thompson, 1965) of 83 patients with mesothelioma reported 52.6% had occupational exposure to asbestos or lived with asbestos workers compared with 11.8% of the controls. Of the remaining subjects, 30.6% of the mesothelioma cases lived within one-half mile of an asbestos factory compared with 7.6% of the controls.

The occurrence of pleural mesothelioma has been associated with the presence of asbestos fibers in water, fields and streets in a region of Turkey with very high environmental levels of naturally-occurring asbestos (Baris et al., 1979).

Kanarek et al. (1980) conducted an ecologic study of cancer deaths in 722 census tracts in the San Francisco Bay area, using cancer incidence data from the period of 1969-1971. Chrysotile asbestos concentrations in drinking water ranged from nondetectable to $3.6E+7$ fibers/L. Statistically significant dose-related trends were reported for lung and peritoneal cancer in white males and for gall bladder, pancreatic and peritoneal cancer in white females. Weaker correlations were reported between asbestos

levels and female esophageal, pleural and kidney cancer, and stomach cancer in both sexes. In an extension of this study, Conforti et al. (1981) included cancer incidence data from the period of 1969-1974. Statistically significant positive associations were found between asbestos concentration and cancer of the digestive organs in white females, cancers of the digestive tract in white males and esophageal, pancreatic and stomach cancer in both sexes. These associations appeared to be independent of socioeconomic status and occupational exposure to asbestos.

Marsh (1983) reviewed eight independent ecologic studies of asbestos in drinking water carried out in five geographic areas. It was concluded that even though one or more studies found an association between asbestos in water and cancer mortality (or incidence) due to neoplasms of various organs, no individual study or aggregation of studies exists that would establish risk levels from ingested asbestos. Factors confounding the results of these studies include the possible underestimates of occupational exposure to asbestos and the possible misclassification of peritoneal mesothelioma as GI cancer.

Polissar et al. (1984) carried out a case-control study which included better control for confounding variables at the individual level. The authors concluded that there was no convincing evidence for increased cancer risk from asbestos ingestion. At the present time, an important limitation of both the case-control and the ecologic studies is the short follow-up time relative to the long latent period for the appearance of tumors from asbestos exposure.

Animal Carcinogenicity Data

Sufficient. There have been about 20 animal bioassays of asbestos. Gross et al. (1967) exposed 61 white male rats (strain not reported) to 86 mg chrysotile asbestos dust/cu.m for 30 hours/week for 16 months. Of the 41 animals that survived the exposure period, 10 had lung cancer. No lung cancer was observed in 25 controls.

Reeves (1976) exposed 60-77 rats/group for 4 hours/day, 4 days/week for 2 years to doses of 48.7-50.2 mg/cu.m crocidolite, 48.2-48.6 mg/cu.m amosite and 47.4-47.9 mg/cu.m chrysotile. A 5-14% incidence of lung cancer was observed among concentration groups and was concentration-dependent.

Wagner et al. (1974) exposed CD Wistar rats (19-52/group) to 9.7-14.7 mg/cu.m of several types of asbestos for 1 day to 24 months for 7 hours/day, 5 days/week. A duration-dependent increased incidence of lung carcinomas and mesotheliomas was seen for all types of asbestos after 3 months of exposure compared with controls.

F344 rats (88-250/group) were exposed to intermediate range chrysotile asbestos (1291E+8 f/g) in drinking water by gavage to dams during lactation and then in diet throughout their lifetime (NTP, 1985). A

statistically significant increase in incidence of benign epithelial neoplasms (adenomatous polyps in the large intestine) was observed in male rats compared with pooled controls of all NTP oral lifetime studies (3/524). In the same study, rats exposed to short range chrysotile asbestos (6081E+9 f/g) showed no significant increase in tumor incidence.

Ward et al. (1980) administered 10 mg UICC amosite asbestos 3 times/week for 10 weeks by gavage to 50 male F344 rats. The animals were observed for an additional 78-79 weeks post-treatment. A total of 17 colon carcinomas were observed. This result was statistically significant compared with historical controls; no concurrent controls were maintained.

Syrian golden hamsters (126-253/group) were exposed to short and intermediate range chrysotile asbestos at a concentration of 1% in the diet for the lifetime of the animals (NTP, 1983). An increased incidence of neoplasia of the adrenal cortex was observed in both males and females exposed to intermediate range fibers and in males exposed to short range fibers. This increase was statistically significant by comparison to pooled controls but not by comparison to concurrent controls. NTP suggested that the biologic importance of adrenal tumors in the absence of target organ (GI tract) neoplasia was questionable.

Quantitative Estimate Of Carcinogenic Risk From Oral Exposure

Not available.

Quantitative Estimate Of Carcinogenic Risk From Inhalation Exposure

SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 2.3E-1 per (f/mL)

Extrapolation Method -- Additive risk of lung cancer and mesothelioma, using relative risk model for lung cancer and absolute risk model for mesothelioma.

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-4 f/mL
E-5 (1 in 100,000)	4E-5 f/mL
E-6 (1 in 1,000,000)	4E-6 f/mL

Additional Comments (Carcinogenicity, Inhalation Exposure)

Risks have been calculated for males and females according to smoking habits for a variety of exposure scenarios (U.S. EPA, 1986). The unit risk value is calculated for the additive combined risk of lung cancer and mesothelioma, and is calculated as a composite value for males and females. The epidemiological data show that cigarette smoking and asbestos exposure interact synergistically for production of lung cancer and do not interact with regard to mesothelioma. The unit risk value is based on risks calculated using U.S. general population cancer rates and mortality patterns without consideration of smoking habits. The risks associated with occupational exposure were adjusted to continuous exposure by applying a factor of 140 cu.m/50 cu.m based on the assumption of 20 cu.m/day for total ventilation and 10 cu.m/8-hour workday in the occupational setting.

The unit risk is based on fiber counts made by phase contrast microscopy (PCM) and should not be applied directly to measurements made by other analytical techniques. The unit risk uses PCM fibers because the measurements made in the occupational environment use this method. Many environmental monitoring measurements are reported in terms of fiber counts or mass as determined by transmission electron microscopy (TEM). PCM detects only fibers longer than 5 μm and $>0.4 \mu\text{m}$ in diameter, while TEM can detect much smaller fibers. TEM mass units are derived from TEM fiber counts. The correlation between PCM fiber counts and TEM mass measurements is very poor. Six data sets which include both measurements show a conversion between TEM mass and PCM fiber count that range from 5-150 (ug/cu.m)/(f/mL). The geometric mean of these results, 30 (ug/cu.m)/(f/mL), was adopted as a conversion factor (U.S. EPA, 1986), but it should be realized that this value is highly uncertain. Likewise, the correlation between PCM fiber counts and TEM fiber counts is very uncertain and no generally applicable conversion factor exists for these two measurements.

In some cases TEM results are reported as numbers of fibers $<5 \mu\text{m}$ long and of fibers longer than 5 μm . Comparison of PCM fiber counts and TEM counts of fibers $>5 \mu\text{m}$ show that the fraction of fibers detected by TEM that are also $>0.4 \mu\text{m}$ in diameter (and detectable by PCM) varies from 22-53% (U.S. EPA, 1986).

It should be understood that while TEM can be specific for asbestos, PCM is a nonspecific technique and will measure any fibrous material. Measurements by PCM which are made in conditions where other types of fibers may be present may not be reliable.

In addition to the studies cited above, there were three studies of asbestos workers in mining and milling which showed an increase in lung cancer (McDonald et al., 1980, Nicholson et al., 1979; Rubino et al., 1979). The slope factor calculated from these studies was lower than the other studies, possibly because of a substantially different fiber size distribution, and they were not included in the calculation. The slope

factor was calculated by life table methods for lung cancer using a relative risk model, and for mesothelioma using an absolute risk model. The final slope factor for lung cancer was calculated as the weighted geometric mean of estimates from the 11 studies cited in section II.C.2. The final slope factor for mesothelioma is based on the calculated values from the studies of Selikoff et al. (1979), Peto et al. (1982), Seidman et al. (1979), Peto (1980) and Finkelstein (1983) adjusted for the mesothelioma incidence from several additional studies cited previously.

There is some evidence which suggests that the different types of asbestos fibers vary in carcinogenic potency relative to one another and site specificity. It appears, for example, that the risk of mesothelioma is greater with exposure to crocidolite than with amosite or chrysotile exposure alone. This evidence is limited by the lack of information on fiber exposure by mineral type. Other data indicates that differences in fiber size distribution and other process differences may contribute at least as much to the observed variation in risk as does the fiber type itself.

The unit risk should not be used if the air concentration exceeds $4E-2$ fibers/ml, since above this concentration the slope factor may differ from that stated.

Discussion Of Confidence (Carcinogenicity, Inhalation Exposure)

A large number of studies of occupationally-exposed workers have conclusively demonstrated the relationship between asbestos exposure and lung cancer or mesothelioma. These results have been corroborated by animal studies using adequate numbers of animals. The quantitative estimate is limited by uncertainty in the exposure estimates, which results from a lack of data on early exposure in the occupational studies and the uncertainty of conversions between various analytical measurements for asbestos.

F.7.3 BARIUM

F.7.3.1 NONCANCER TOXICITY

Barium is a naturally occurring alkaline earth metal that comprises approximately 0.04 percent of the earth's crust (Reeves 1986a). Acute oral toxicity was manifested by GI upset, altered cardiac performance, and transient hypertension, convulsions, and muscular paralysis. Repeated oral exposures were associated with hypertension. Occupational exposure to insoluble barium sulfate induced benign pneumoconiosis (ACGIH 1991). The EPA (1997) presented a verified chronic oral RfD of 0.07 mg/kg/day, based on an NOAEL of 0.21 mg/kg/day in a ten-week study in humans exposed to barium in drinking water and an uncertainty factor of 3. The EPA (1997) presented the same value as a provisional RfD for subchronic oral exposure. A provisional chronic inhalation RfC of 0.0005 mg/m³ and a provisional subchronic inhalation RfC of 0.005 mg/m³ were based on an NOEL for fetotoxicity in a four-month intermittent-exposure inhalation study in rats (EPA 1997). Uncertainty factors of 1000 and 100 were used for the chronic and subchronic RfC values, respectively. The chronic and subchronic inhalation RfC values are equivalent to 0.0001 and 0.001 mg/kg/day, assuming a human inhalation rate of 20 m³/day and body weight of 70 kg. Barium is principally a muscle toxin. Its targets are the GI system, skeletal muscle, the cardiovascular system, and the fetus.

F.7.3.2 CARCINOGENICITY

The EPA (1997) classifies barium as a cancer weight-of-evidence Group D substance (not classifiable as to carcinogenicity in humans). Cancer risk is not estimated for Group D substances.

F.7.4 BENZO[A]ANTHRACENE

F.7.4.1 NONCANCER TOXICITY

The oral and inhalation RfD and RfC are not available at this time (EPA 1998).

F.7.4.2 CARCINOGENICITY

Benzo[a]anthracene has a weight of evidence classification of B2, a probable human carcinogen. The classification was based on sufficient data from animal bioassays. Benzo[a]anthracene produced tumors in mice exposed by gavage; intraperitoneal, subcutaneous or intramuscular injection; and topical application. Benzo[a]anthracene produced mutations in bacteria and in mammalian cells, and transformed mammalian cells in culture.

Although there are no human data that specifically link exposure to benzo[a]anthracene to human cancers, benzo[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soot, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1984; Lee et al., 1976; Brockhaus and Tomingas, 1976).

Benzo[a]anthracene administration caused an increase in the incidence of tumors by gavage (Klein, 1963); dermal application (IARC, 1973); and both subcutaneous injection (Steiner and Faulk, 1951; Steiner and Edgecomb, 1952) and intraperitoneal injection (Wislocki et al., 1986) assays. A group of male mice was exposed to gavage solutions containing 3% benzo[a]anthracene for 5 weeks. There was an increased incidence of pulmonary adenomas and hepatomas.

Supporting data for carcinogenicity include genetic mutations in five different strains of Salmonella typhimurium. Benzo[a]anthracene produced positive results in an assay for mutations in Drosophila melongaster (Fahmy and Fahmy, 1973).

The currently used Oral Slope Factor (CSF) for Benzo[a]anthracene is 7.3E-01 per (mg/kg)/day which is extrapolated from the CSF for Benzo[a]pyrene (BaP), i.e., 0.1×7.3 (BaP) = 7.3E-01 per (mg/kg)/day (USEPA Region III Risk-Based Concentration Table, 4/1/98).

The inhalation CSF is not available.

F.7.5 BENZO [A]PYRENE (BAP)

F.7.5.1 PHARMACOKINETICS

Benzo (a)pyrene was readily absorbed across the GI (Rees et al. 1971) and respiratory epithelia (Kotin et al. 1969; Vainich et al. 1976). Benzo (a)pyrene was distributed widely in the tissues of treated rats and mice, but primarily to tissues high in fat, such as adipose tissue and mammary gland (Kotin et al. 1969; Schlede et al. 1970a).

Studies of the metabolism of benzo(a)pyrene provide information relevant to other PAHs because of the structural similarities of all members of the class. Metabolism involves microsomal mixed function oxidase hydroxylation of one or more of the phenyl rings with the formation of phenols and dihydrodiols, probably via formation of arene oxide intermediates (EPA 1979a). The dihydrodiols may be further oxidized to diol epoxides, which, for certain members of the class, are known to be the ultimate carcinogens (LaVoie et al. 1982). Conjugation with glutathione or glucuronic acid, and reduction to tetrahydrotetraols are important detoxification pathways.

Excretion of benzo(a)pyrene residue was reported to be rapid, although quantitative data were not located (EPA 1979b). Excretion occurred mainly via the feces, probably largely due to biliary secretion (Schlede et al. 1970a, 1970b). The EPA (1980a) concluded that accumulation in the body tissues of PAHs from chronic low level exposure would be unlikely.

F.7.5.2 NONCANCER TOXICITY

The oral RfD and inhalation RfC are not available at this time.

F.7.5.3 CARCINOGENICITY

The PAHs are ubiquitous, being released to the environment from anthropogenic as well as from natural sources (ATSDR 1987). Benzo (a)pyrene is the most extensively studied member of the class, inducing tumors in multiple tissues of virtually all laboratory species tested by all routes of exposure. Although epidemiology studies suggested that complex mixtures that contain PAHs (coal tar, soots, coke oven emissions, cigarette smoke) are carcinogenic to humans (EPA 1994), the carcinogenicity cannot be attributed to PAHs alone because of the presence of other potentially carcinogenic substances in these mixtures (ATSDR 1987). In addition, recent investigations showed that the PAH fraction of roofing tar, cigarette smoke, and coke oven emissions accounted for only 0.1 to 8 percent of the total mutagenic activity of the unfractionated complex mixture in Salmonella (Lewtas 1988). Aromatic amines, nitrogen heterocyclic compounds, highly oxygenated quinones, diones, and nitrooxygenated compounds, none of which would be expected to arise from in vivo metabolism of PAHs, probably accounted for the majority of the mutagenicity of

coke oven emissions and cigarette smoke. Coal tar, which contains a mixture of many PAHs, has a long history of use in the clinical treatment of a variety of skin disorders in humans (ATSDR 1987).

Because of the lack of human cancer data, assignment of individual PAHs to EPA cancer weight-of-evidence groups was based largely on the results of animal studies with large doses of purified compound (EPA 1994). Frequently, unnatural routes of exposure, including implants of the test chemical in beeswax and trioctanoin in the lungs of female Osborne-Mendel rats, intratracheal instillation, and subcutaneous or intraperitoneal injection, were used. Benzo (a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene were classified in Group B2 (probable human carcinogens).

The EPA (1998) verified a slope factor for oral exposure to benzo(a)pyrene of 7.3 per mg/kg/day, based on several dietary studies in mice and rats. Neither verified nor provisional quantitative risk estimates were available for the other PAHs in Group B2. The EPA (1980) promulgated an ambient water quality criterion for "total carcinogenic PAHs," based on an oral slope factor derived from a study with benzo(a)pyrene, as being sufficiently protective for the class. Largely because of this precedent, the quantitative risk estimates for benzo(a)pyrene were adopted for the other carcinogenic PAHs when quantitative estimates were needed.

Human data specifically linking benzo[a]pyrene (BAP) to a carcinogenic effect are lacking. There are, however, multiple animal studies in many species demonstrating BAP to be carcinogenic following administration by numerous routes. In addition, BAP has produced positive results in numerous genotoxicity assays.

The data for animal carcinogenicity was sufficient. The animal data consist of dietary, gavage, inhalation, intratracheal instillation, dermal and subcutaneous studies in numerous strains of at least four species of rodents and several primates. Repeated BAP administration has been associated with increased incidences of total tumors and of tumors at the site of exposure. The tumor types in mice from oral diet studies include forestomach, squamous cell papillomas and carcinomas (Neal and Rigdon 1967).

Benzo [a]pyrene has been shown to cause genotoxic effects in a broad range of prokaryotic and mammalian cell assay systems (EPA 1991a).

The oral slope factor presented in the Region III Risk-Based Concentration Table is 7.3E+0 per mg/kg/day. The cancer slope factor for inhalation is not available.

F.7.6 BENZO(B)FLUORANTHENE

F.7.6.1 NONCANCER TOXICITY

Little information is available on benzo(b)fluoranthene. However based on the similarities of chemical structures, most properties should be similar to benzo(a)pyrene.

F.7.6.2 CARCINOGENICITY

A Toxicity Equivalency Factor (TEF) has been developed (EPA, 1993) for benzo(b)fluoranthene which allows the estimation of an oral CSF of 0.73 mg/g/day. The EPA (1998b) has classified benzo(b)fluoranthene in cancer weight-of-evidence Group B2 (Probable Human Carcinogen, sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans) based on lung tumors in mice.

F.7.7 COPPER

F.7.7.1 NONCANCER TOXICITY

Copper is a nutritionally essential element that functions as a cofactor in several enzyme systems (Aaseth and Norseth 1986). Acute exposure to large oral doses of copper salts was associated with GI disturbances, hemolysis, and liver and kidney lesions. Chronic oral toxicity in humans has not been reported. Chronic oral exposure of animals was associated with an iron-deficiency type of anemia, hemolysis, and lesions in the liver and kidneys. Occupational exposure may induce metal fume fever, and, in cases of chronic exposure to high levels, hemolysis and anemia (ACGIH 1991). Neither oral nor inhalation RfD or RfC values were located for copper. The target organs for copper are the erythrocyte, liver, and kidney, and, for inhalation exposure, the lung. An oral RfD of 0.04 mg/kg/day was presented for copper (EPA, 1997). A RfC value was not located for copper.

F.7.7.2 CARCINOGENICITY

Copper is classified in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) (EPA 1997). Quantitative risk estimates are not derived for Group D chemicals.

F.7.8 DIBENZO[A,H]ANTHRACENE

F.7.8.1 NONCANCER TOXICITY

The oral RfD and inhalation RfC are not available.

F.7.8.2 CARCINOGENICITY

Classification -- B2; probable human carcinogen

The EPA (1997) has classified dibenzo(a,h)anthracene in cancer weight-of-evidence group B2 (Probable Human Carcinogen, sufficient evidence of carcinogenicity in animals). Based on carcinomas in mice following oral or dermal exposure and injection site tumors in several species following subcutaneous or intramuscular administration. Dibenzo[a,h]anthracene has induced DNA damage and gene mutations in bacteria as well as gene mutations and transformation in several types of mammalian cell cultures.

Although there are no human data that specifically link exposure to dibenzo[a,h]anthracene with human cancers, dibenzo[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soot, coke oven emissions and cigarette smoke (EPA, 1984, 1990; IARC, 1984).

Dibenzo[a,h]anthracene has been shown to be carcinogenic when administered to mice by the oral route (Snell and Stewart, 1962, 1963) in a water-olive oil emulsion. Mice developed pulmonary adenomas, pulmonary carcinomas, and mammary carcinomas.

Dibenzo[a,h]anthracene has produced positive results in bacterial DNA damage and mutagenicity assays and in mammalian cell DNA damage, mutagenicity and cell transformation assays.

The currently used Oral Slope Factor (CSF) for Dibenzo[a,h]anthracene is 7.3E+00 per (mg/kg)/day which is extrapolated from the CSF for Benzo[a]pyrene i.e., 1.0×7.3 (BaP) = 7.3 per (mg/kg)/day (USEPA Region III Risk-Based Concentration Table, 4/1/98).

The inhalation Cancer Slope Factor for dibenzo(a,h)anthracene is not available.

F.7.9 1,1-DICHLOROETHENE

F.7.9.1 NONCANCER TOXICITY

Chronic oral exposure of laboratory animals to 1,1-dichloroethene induced liver effects (EPA 1998b). In animals, inhalation exposure induced degenerative changes in the liver and kidneys (ATSDR 1989b). No health effects were observed in a limited study of 138 exposed workers (ACGIH 1986). The EPA (1998b) presented a verified RfD for chronic oral exposure of 0.009 mg/kg/day, based on an NOAEL for liver effects in a chronic drinking water study in rats and an uncertainty factor of 1000. The EPA (1998b) presented the same value as a provisional subchronic oral RfD. The liver and kidneys are the target organs for exposure to 1,1-dichloroethene.

F.7.9.2 CARCINOGENICITY

EPA classified 1,1-dichloroethene as a cancer weight-of-evidence Group C compound (possible human carcinogen), based on an inadequate occupational exposure cancer study, limited data in several animal studies, its mutagenicity and ability to alkylate deoxyribonucleic acid (DNA), and its structural similarity to vinyl chloride, a known human carcinogen (EPA 1998b). The eighteen available animal studies (11 by inhalation exposure, 5 by oral exposure, and 1 each by dermal application and subcutaneous injection) were limited in sensitivity by various deficiencies in design. Credible evidence that 1,1-dichloroethene was a complete carcinogen was provided only by one 12-month inhalation study in mice, in which the incidence of kidney adenocarcinomas was significantly greater in the high-dose males than in the control males. A slope factor of 0.6 per mg/kg/day for oral exposure was based on the increase in incidence of adrenal pheochromocytomas in male rats treated by gavage for two years, even though the increase was not statistically significant (EPA, 1998b). A unit risk for inhalation exposure of 5.0×10^{-5} per mg/m³ was based on the incidence of kidney adenocarcinomas in male mice in the inhalation study mentioned above (EPA, 1998b). The unit risk is equivalent to 0.175 per mg/kg/day, assuming humans inhale 20 m³ of air/day and weigh 70 kg.

F.7.10 DIELDRIN

F.7.10.1 NONCANCER TOXICITY

The EPA (1998) derived a RfD of 5×10^{-5} mg/kg/day for chronic oral exposure based on a NOAEL of 0.005 mg/kg/day for liver lesions in a two-year rat feeding study (Walker et al., 1969) with an uncertainty factor of 100. The LOAEL was identified as 0.05 mg/kg/day.

At the end of two years the rats had increased liver weights and histopathological examinations revealed liver parenchymal cell changes. These hepatic lesions were considered to be characteristic of exposure to an organochlorine insecticide.

The chronic inhalation RfC is not available at this time.

F.7.10.2 CARCINOGENICITY

EPA (1997) classifies dieldrin in cancer weight-of-evidence B2. Dieldrin is carcinogenic in seven strains of mice when administered orally. Dieldrin is structurally related to compounds (aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid) which produce tumors in rodents.

Human carcinogenicity data is considered inadequate. Two studies of workers exposed to aldrin and to dieldrin reported no increased incidence of cancer. Both studies were limited in their ability to detect an excess of cancer deaths.

Animal carcinogenicity data was sufficient. Dieldrin has been shown to be carcinogenic in various strains of mice of both sexes. At different dose levels the effects range from benign liver tumors, to hepatocarcinomas with transplantation confirmation, to pulmonary metastases.

Supporting data for carcinogenicity include genotoxicity tests. Dieldrin causes chromosomal aberrations in mouse cells (Markaryan, 1966; Majumdar et al., 1976) and in human lymphoblastoid cells (Trepanier et al., 1977), mutation in Chinese hamster cells (Ahmed et al., 1977), and unscheduled DNA synthesis in rat (Probst et al., 1981) and human cells (Rocchi et al., 1980).

EPA (1998) reports an Oral Slope Factor of 16 per (mg/kg)/day based on a diet study in mice which produced liver carcinomas.

This inhalation cancer slope factor of 16 per mg/kg/day was calculated from the oral slope factor.

F.7.11 DIOXINS

Specific congeners and homologues of these classes of interest at this site include 1,2,3,4,6,7,8-heptachlorodibenzofuran and -heptachlorodibenzo-p-dioxin; 1,2,3,4,7,8,9-heptachlorodibenzofuran and -heptachlorodibenzo-p-dioxin; 1,2,3,4,7,8-hexachlorodibenzofuran and -hexachlorodibenzo-p-dioxin; 1,2,3,6,7,8- and 2,3,4,6,7,8-hexachlorodibenzofuran; 1,2,3,7,8,9-hexachlorodibenzofuran and -hexachlorodibenzo-p-dioxin; 1,2,3,6,7,8-hexachlorodibenzo-p-dioxin; unspecified hexachlorodibenzofurans and dibenzo-p-dioxins; 1,2,3,7,8- and 2,3,4,7,8-pentachlorodibenzofuran; unspecified pentachlorodibenzofurans; 2,3,7,8-tetrachlorodibenzofuran; and unspecified tetrachlorodibenzofurans.

F.7.11.1 NONCANCER TOXICITY

Of the members of these classes, the toxicity of 2,3,7,8-TCDD has been studied most extensively. The only effect in humans clearly attributable to 2,3,7,8-TCDD was chloracne (ATSDR 1989e). The data, however, also associated exposure to 2,3,7,8-TCDD with hepatotoxicity and neurotoxicity in humans. In animals, toxicity of 2,3,7,8-TCDD is most commonly manifested as a wasting syndrome with thymic atrophy, terminating in death, with a large number of organ systems showing nonspecific effects. Chronic treatment of animals with 2,3,7,8-TCDD or a mixture of two isomers of hexachlorodibenzo-p-dioxin resulted in liver damage. Immunologic effects may be among the more sensitive endpoints of exposure to the PCDDs in animals. In animals 2,3,7,8-TCDD is a developmental and reproductive toxicant. No verified or provisional noncancer toxicity values were located for any of the chemicals of interest in these classes (EPA 1994, 1992b).

F.7.11.3 CARCINOGENICITY

Data regarding the carcinogenicity of 2,3,7,8-TCDD to humans, obtained from epidemiologic studies of workers exposed to pesticides or to other chlorinated chemicals known to be contaminated with 2,3,7,8-TCDD, are conflicting (ATSDR 1989e). The interpretation of these studies is not clear because exposure to 2,3,7,8-TCDD was not quantified, multiple routes of exposure (dermal, inhalation, oral) were involved, and the workers were exposed to other potentially carcinogenic compounds. In animals, however, 2,3,7,8-TCDD is clearly carcinogenic, inducing thyroid, lung, and liver tumors in orally treated rats and mice (EPA 1985). Similarly, oral treatment with a mixture of two hexachlorodibenzo-p-dioxin isomers induced liver tumors in rats and mice. On the basis of the animal data, 2,3,7,8-TCDD and the hexachlorodibenzo-p-dioxins were assigned to EPA cancer weight-of-evidence Group B2 (probable human carcinogen). Although the other PCDDs and PCDFs were not formally classified as to carcinogenicity to humans, for regulatory purposes they are treated as probable human carcinogens.

The EPA (1993b) presents provisional oral and inhalation slope factors for 2,3,7,8-TCDD of 150,000 per mg/kg/day, based on the incidence of liver and lung tumors in an oral study in rats (Kociba et al. 1978).

Much less is known about the toxicity of other CDD and CDF congeners. Based on available toxicity data, EPA has developed a method for expressing toxicities of these compounds in terms of equivalent amounts of 2,3,7,8-TCDD. "Toxicity equivalency factors", or TEFs, are used to convert the concentration of a given CDD/CDF into an equivalent concentration of 2,3,7,8-TCDD.

F.7.12 HEPTACHLOR EPOXIDE (CLEMENT, 1985)

F.7.12.1 HEALTH EFFECTS

Heptachlor epoxide is a liver carcinogen when administered orally to mice. Results from mutagenicity bioassays suggest that this compound also may have genotoxic activity. Reproductive and teratogenic effects in rats include decreased litter size, shortened life span of suckling rats, and development of cataracts in offspring.

Tests with laboratory animals, primarily rodents, demonstrate acute and chronic toxic effects due to heptachlor exposure. Although heptachlor epoxide is absorbed most readily through the gastrointestinal tract, inhalation and skin contact are also potential routes of exposure. Acute exposure by various routes can cause development of hepatic vein thrombi and can affect the central nervous system and cause death. Chronic exposure induces liver changes, affects hepatic microsomal enzyme activity, and causes increased mortality in offspring. The oral LD₅₀ for heptachlor epoxide in the rat is 47 mg/kg.

Although there are reports of acute and chronic toxicity in humans, with symptoms including tremors, convulsions, kidney damage, respiratory collapse, and death, details of such episodes are not well documented. Heptachlor epoxide has been found in a high percentage of human adipose tissue samples, and also in human milk samples and biomagnification of heptachlor epoxide occurs. This compound also has been found in the tissues of stillborn infants, suggesting an ability to cross the placenta and bioaccumulate in the fetus.

The oral RfD for heptachlor epoxide is 1.30E-05 mg/kg-day based on increased liver to weight ratios in male and female dogs. Heptachlor epoxide is classified as a B2 carcinogen oral CSF for heptachlor epoxide is 9.1 per mg/kg-day based on an increased incidence of liver carcinomas. The inhalation CSF for heptachlor epoxide is 9.1 per mg/kg-day.

F.7.13 INDENO(1,2,3-CD)PYRENE

F.7.13.1 NONCANCER TOXICITY

Little information was found on the toxicity of indeno(1,2,3-cd)pyrene. Because of its structural similarity its properties should resemble benzo(a)pyrene.

F.7.13.2 CARCINOGENICITY

A Toxicity Equivalency Factor (TEF) has been developed for indeno(1,2,3-cd)pyrene (EPA 1993). This allows the estimation of an oral CSF of 0.73 mg/kg/day. The EPA (1998b) has classified indeno(1,2,3-cd)pyrene in cancer weight-of-evidence Group B2 (Probable Human Carcinogen, sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans) based on tumors in mice following lung implants.

F.7.14 LEAD

F.7.14.1 PHARMACOKINETICS

Studies in humans indicate that an average of 10 percent of ingested lead is absorbed, but estimates as high as 40 percent were obtained in some individuals (Tsuchiya, 1986). Nutritional factors have a profound effect on GI absorption efficiency. Children absorb ingested lead more efficiently than adults; absorption efficiencies up to 53 percent were recorded for children three months to eight years of age. Similar results were obtained for laboratory animals; absorption efficiencies of 5 to 10 percent were obtained for adults and > 50 percent were obtained for young animals. The deposition rate of inhaled lead averages approximately 30 to 50 percent, depending on particle size, with as much as 60 percent deposition of very small particles (0.03 mm) near highways. All lead deposited in the lungs is eventually absorbed.

Approximately 95 percent of the lead in the blood is located in the erythrocytes (EPA, 1998). Lead in the plasma exchanges with several body compartments, including the internal organs, bone, and several excretory pathways. In humans, lead concentrations in bone increase with age (Tsuchiya, 1986). About 90 percent of the body burden of lead is located in the skeleton. Neonatal blood concentrations are about 85 percent of maternal concentrations (EPA, 1998). Excretion of absorbed lead is principally through the urine, although GI secretion, biliary excretion, and loss through hair, nails, and sweat are also significant.

F.7.14.2 NONCANCER TOXICITY

The noncancer toxicity of lead to humans has been well characterized through decades of medical observation and scientific research (EPA, 1990). The principal effects of acute oral exposure are colic with diffuse paroxysmal abdominal pain (probably due to vagal irritation), anemia, and, in severe cases, acute encephalopathy, particularly in children (Tsuchiya, 1986). The primary effects of long-term exposure are neurological and hematological. Limited occupational data indicate that long-term exposure to lead may induce kidney damage. The principal target organs of lead toxicity are the erythrocyte and the nervous system. Some of the effects on the blood, particularly changes in levels of certain blood enzymes, and subtle neurobehavioral changes in children, appear to occur at levels so low as to be considered nonthreshold effects.

The USEPA (1990; July 1995) determined that it is inappropriate to derive an RfD for oral exposure to lead for several reasons. First, the use of an RfD assumes that a threshold for toxicity exists, below which adverse effects are not expected to occur; however, the most sensitive effects of lead exposure, impaired neurobehavioral development in children and altered blood enzyme levels associated with anemia, may occur at blood lead concentrations so low as to be considered practically nonthreshold in nature. Second,

RfD values are specific for the route of exposure for which they are derived. Lead, however, is ubiquitous, so that exposure occurs from virtually all media and by all pathways simultaneously, making it practically impossible to quantify the contribution to blood lead from any one route of exposure. Finally, the dose-response relationships common to many toxicants, and upon which derivation of an RfD is based, do not hold true for lead. This is because the fate of lead within the body depends, in part, on the amount and rate of previous exposures, the age of the recipient, and the rate of exposure. There is, however, a reasonably good correlation between blood lead concentration and effect. Therefore, blood lead concentration is the appropriate parameter on which to base the regulation of lead.

USEPA (1997) presented no inhalation RfC for lead, but referred to the National Ambient Air Quality Standard (NAAQS) for lead, which could be used in lieu of an inhalation RfC. The NAAQSs are based solely on human health considerations and are designed to protect the most sensitive subgroup of the human population. The NAAQS for lead is 1.5 mg/m³, averaged quarterly.

F.7.14.3 CARCINOGENICITY

USEPA (February 1998) classifies lead in cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate evidence of cancer in humans and sufficient animal evidence. The human data consist of several epidemiologic occupational studies that yielded confusing results. All of the studies lacked quantitative exposure data and failed to control for smoking and concomitant exposure to other possibly carcinogenic metals. Rat and mouse bioassays showed statistically significant increases in renal tumors following dietary and subcutaneous exposure to several soluble lead salts. Various lead compounds were observed to induce chromosomal alterations in vivo and in vitro, sister chromatid exchange in exposed workers, and cell transformation in Syrian hamster embryo cells; to enhance simian adenovirus induction; and to alter molecular processes that regulate gene expression. USEPA (July 1997) declined to estimate risk for oral exposure to lead because many factors (e.g., age, general health, nutritional status, existing body burden and duration of exposure) influence the bioavailability of ingested lead, introducing a great deal of uncertainty into any estimate of risk.

The USEPA IEUBK lead model is an iterated set of equations that estimate blood lead concentration in children aged 0 to 7 years (USEPA, February 1994). The biokinetic part of the model describes the movement of lead between the plasma and several body compartments and estimates the resultant blood lead concentration. The rate of the movement of lead between the plasma and each compartment is a function of the transition or residence time (i.e., the mean time for lead to leave the plasma and enter a given compartment, or the mean residence time for lead in that compartment). Compartments modeled include the erythrocytes, liver, kidneys, all the other soft tissue of the body, cortical bone, and trabecular bone. Excretory pathways and their rates are also modeled. These include the mean time for excretion

from the plasma to the urine, from the liver to the bile, and from the other soft tissues to the hair, skin, sweat, etc. The model permits the user to adjust the transition and residence times.

USEPA guidance (USEPA, July 1994) recommends using 400 mg/kg as a screening level for lead in soil for residential scenarios at CERCLA sites and at RCRA Corrective Action sites. Residential areas with soil lead below 400 mg/kg generally require no further action. However, in some special situations, further study is warranted below the screening level (e.g., wetlands, agricultural areas).

F.7.15 POLYAROMATIC HYDROCARBONS

PAHs are a large class of ubiquitous natural and anthropogenic chemicals, all with similar chemical structures (ATSDR 1990).

F.7.15.1 PHARMACOKINETICS

Although quantitative absorption data for the PAHs were not located, benzo(a)pyrene was readily absorbed across the GI (Rees et al. 1971) and respiratory epithelia (Kotin et al. 1969; Vainich et al. 1976). The high lipophilicity of other compounds in this class suggests that other PAHs also would be readily absorbed across GI and respiratory epithelia.

Benzo(a)pyrene was distributed widely in the tissues of treated rats and mice, but primarily to tissues high in fat, such as adipose tissue and mammary gland (Kotin et al. 1969; Schlede et al. 1970a). Patterns of tissue distribution of other PAHs would be expected to be similar because of the high lipophilicity of the members of this class.

Studies of the metabolism of benzo(a)pyrene provide information relevant to other PAHs because of the structural similarities of all members of the class. Metabolism involves microsomal mixed function oxidase hydroxylation of one or more of the phenyl rings with the formation of phenols and dihydrodiols, probably via formation of arene oxide intermediates (EPA 1979a). The dihydrodiols may be further oxidized to diol epoxides, which, for certain members of the class, are known to be the ultimate carcinogens (LaVoie et al. 1982). Conjugation with glutathione or glucuronic acid, and reduction to tetrahydrotetraols are important detoxification pathways. Metabolism of naphthalene resulted in the formation of 1,2-naphthoquinone, which induced cataract formation and retinal damage in rats and rabbits.

Excretion of benzo(a)pyrene or dibenzo(a,h)anthracene residues was reported to be rapid, although quantitative data were not located (EPA 1979b). Excretion occurred mainly via the feces, probably largely due to biliary secretion (Schlede et al. 1970a, 1970b). The EPA (1980a) concluded that accumulation in the body tissues of PAHs from chronic low level exposure would be unlikely.

F.7.15.2 NONCANCER TOXICITY

Oral noncancer toxicity data are available for acenaphthene, anthracene, fluoranthene, fluorene, and naphthalene. Newborn infants, children, and adults exposed to naphthalene by ingestion, inhalation, or possibly by skin contact developed hemolytic anemia with associated jaundice and occasionally renal disease (EPA 1979c). In a 13-week gavage study in rats, treatment with 50 mg naphthalene/kg, 5 days/week for 13 weeks (35.7 mg/kg/day) induced no effects; higher doses presumably reduced the

growth rate (National Toxicology Program (NTP) 1980). Application of an uncertainty factor of 1000 yielded a provisional RfD for chronic oral exposure of 0.04 mg/kg/day (EPA 1997). The very mild effect (decreased growth rate) apparently observed at higher doses suggests that the RfD is very conservatively protective.

F.7.15.3 CARCINOGENICITY

The PAHs are ubiquitous, being released to the environment from anthropogenic as well as from natural sources (ATSDR 1987). Benzo(a)pyrene is the most extensively studied member of the class, inducing tumors in multiple tissues of virtually all laboratory species tested by all routes of exposure. Although epidemiology studies suggested that complex mixtures that contain PAHs (coal tar, soots, coke oven emissions, cigarette smoke) are carcinogenic to humans (EPA 1994), the carcinogenicity cannot be attributed to PAHs alone because of the presence of other potentially carcinogenic substances in these mixtures (ATSDR 1987). In addition, recent investigations showed that the PAH fraction of roofing tar, cigarette smoke, and coke oven emissions accounted for only 0.1 to 8 percent of the total mutagenic activity of the unfractionated complex mixture in Salmonella (Lewtas 1988). Aromatic amines, nitrogen heterocyclic compounds, highly oxygenated quinones, diones, and nitrooxygenated compounds, none of which would be expected to arise from in vivo metabolism of PAHs, probably accounted for the majority of the mutagenicity of coke oven emissions and cigarette smoke. Furthermore, coal tar, which contains a mixture of many PAHs, has a long history of use in the clinical treatment of a variety of skin disorders in humans (ATSDR 1987).

Because of the lack of human cancer data, assignment of individual PAHs to EPA cancer weight-of-evidence groups was based largely on the results of animal studies with large doses of purified compound (EPA 1994). Frequently, unnatural routes of exposure, including implants of the test chemical in beeswax and trioctanoin in the lungs of female Osborne-Mendel rats, intratracheal instillation, and subcutaneous or intraperitoneal injection, were used. Benzo(a)anthracene, benzo(a)pyrene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene were classified in Group B2 (probable human carcinogens).

The EPA (1993a) verified a slope factor for oral exposure to benzo(a)pyrene of 7.3 per mg/kg/day, based on several dietary studies in mice and rats. Neither verified nor provisional quantitative risk estimates were available for the other PAHs in Group B2. The EPA (1980a) promulgated an ambient water quality criterion for "total carcinogenic PAHs," based on an oral slope factor derived from a study with benzo(a)pyrene, as being sufficiently protective for the class. Largely because of this precedent, the quantitative risk estimates for benzo(a)pyrene were adopted for the other carcinogenic PAHs when quantitative estimates were needed.

Recent reevaluations of the carcinogenicity and mutagenicity of the Group B2 PAHs suggest that there are large differences between individual PAHs in cancer potency (Krewski et al., 1989). Based on the available cancer and mutagenicity data, and assuming that there is a constant relative potency between different carcinogens across different bioassay systems and that the PAHs under consideration have similar dose-response curves, Thorslund and Charnley (1988) derived relative potency values for several PAHs. A more recent Relative Potency Factor (RPF) scheme for the Group B2 PAHs was based only on the induction of lung epidermoid carcinomas in female Osborne-Mendel rats in the lung-implantation experiments (Clement International 1990).

F.7.16 POLYCHLORINATED BIPHENYLS

F.7.16.1 NONCANCER TOXICITY

Epidemiologic studies of women in the United States associated oral PCB exposure with low birth weight or retarded musculoskeletal or neurobehavioral development of their infants (ATSDR 1991). Oral studies in animals established the liver as the target organ in all species, and the thyroid as an additional target organ in the rat. Effects observed in monkeys included gastritis, anemia, chloracne-like dermatitis, and immunosuppression. Oral treatment of animals induced developmental effects, including retarded neurobehavioral and learning development in monkeys. Oral RfD values of 0.02 ug/kg/day for Aroclor-1254 and 0.07 ug/kg/day for Aroclor-1016 were located.

Occupational exposure to PCBs was associated with upper respiratory tract and ocular irritation, loss of appetite, liver enlargement, increased serum concentrations of liver enzymes, skin irritation, rashes and chloracne, and, in heavily exposed female workers, decreased birth weight of their infants (ATSDR 1991). Concurrent exposure to other chemicals confounded the interpretation of the occupational exposure studies. Laboratory animals exposed by inhalation to Aroclor-1254 vapors exhibited moderate liver degeneration, decreased body weight gain and slight renal tubular degeneration. Neither subchronic nor chronic inhalation RfC values were available.

Target organs for PCBs include the skin, liver, fetus, and neonate.

F.7.16.2 CARCINOGENICITY

The EPA (1997) classifies the PCBs as EPA cancer weight-of-evidence Group B2 substances (probable human carcinogens), based on inadequate data in humans and sufficient data in animals. The human data consist of several epidemiologic occupational and accidental oral exposure studies with serious limitations, including poorly quantified concentrations of PCBs and durations of exposure, and probable exposures to other potential carcinogens.

The animal data consist of several oral studies in rats and mice with various aroclors, kanechlors, or clophens (commercial PCB mixtures manufactured in the United States, Japan and Germany, respectively) that reported increased incidence of liver tumors in both species (EPA 1994).

The EPA (1998) presents a verified oral slope factor and an inhalation slope factor of 2.0 per mg/kg/day for PCBs based on liver tumors in rats treated with Aroclor-1260.

F.7.17 VINYL CHLORIDE

F.7.17.1 NONCANCER TOXICITY

Data were not located regarding oral exposure of humans to vinyl chloride (ATSDR 1989i). In rats, lifetime dietary ingestion of vinyl chloride slightly but significantly increased mortality and induced mild histopathologic effects in the liver. Several early occupational studies associated vinyl chloride exposure with a syndrome known as vinyl chloride disease, which includes acroosteolysis (dissolution of the ends of the distal phalanges of the hands), circulatory disturbances in the extremities, Raynaud syndrome (sudden, recurrent bilateral cyanosis of the digits), scleroderma, hematologic effects, effects on the lungs, and impaired liver function and liver damage. Mild neurologic effects were also associated with occupational exposure. Long-term inhalation studies in rats and mice identified elevated relative liver weight as a sensitive indicator of liver effects. Neither inhalation RfC values nor oral RfD values for vinyl chloride were located. The principal target organs for vinyl chloride appear to be the CNS and the liver.

F.7.17.2 CARCINOGENICITY

The EPA (1997) lists vinyl chloride as an EPA cancer weight-of-evidence Group A compound (human carcinogen) and presents a verified oral slope factor of 1.9 per mg/kg/day, based on the increased incidence of liver and lung tumors in a lifetime dietary study in rats. An inhalation unit risk of 8.4E-05 per g/m³, equivalent to 0.3 per mg/kg/day, assuming humans inhale 20 m³ of air/day and weigh 70 kg, is based on liver tumors in rats intermittently exposed by inhalation for 12 months.

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Appendix F.8

Sample Calculations

CLIENT Rayman K - Ferry Creek	JOB NUMBER 4491
SUBJECT Incidental Ingestion of Soil - Commercial Worker (Surface Soil)	
BASED ON RAGs 1989	DRAWING NUMBER
BY KAC	CHECKED BY RJJ
APPROVED BY	DATE 9/23/99

Purpose: To calculate Noncarcinogenic and Carcinogenic risk for the Commercial Worker via incidental ingestion of site soil.

Relevant Equations:

$$Intake (mg/Kg/day) = \frac{C_s \times IR \times CF \times FI \times EF \times ED}{BW \times AT}$$

Where:

- C_s = Concentration in soil (mg/kg)
- IR = Soil Ingestion Rate (mg/day)
- CF = Conversion Factor (kg/mg)
- FI = Fraction of soil from contaminated source (unitless)
- EF = Exposure Frequency (days/year)
- ED = Exposure Duration (years)
- BW = Body Weight (kg)
- AT_N = Averaging Time for noncarcinogenic exposures (days)
- AT_C = Averaging Time for carcinogenic exposures (days)

	RM _E Reasonable Maximum Exposure	CT _E Central Tendency Exposure
C _s	Chemical specific	Chemical specific
IR	100 mg/day	50 mg/day
CF	1.0 x 10 ⁻⁶ kg/mg	1.0 x 10 ⁻⁶ kg/mg
FI	1 unitless	1 unitless
EF	250 days/year	250 days/year
ED	25 years	9 years
BW	70 kg	70 kg
AT _N	9125 days	3285 days
AT _C	25,550 days	25,550 days

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Incidental Ingestion of soil - Commercial Worker (surface soil)		
BASED ON	RAGS 1989	DRAWING NUMBER	
BY	KAC	CHECKED BY	RJJ
		APPROVED BY	
		DATE	9/23/99

Antake of Arsenic at a concentration of 21.9 mg/kg in surface soil at Area A1 under Reasonable Maximum Exposure assumptions.

$$\begin{aligned} \text{RME Noncarcinogenic Intake (mg/kg-day) of Arsenic} &= \frac{21.9 \text{ mg/kg} \times 100 \text{ mg/day} \times 1 \text{E-6 kg/mg} \times 1 \times 250 \text{ days/year} \times 25 \text{ years}}{70 \text{ kg} \times 9125 \text{ days}} \\ &= \boxed{2.14 \text{E-5 mg/kg}} \end{aligned}$$

$$\begin{aligned} \text{RME Carcinogenic Intake (mg/kg-day) of Arsenic} &= \frac{21.9 \text{ mg/kg} \times 100 \text{ mg/day} \times 1 \text{E-6 kg/mg} \times 1 \times 250 \text{ days/year} \times 25 \text{ years}}{70 \text{ kg} \times 25550 \text{ days}} \\ &= \boxed{7.65 \text{E-6 mg/kg}} \end{aligned}$$

Then Incremental Cancer Risk (ICR) and Hazard Quotient (HQ) are calculated via the following equations.

$$\text{ICR (unitless)} = \frac{\text{Carcinogenic Intake (mg/kg-day)}}{\text{kg/mg-day}} \times \text{Cancer Slope Factor (CSF)}$$

$$\text{HQ (unitless)} = \frac{\text{noncarcinogenic Intake (mg/kg-day)}}{\text{Reference Dose (RfD) (mg/kg-day)}}$$

where the CSF for Arsenic = 1.5E+0 and the RfD for Arsenic = 3E-4

$$\text{ICR Arsenic (unitless)} = 7.65 \text{E-6 mg/kg-day} \times 1.5 \text{E+0 kg/mg-day} = \boxed{1.1 \text{E-5}} \text{ under RME}$$

$$\text{HQ Arsenic (unitless)} = \frac{2.14 \text{E-5 mg/kg-day}}{3.0 \text{E-4 mg/kg-day}} = \boxed{7.1 \text{E-2}} \text{ under RME}$$

CLIENT	Raymark - Ferry Creek		JOB NUMBER	
SUBJECT	Accidental Ingestion of soil - Commercial Worker (surface soil)			
BASED ON	RAGs 1989		DRAWING NUMBER	
BY	KAC	CHECKED BY	RJJ	APPROVED BY
				DATE 9/23/99

Intake of Arsenic at a concentration of 8.1 mg/kg in Area A1 surface soil under the Central Tendency Exposure (CTE) assumptions.

CTE

$$\begin{aligned} \text{Noncarcinogenic Intake (mg/kg-day)} &= \frac{8.1 \text{ mg/kg} \times 50 \text{ mg/day} \times 1 \text{E-6 kg/mg} \times 1 \times 250 \text{ days/yr} \times 9 \text{ years}}{70 \text{ kg} \times 365 \text{ days}} \\ &= \boxed{4.0 \text{E-6 mg/kg-day}} \end{aligned}$$

CTE

$$\begin{aligned} \text{Carcinogenic Intake (mg/kg-day)} &= \frac{8.1 \text{ mg/kg} \times 50 \text{ mg/day} \times 1 \text{E-6 kg/mg} \times 1 \times 250 \text{ days/yr} \times 9 \text{ years}}{70 \text{ kg} \times 25550 \text{ days}} \\ &= \boxed{5.1 \text{E-7 mg/kg-day}} \end{aligned}$$

Then ICR and HQ are calculated for Arsenic where the

$$\text{ICR}_{\text{Arsenic}} = 5.1 \text{E-7 mg/kg-day} \times 1.5 \text{ kg/mg-day} = \boxed{7.6 \text{E-7}} \text{ CTE (unitless)}$$

$$\text{HQ}_{\text{Arsenic}} = \frac{4.0 \text{E-6 mg/kg-day}}{3.0 \text{E-4 mg/kg-day}} = \boxed{1.3 \text{E-2}} \text{ CTE (unitless)}$$

References

USEPA Dec. 1989. Risk Assessment Guidance for Superfund Volume 1 Human Health Evaluation Manual (Part A) Interim Final. OSWER EPA/540/1-89/002.

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Direct Dermal Contact with Soil - Commercial Worker (Surface Soil)		
BASED ON	RAGs 1989	DRAWING NUMBER	
BY	KAC	CHECKED BY	RJS
APPROVED BY		DATE	9/23/99

Purpose: To calculate carcinogenic and noncarcinogenic risk for the commercial worker via direct dermal contact with site soils.

Relevant Equation:

$$\text{Absorbed Dose} = \frac{C_s \times CF \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

($\mu\text{g}/\text{kg}\text{-day}$)

Where:

- C_s = Chemical Concentration in Soil ($\mu\text{g}/\text{kg}$)
 CF = Conversion Factor (kg/mg)
 SA = Skin Surface available for contact (cm^2/event)
 AF = Soil to Skin adherence factor (mg/cm^2)
 ABS = Absorption factor (unitless)
 EF = Exposure Frequency (events/year)
 ED = Exposure Duration (years)
 BW = Body Weight (kg)
 AT_N = Averaging Time for noncarcinogenic exposures (days)
 AT_C = Averaging Time for carcinogenic exposures (days)

	Reasonable Maximum Exposure (RME)	Central Tendency Exposure (CTE)
C_s	Chemical-specific $\mu\text{g}/\text{kg}$	Chemical-specific $\mu\text{g}/\text{kg}$
CF	$1E-6 \text{ kg}/\text{mg}$	$1E-6 \text{ kg}/\text{mg}$
SA	$2500 \text{ cm}^2/\text{event}$	$2500 \text{ cm}^2/\text{event}$
AF	$0.2 \text{ mg}/\text{cm}^2$	$0.02 \text{ mg}/\text{cm}^2$
ABS	Chemical-specific	Chemical specific
EF	$250 \text{ events}/\text{year}$	$250 \text{ events}/\text{year}$
ED	25 years	9 years
BW	70 kg	70 kg
AT_N	9125 days	3285 days
AT_C	25550 days	25550 days

CLIENT	Raymark - Ferry Creek		JOB NUMBER	7491
SUBJECT	Direct Dermal Contact with Soil - Commercial Worker (surface Area)			
BASED ON	RAGS 1989		DRAWING NUMBER	
BY	KAC	CHECKED BY	RJS	APPROVED BY
				DATE
				9/23/99

Absorbed Dose of arsenic at a concentration of 21.9 mg/kg in Area A1 surface soil under Reasonable Maximum Exposure (RME) assumptions is calculated:

$$\begin{aligned} \text{RME Noncancer} &= \frac{21.9 \text{ mg/kg} \times 2500 \frac{\text{cm}^2}{\text{event}} \times 0.2 \frac{\text{m}^2}{\text{cm}^2} \times 0.03 \times 250 \frac{\text{event}}{\text{yr}} \times 25 \text{ years} \times 1 \text{E-6} \frac{\text{kg}}{\text{m}^3}}{70 \text{ kg} \times 9125 \text{ days}} \\ \text{Absorbed Dose} & \\ (\text{mg/kg-day}) & \\ &= \boxed{3.21 \text{E-6 mg/kg-day RME}} \end{aligned}$$

$$\begin{aligned} \text{RME Carcinog.} &= \frac{21.9 \text{ mg/kg} \times 1 \text{E-6} \frac{\text{kg}}{\text{m}^3} \times 2500 \frac{\text{cm}^2}{\text{event}} \times 0.2 \frac{\text{m}^2}{\text{cm}^2} \times 0.03 \times 250 \frac{\text{events}}{\text{year}} \times 25 \text{ years}}{70 \text{ kg} \times 25550 \text{ days}} \\ \text{Absorbed Dose} & \\ (\text{mg/kg-day}) & \\ &= \boxed{1.15 \text{E-6 mg/kg-day RME}} \end{aligned}$$

Then Incremental Cancer Risk (ICR) and Hazard Quotient (HQ) are calculated via the following equations:

$$\text{ICR} = \frac{\text{Carcinogenic Absorbed Dose (mg/kg-day)}}{(\text{unitless})} \times \frac{\text{Cancer Slope Factor (CSF)}}{\text{kg/mg-day}}$$

$$\text{HQ} = \frac{\text{noncarcinogenic Absorbed Dose (mg/kg-day)}}{(\text{unitless})} \times \frac{\text{Reference Dose (RfD) (mg/kg-day)}}{(\text{mg/kg-day})}$$

Where the CSF of Arsenic = $1.5 \text{E}+0$ and the RfD = 3.0E-4

$$\text{ICR Arsenic} = 1.15 \text{E-6 mg/kg-day} \times 1.5 \text{E}+0 \frac{\text{kg}}{\text{mg-day}} = \boxed{1.72 \text{E-6 RME}} \quad (\text{unitless})$$

$$\text{HQ Arsenic} = \frac{3.21 \text{E-6 mg/kg-day}}{3.0 \text{E-4 mg/kg-day}} = \boxed{1.1 \text{E-2 RME}} \quad (\text{unitless})$$

CLIENT Raymark - Ferry Creek	JOB NUMBER 7491		
SUBJECT Direct Dermal Contact with Soil - Commercial Worker (surface soil)			
BASED ON RAGS 1989		DRAWING NUMBER	
BY KAC	CHECKED BY RJS	APPROVED BY	DATE 9/23/99

Absorbed dose of Arsenic at a concentration of 8.1 mg/kg in Area A1 surface soil under Central Tendency Exposure (CTE) assumptions is calculated:

$$\text{CTE Noncanc.} = \frac{8.1 \frac{\text{mg}}{\text{kg}} \times 1 \times 10^{-6} \frac{\text{kg}}{\text{m}^2} \times 2500 \frac{\text{cm}^2}{\text{event}} \times 0.02 \frac{\text{m}^2}{\text{cm}^2} \times 0.03 \times 250 \frac{\text{events}}{\text{yr}} \times 9 \text{ years}}{70 \text{ kg} \times 3285 \text{ days}}$$

Absorbed Dose (mg/kg-day)

$$= \boxed{1.19 \times 10^{-7} \text{ mg/kg-day CTE}} \quad \checkmark$$

$$\text{CTE Canc.} = \frac{8.1 \frac{\text{mg}}{\text{kg}} \times 1 \times 10^{-6} \frac{\text{kg}}{\text{m}^2} \times 2500 \frac{\text{cm}^2}{\text{event}} \times 0.02 \frac{\text{m}^2}{\text{cm}^2} \times 0.03 \times 250 \frac{\text{event}}{\text{yr}} \times 9 \text{ yrs}}{70 \text{ kg} \times 25550 \text{ days}}$$

Absorbed Dose (mg/kg-day)

$$= \boxed{1.53 \times 10^{-8} \text{ mg/kg-day CTE}} \quad \checkmark$$

$$\text{ICR Arsenic CTE} = \frac{1.53 \times 10^{-8} \text{ (mg/kg-day)}}{1 \text{ (unitless)}} \times \frac{1.5 \times 10^{-6} \text{ (kg/mg-day)}}{1 \text{ (unitless)}} = \boxed{2.3 \times 10^{-8} \text{ CTE}} \quad \checkmark$$

$$\text{HQ Arsenic CTE} = \frac{1.19 \times 10^{-7} \text{ mg/kg-day}}{3.0 \times 10^{-4} \text{ mg/kg-day}} = \boxed{4.0 \times 10^{-4} \text{ CTE}} \quad \checkmark$$

Reference

USEPA 1989, Risk Assessment Guidance for Superfund Volume 1 Human Health Evaluation manual (Part A) Interim Final, OSWER EPA 540/1-89/002.

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Incidental Ingestion of Soil - Frequent Recreational User (Adult)		
BASED ON	KAGs 1989	DRAWING NUMBER	
BY	KAC	CHECKED BY	RJJ
		APPROVED BY	
		DATE	9/23/99

Purpose: To calculate carcinogenic and noncarcinogenic risk for the adult frequent recreational user via incidental ingestion of soil

Relevant Equation:

$$\text{Intake} = \frac{C_s \times IR \times CF \times FI \times EF \times ED}{BW \times AT}$$

mg/Kg-day

Where:

- C_s = Concentration in soil (mg/Kg)
- IR = Soil Ingestion Rate (mg/day)
- CF = Conversion Factor (Kg/mg)
- FI = Fraction ingested from contaminated source (unitless)
- EF = Exposure Frequency (days/year)
- ED = Exposure Duration (years)
- BW = Body Weight (Kg)
- AT_N = Averaging Time for noncarcinogenic exposures (days)
- AT_C = Averaging Time for carcinogenic exposures (days)

	Reasonable Maximum Exposure (RME)	Central Tendency Exposure (CTE)
C_s	Chemical specific (mg/Kg)	Chemical specific (mg/Kg)
IR	100 mg/day	50 mg/day
CF	$1E-6$ Kg/mg	$1E-6$ Kg/mg
FI	1 unitless	1 unitless
EF	150 days/year	150 days/year
ED	24 years	7 years
BW	70 Kg	70 Kg
AT_N	8760 days	2555 days
AT_C	25550 days	25550 days

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Accidental Ingestion of Soil - Frequent Recreational User Adult		
BASED ON	RAG 1989	DRAWING NUMBER	
BY	KAC	CHECKED BY	RJJ
		APPROVED BY	
		DATE	9/23/99

Intake of Arsenic at a concentration of 28.1 mg/kg in Area A-1 surface soil under Reasonable Maximum Exposure assumption

$$\begin{aligned} \text{RME Noncarcinog} &= \frac{28.1 \frac{\text{mg}}{\text{kg}} \times 100 \frac{\text{mg}}{\text{day}} \times 1\text{E-}6 \frac{\text{kg}}{\text{mg}} \times 1 \times 150 \frac{\text{days}}{\text{year}} \times 24 \text{ years}}{70 \text{ kg} \times 8760 \text{ days}} \\ \text{Intake of Arsenic} & \\ (\text{mg/kg-day}) & \\ &= \boxed{1.6\text{E-}5 \text{ mg/kg-day RME}} \end{aligned}$$

$$\begin{aligned} \text{RME Carcinogenic} &= \frac{28.1 \text{ mg/kg} \times 100 \text{ mg/day} \times 1\text{E-}6 \frac{\text{kg}}{\text{mg}} \times 150 \frac{\text{days}}{\text{year}} \times 24 \text{ years}}{70 \text{ kg} \times 25550 \text{ days}} \\ \text{Intake of Arsenic} & \\ (\text{mg/kg-day}) & \\ &= \boxed{5.7\text{E-}6 \text{ mg/kg-day RME}} \end{aligned}$$

Then Incremental Cancer Risk (ICR) and Hazard Quotient (HQ) are calculated via the following equations:

$$\text{ICR} = \frac{\text{Carcinogenic Intake (mg/kg-day)}}{\text{Cancer Slope Factor (CSF) (kg/mg-day)}} \quad (\text{unitless})$$

$$\text{HQ} = \frac{\text{noncarcinogenic Intake (mg/kg-day)}}{\text{Reference Dose (RFD) (mg/kg-day)}} \quad (\text{unitless})$$

where the CSF for Arsenic = 1.5E+0 and the RFD = 3.0E-4

$$\text{ICR Arsenic RME} = 5.7\text{E-}6 \text{ mg/kg-day} \times 1.5\text{E+}0 \text{ kg/mg-day} = \boxed{8.5\text{E-}6 \text{ RME}} \quad (\text{unitless})$$

$$\text{HQ Arsenic RME} = \frac{1.6\text{E-}5 \text{ mg/kg-day}}{3\text{E-}4 \text{ mg/kg-day}} = \boxed{5.3\text{E-}2 \text{ RME}} \quad (\text{unitless})$$

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Accidental Ingestion of soil - Frequent Recreational User - Adult		
BASED ON	RAGs 1989	DRAWING NUMBER	
BY	KAC	CHECKED BY	RJJ
APPROVED BY		DATE	9/23/99

Intake of Arsenic at a concentration of 9 mg/kg in surface soil from Area A1 under central tendency exposures (CTE)

$$\begin{aligned} \text{CTE Noncancer Intake (mg/kg-day)} &= \frac{9 \frac{\text{mg}}{\text{kg}} \times 50 \frac{\text{mg}}{\text{day}} \times 1 \text{E-6} \frac{\text{kg}}{\text{mg}} \times 1 \times 150 \frac{\text{days}}{\text{year}} \times 7 \text{ years}}{70 \text{ kg} \times 2555 \text{ days}} \\ &= \boxed{2.6 \text{E-6 mg/kg CTE}} \end{aligned}$$

$$\begin{aligned} \text{CTE Cancer Intake (mg/kg-day)} &= \frac{9 \frac{\text{mg}}{\text{kg}} \times 50 \frac{\text{mg}}{\text{day}} \times 1 \text{E-6} \frac{\text{kg}}{\text{mg}} \times 1 \times 150 \frac{\text{days}}{\text{year}} \times 7 \text{ years}}{70 \text{ kg} \times 25550 \text{ days}} \\ &= \boxed{2.6 \text{E-7 mg/kg CTE}} \end{aligned}$$

and the ICR and HQ for CTE exposures to Arsenic in soil

$$\text{ICR Arsenic CTE} = 2.6 \text{E-7 mg/kg-day} \times 1.5 \text{ kg/mg/day} = \boxed{4.0 \text{E-7 CTE}} \text{ (unitless)}$$

$$\text{HQ Arsenic CTE} = \frac{2.6 \text{E-6 mg/kg-day}}{3 \text{E-4 mg/kg-day}} = \boxed{8.7 \text{E-3 CTE}} \text{ (unitless)}$$

Reference

USEPA Dec. 1989, Risk Assessment Guidance for Superfund Volume 1 Human Health Evaluation Manual (Part A) Interim Final, OSWER EPA/540/1-89/002.

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Direct Dermal Contact with soil - Frequent Recreational		
BASED ON	RAGS 1989	DRAWING NUMBER	User - adult
BY	KAC	CHECKED BY	RJJ
		APPROVED BY	
		DATE	9/23/99

Purpose: To calculate carcinogenic and noncarcinogenic risks for the frequent recreational user (adult) via direct dermal contact with site soils.

Relevant Equation:

$$\text{Absorbed Dose} = \frac{C_s \times CF \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

(mg/kg-day)

Where:

- C_s = Chemical concentration in soil (mg/kg)
 CF = Conversion Factor (kg/mg)
 SA = Skin surface available for contact (cm²/event)
 AF = Soil to skin adherence factor (mg/cm²)
 ABS = Absorption factor (unitless)
 EF = Exposure frequency (events/year)
 ED = Exposure Duration (years)
 BW = Body Weight (kg)
 AT_N = Averaging time for noncarcinogens (days)
 AT_C = Averaging time for carcinogens (days)

	Reasonable Maximum Exposure	Central Tendency Exposure
C_s	chemical specific mg/kg	chemical specific mg/kg
CF	1E-6 kg/mg	1E-6 kg/mg
SA	5700 cm ² /event	5700 cm ² /event
AF	0.07 mg/cm ²	0.01 mg/cm ²
ABS	chemical specific	chemical-specific
EF	150 events/year	150 events/year
ED	34 years	7 years
BW	70 kg	70 kg
AT_N	8760 days	2555 days
AT_C	25550 days	25550 days

CLIENT	Raymark Ferry Creek	JOB NUMBER	7491
SUBJECT	Direct Dermal Contact with Soil - Frequent Recreational		
BASED ON	RAGs 1989	DRAWING NUMBER	User - Adult
BY	KAC	CHECKED BY	RJS
		APPROVED BY	
		DATE	9/23/99

Absorbed dose of arsenic at a concentration of 28.1 mg/kg in Area A1 surface soil under the Reasonable Maximum Exposure (RME) assumptions.

$$\begin{aligned} \text{RME Noncarc.} &= \frac{28.1 \text{ mg/kg} \times 5700 \frac{\text{cm}^2}{\text{event}} \times 1 \text{E-}6 \frac{\text{kg}}{\text{m}^2} \times 0.07 \frac{\text{m}^2}{\text{cm}^2} \times 0.03 \times 150 \frac{\text{event}}{\text{yr}} \times 24 \text{ yr}}{70 \text{ Kg} \times 8760 \text{ days}} \\ \text{Absorbed Dose} & \\ \text{(mg/kg-day)} & \\ &= \boxed{1.97 \text{E-}6 \text{ mg/kg-day RME}} \end{aligned}$$

$$\begin{aligned} \text{RME Carc.} &= \frac{28.1 \text{ mg/kg} \times 5700 \frac{\text{cm}^2}{\text{event}} \times 1 \text{E-}6 \frac{\text{kg}}{\text{m}^2} \times 0.07 \frac{\text{m}^2}{\text{cm}^2} \times 0.03 \times 150 \frac{\text{event}}{\text{yr}} \times 24 \text{ years}}{70 \text{ Kg} \times 25550 \text{ days}} \\ \text{Absorbed Dose} & \\ \text{(mg/kg-day)} & \\ &= \boxed{6.77 \text{E-}7 \text{ mg/kg-day RME}} \end{aligned}$$

Then Incremental Cancer Risk (ICR) and Hazard Quotient (HQ) are calculated via the following equations:

$$\text{ICR (unitless)} = \frac{\text{Carcinogenic absorbed dose (mg/kg-day)}}{\text{mg/kg-day}} \times \frac{\text{Cancer Slope Factor (CSF)}}{\text{Kg/mg-day}}$$

$$\text{HQ (unitless)} = \frac{\text{noncarcinogenic absorbed dose (mg/kg-day)}}{\text{Reference Dose (RFD) (mg/kg-day)}}$$

Where the CSF for arsenic = 1.5E+0 and the RFD = 3.0E-4

$$\text{ICR Arsenic RME} = \frac{6.77 \text{E-}7 \text{ mg/kg-day}}{\text{mg/kg-day}} \times \frac{1.5}{\text{Kg/mg-day}} = \boxed{1.0 \text{E-}6 \text{ RME}}$$

$$\text{HQ Arsenic RME} = \frac{1.97 \text{E-}6 \text{ mg/kg-day}}{3.0 \text{E-}4 \text{ mg/kg-day}} = \boxed{6.6 \text{E-}3 \text{ RME}}$$

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Direct Dermal Contact with soil - Frequent Recreational		
BASED ON	RAG 1984	DRAWING NUMBER	User - adult
BY	KAC	CHECKED BY	RJS
		APPROVED BY	
		DATE	9/23/99

Absorbed dose of Arsenic at a concentration of 9 mg/kg in Area A-1 surface soil under the Central Tendency Exposure (CTE) Assumptions:

$$\text{CTE Noncancer Absorbed Dose (mg/kg-day)} = \frac{9 \text{ mg/kg} \times 1 \text{E-6} \frac{\text{kg}}{\text{mg}} \times 5700 \frac{\text{cm}^2}{\text{event}} \times 0.01 \frac{\text{mg}}{\text{cm}^2} \times 0.03 \times 150 \frac{\text{events}}{\text{year}} \times 7 \text{ years}}{70 \text{ kg} \times 2555 \text{ days}}$$

$$= \boxed{9.04 \text{E-8 mg/kg-day CTE}}$$

$$\text{CTE Cancer Absorbed Dose (mg/kg-day)} = \frac{9 \frac{\text{mg}}{\text{kg}} \times 1 \text{E-6} \frac{\text{kg}}{\text{mg}} \times 5700 \frac{\text{cm}^2}{\text{event}} \times 0.01 \frac{\text{mg}}{\text{cm}^2} \times 0.03 \times 150 \frac{\text{event}}{\text{year}} \times 7 \text{ years}}{70 \text{ kg} \times 25550}$$

$$= \boxed{9.04 \text{E-9 mg/kg-day CTE}}$$

The ICR and HQ for Arsenic is then calculated

$$\text{ICR Arsenic CTE} = \frac{9.04 \text{E-9 mg/kg-day}}{1 \text{E-9 mg/kg-day}} \times 1.5 = \boxed{1.36 \text{E-8 CTE}}$$

unitless

$$\text{HQ Arsenic CTE} = \frac{9.04 \text{E-8 mg/kg-day}}{3 \text{E-4 mg/kg-day}} = \boxed{3.0 \text{E-4 CTE}}$$

unitless

Reference

USEPA 1989. Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual (Part A) Interim Final. OSWER EPA 546/1-89/002.

CLIENT	Raymark - Ferry Creek		JOB NUMBER	1491
SUBJECT	Accidental Ingestion of Soil - Frequent Recreational User - Child			
BASED ON	RAGS 1989		DRAWING NUMBER	
BY	KAC	CHECKED BY	RJA	APPROVED BY
				DATE 9/23/99

Purpose: To calculate carcinogenic and noncarcinogenic risk for the frequent recreational user - child via the ingestion of site soil.

Relevant Equations:

$$\text{Intake} = \frac{C_s \times IR \times CF \times FI \times EF \times ED}{BW \times AT}$$

(mg/kg-day)

Where:

- C_s = Chemical concentration in soil (mg/kg)
- IR = Soil Ingestion Rate (mg/day)
- CF = Conversion Factor (kg/mg)
- FI = Fraction ingested from contaminated source (unitless)
- EF = Exposure frequency (days/year)
- ED = Exposure Duration (years)
- BW = Body Weight (kg)
- AT_N = Averaging Time for noncarcinogenic exposures (days)
- AT_C = Averaging time for carcinogenic exposures (days)

	Reasonable Maximum Exposure	Central Tendency Exposure
C_s	chemical-specific mg/kg	chemical-specific mg/kg
IR	200 mg/day	100 mg/day
CF	$1E-6$ kg/mg	$1E-6$ kg/mg
FI	1 unitless	1 unitless
EF	150 days/year	150 days/year
ED	6 years	2 years
BW	15 kg	15 kg
AT_N	2190 days	730 days
AT_C	25550 days	25550 days

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Accidental Ingestion of Soil Frequent Recreational User - Child		
BASED ON	RAGs 1989	DRAWING NUMBER	
BY	KAC	CHECKED BY	RJA
		APPROVED BY	
		DATE	9/23/99

Intake of Arsenic at a concentration of 28.1 mg/kg in Area A-1 surface soil under Reasonable Maximum Exposure (RME) assumptions.

$$\begin{aligned} \text{RME Noncancer} &= \frac{28.1 \frac{\text{mg}}{\text{kg}} \times 200 \frac{\text{mg}}{\text{day}} \times 10^{-6} \frac{\text{kg}}{\text{mg}} \times 1 \text{ unitless} \times 150 \frac{\text{days}}{\text{year}} \times 6 \text{ years}}{15 \text{ kg} \times 2190 \text{ days}} \\ \text{Intake (mg/kg-day)} &= \boxed{1.5 \text{E-4 mg/kg-day RME}} \quad \checkmark \end{aligned}$$

$$\begin{aligned} \text{RME Cancer} &= \frac{28.1 \frac{\text{mg}}{\text{kg}} \times 200 \frac{\text{mg}}{\text{day}} \times 10^{-6} \frac{\text{kg}}{\text{mg}} \times 1 \text{ unitless} \times 150 \frac{\text{days}}{\text{year}} \times 6 \text{ years}}{15 \text{ kg} \times 25550 \text{ days}} \\ \text{Intake (mg/kg-day)} &= \boxed{1.3 \text{E-5 mg/kg-day RME}} \quad \checkmark \end{aligned}$$

Then Incremental Cancer Risk (ICR) and Hazard Quotient (HQ) are calculated via the following equations:

$$\text{ICR} = \frac{\text{Carcinogenic Intake (mg/kg-day)}}{\text{unitless}} \times \frac{\text{Cancer Slope Factor (CSF) (kg/mg-day)}}{\text{unitless}}$$

$$\text{HQ} = \frac{\text{noncarcinogenic Intake (mg/kg-day)}}{\text{unitless}} \times \frac{\text{Reference Dose (RFD) (mg/kg-day)}}{\text{unitless}}$$

Where the CSF for Arsenic = $1.5 \text{E}+0$ and the RFD = 3.0E-4

$$\text{ICR Arsenic RME} = \frac{1.3 \text{E-5 (mg/kg-day)}}{\text{unitless}} \times \frac{1.5 \text{ (kg/mg-day)}}{\text{unitless}} = \boxed{1.95 \text{E-5 RME}} \quad \checkmark$$

$$\text{HQ Arsenic RME} = \frac{1.5 \text{E-4 mg/kg-day}}{3 \text{E-4 mg/kg-day}} = \boxed{5.0 \text{E-1 RME}} \quad \checkmark$$

CLIENT	Raymark - Ferry Creek		JOB NUMBER	7491
SUBJECT	Accidental Ingestion of soil - Frequent Recreational User - child			
BASED ON	RAGS 1989		DRAWING NUMBER	
BY	KAC	CHECKED BY	RJJ	APPROVED BY
				DATE 9/23/99

Intake of arsenic at a concentration of 9 mg/kg in Area A-1 surface soils under central tendency exposure (CTE) assumptions

$$\text{CTE Noncarc. Intake (mg/kg-day)} = \frac{9 \frac{\text{mg}}{\text{kg}} \times 100 \frac{\text{mg}}{\text{day}} \times 1 \text{E-6} \frac{\text{kg}}{\text{mg}} \times 1 \times 150 \frac{\text{day}}{\text{year}} \times 2 \text{ years}}{15 \text{ kg} \times 730 \text{ days}}$$

$$= \boxed{2.5 \text{E-5 CTE mg/kg-day}} \quad \checkmark$$

$$\text{CTE Carc. Intake (mg/kg-day)} = \frac{9 \frac{\text{mg}}{\text{kg}} \times 100 \frac{\text{mg}}{\text{day}} \times 1 \text{E-6} \frac{\text{kg}}{\text{mg}} \times 1 \times 150 \frac{\text{day}}{\text{year}} \times 2 \text{ years}}{15 \text{ kg} \times 25550 \text{ days}}$$

$$= \boxed{7.0 \text{E-7 CTE mg/kg-day}} \quad \checkmark$$

Then ICR and HQ are calculated for arsenic CTE.

$$\text{ICR CTE Arsenic} = \frac{7.0 \text{E-7 CTE} \times 1.5}{\text{unitless mg/kg-day kg/mg-day}} = \boxed{1.0 \text{E-6 CTE}} \quad \checkmark$$

$$\text{HQ CTE Arsenic} = \frac{2.5 \text{E-5 mg/kg-day}}{3.0 \text{E-4 mg/kg-day}} = \boxed{8.3 \text{E-2 CTE}} \quad \checkmark$$

Reference

USEPA Dec. 1989. Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual (Part A) Interim Final. OSWER EPA 540/1-89/002.

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Direct Dermal Contact with Soil - Frequent Recreational User		
BASED ON	RAGS 1989	DRAWING NUMBER	-Child
BY	KAC	CHECKED BY	RJJ
APPROVED BY		DATE	9/24/99

Purpose: To calculate carcinogenic and noncarcinogenic risk for the child frequent recreational user for exposure via direct dermal contact with soil.

Relevant Equation:

$$\text{Absorbed Dose} = \frac{C_s \times CF \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

mg/kg-day

Where:

- C_s = Chemical Concentration in soil (mg/kg)
 CF = Conversion Factor (kg/mg)
 SA = Skin surface area available for contact (cm²/event)
 AF = Soil to Skin adherence factor (mg/cm²)
 ABS = Absorption factor (unitless)
 EF = Exposure Frequency (events/year)
 ED = Exposure Duration (years)
 BW = Body Weight (kg)
 AT_N = Averaging Time for noncarcinogenic exposures (days)
 AT_C = Averaging Time for carcinogenic exposures (days)

	Reasonable Maximum Exposure	Central Tendency Exposure
C_s	Chemical specific mg/kg	Chemical specific mg/kg
CF	1E-6 kg/mg	1E-6 kg/mg
SA	2900 cm ² /event	2900 cm ² /event
AF	0.2 mg/cm ²	0.06 mg/cm ²
ABS	Chemical specific unitless	Chemical specific unitless
EF	150 events/year	150 events/year
ED	6 years	2 years
BW	15 kg	15 kg
AT_N	2190 days	730 days
AT_C	2190 days	730 days

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Direct Dermal Contact with Soil - Frequent Recreational User		
BASED ON	RAGs 1989	DRAWING NUMBER	- Child
BY	KAC	CHECKED BY	RJS
		APPROVED BY	DATE 9/24/99

The absorbed dose of arsenic at a concentration of 28.1 mg/kg for Area A1 surface soils under the Reasonable Maximum Exposure (RME) assumptions is calculated:

$$\begin{aligned} \text{RME Noncarc. Absorbed Dose} &= \frac{28.1 \frac{\text{mg}}{\text{kg}} \times 10^{-6} \frac{\text{kg}}{\text{m}^2} \times 2900 \frac{\text{cm}^2}{\text{event}} \times 0.2 \frac{\text{mg}}{\text{cm}^2} \times 0.03 \times 150 \frac{\text{event}}{\text{yr}} \times 6 \text{ years}}{15 \text{ kg} \times 2190 \text{ days}} \\ &= \boxed{1.34 \text{E-}5 \text{ mg/kg-day RME}} \checkmark \end{aligned}$$

$$\begin{aligned} \text{RME Carc. Absorbed Dose} &= \frac{28.1 \frac{\text{mg}}{\text{kg}} \times 10^{-6} \frac{\text{kg}}{\text{m}^2} \times 2900 \frac{\text{cm}^2}{\text{event}} \times 0.2 \frac{\text{mg}}{\text{cm}^2} \times 0.03 \times 150 \frac{\text{event}}{\text{yr}} \times 6 \text{ years}}{15 \text{ kg} \times 25550 \text{ days}} \\ &= \boxed{1.15 \text{E-}6 \text{ mg/kg-day RME}} \checkmark \end{aligned}$$

Then Incremental Cancer Risk (ICR) and Hazard Quotient (HQ) are calculated via the following equations:

$$\text{ICR (unitless)} = \frac{\text{Carcinogenic Absorbed Dose (mg/kg-day)} \times \text{Cancer Slope Factor (CSF) (kg/mg-day)}$$

$$\text{HQ (unitless)} = \frac{\text{Noncarcinogenic Absorbed Dose (mg/kg-day)}}{\text{Reference Dose (RfD) (mg/kg-day)}}$$

where the CSF for Arsenic = 1.5 and the RfD = 3.0E-4

$$\text{RME ICR Arsenic (unitless)} = \frac{1.15 \text{E-}6 \text{ mg/kg-day} \times 1.5 \text{ (kg/mg-day)}}{1 \text{ mg/kg-day}} = \boxed{1.73 \text{E-}6 \text{ RME}} \checkmark$$

$$\text{RME HQ Arsenic (unitless)} = \frac{1.34 \text{E-}5 \text{ mg/kg-day}}{3.0 \text{E-}4 \text{ mg/kg-day}} = \boxed{4.5 \text{E-}2 \text{ RME}} \checkmark$$

CLIENT	Raymark - Ferry Creek	JOB NUMBER	4491
SUBJECT	Direct Dermal Contact with Soil - Frequent Recreational Use - Child		
BASED ON	RAGs 1989	DRAWING NUMBER	
BY	KAC	CHECKED BY	RJS
APPROVED BY		DATE	9/24/99

The absorbed dose of arsenic at a concentration of 9 mg/kg for direct A1 surface soil under the central tendency exposure (CTE) assumptions is calculated:

$$\begin{aligned} \text{CTE Noncarc. Absorbed Dose (mg/kg-day)} &= \frac{9 \frac{\text{mg}}{\text{kg}} \times 1 \text{E-}6 \frac{\text{kg}}{\text{mg}} \times 2900 \frac{\text{cm}^2}{\text{event}} \times 0.06 \frac{\text{mg}}{\text{cm}^2} \times 0.03 \times 150 \frac{\text{event}}{\text{yr}} \times 2 \text{yr}}{15 \text{ kg} \times 730 \text{ days}} \\ &= \boxed{1.29 \text{E-}6 \text{ mg/kg-day CTE}} \quad \checkmark \end{aligned}$$

$$\begin{aligned} \text{CTE Carcin. Absorbed Dose (mg/kg-day)} &= \frac{9 \frac{\text{mg}}{\text{kg}} \times 1 \text{E-}6 \frac{\text{kg}}{\text{mg}} \times 2900 \frac{\text{cm}^2}{\text{event}} \times 0.06 \frac{\text{mg}}{\text{cm}^2} \times 0.03 \times 150 \frac{\text{event}}{\text{yr}} \times 2 \text{ year}}{15 \text{ kg} \times 25550 \text{ days}} \\ &= \boxed{3.68 \text{E-}8 \text{ mg/kg-day CTE}} \quad \checkmark \end{aligned}$$

Then ICR and HQ are calculated for arsenic:

$$\begin{aligned} \text{ICR CTE Arsenic (unitless)} &= \frac{3.68 \text{E-}8 \text{ (mg/kg-day)}}{3.0 \text{E-}4 \text{ (kg/mg-day)}} \times 1.5 = \boxed{5.5 \text{E-}8 \text{ CTE}} \quad \checkmark \end{aligned}$$

$$\text{HQ CTE Arsenic (unitless)} = \frac{1.29 \text{E-}6 \text{ mg/kg-day}}{3.0 \text{E-}4 \text{ mg/kg-day}} = \boxed{4.3 \text{E-}3 \text{ CTE}} \quad \checkmark$$

References

USEPA Dec. 1989, Risk Assessment Guidance for Superfund Volume 1 Human Health Evaluation Manual (Part A), OSWER EPA/540/1-89/002.

CLIENT <i>Raymark - Ferry Creek</i>	JOB NUMBER <i>7491</i>		
SUBJECT <i>Accidental Ingestion of Soil - Adolescent trespasser</i>	DRAWING NUMBER		
BASED ON <i>RAGs 1989</i>	APPROVED BY		
BY <i>KAC</i>	CHECKED BY <i>RJJ</i>	APPROVED BY	DATE <i>9/24/99</i>

Purpose: To calculate carcinogenic and noncarcinogenic risk for the adolescent trespasser for exposure via incidental ingestion of soil.

Relevant Equation:

$$\text{Intake} = \frac{C_s \times IR \times CF \times FI \times EF \times ED}{BW \times AT}$$

(mg/kg-day)

Where

C_s = Chemical concentration in soil (mg/kg)

IR = Soil Ingestion Rate (mg/day)

CF = Conversion Factor (kg/mg)

FI = Fraction ingested from a contaminated source (unitless)

EF = Exposure Frequency (days/year)

ED = Exposure Duration (years)

BW = Body Weight (kg)

AT_C = Averaging Time carcinogenic exposures (days)

AT_N = Averaging Time noncarcinogenic exposures (days)

	Reasonable Maximum Exposures	Central Tendency Exposure
C_s	Chemical specific mg/kg	Chemical specific mg/kg
IR	100 mg/day	50 mg/day
CF	$1.0E-6$ (kg/mg)	$1E-6$ kg/mg
FI	1 unitless	1 unitless
EF	52 days/year	52 days/year
ED	10 years	5 years
BW	51 kg	51 kg
AT_N	3650 days	1825 days
AT_C	25550 days	25550 days

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Accidental Ingestion of soil - Adolescent Trespasser		
BASED ON	RAGs 1989	DRAWING NUMBER	
BY	KAC	CHECKED BY	RJJ
		APPROVED BY	
		DATE	9/24/99

Intake of Arsenic at a concentration of 9.9 mg/kg for Area A1 Surface soil under the Reasonable Maximum exposure (RME) assumptions is calculated:

$$\begin{aligned} \text{RME Noncancer Intake (mg/kg-day)} &= \frac{9.9 \text{ mg/kg} \times 100 \frac{\text{mg}}{\text{kg}} \times 1 \times 10^{-6} \frac{\text{kg}}{\text{mg}} \times 1 \times 52 \frac{\text{days}}{\text{year}} \times 10 \text{ years}}{51 \text{ kg} \times 3650 \text{ days}} \\ &= \boxed{2.8 \text{E-6 mg/kg-day RME}} \checkmark \end{aligned}$$

$$\begin{aligned} \text{RME Carc. Intake (mg/kg-day)} &= \frac{9.9 \frac{\text{mg}}{\text{kg}} \times 100 \frac{\text{mg}}{\text{kg}} \times 1 \times 10^{-6} \frac{\text{kg}}{\text{mg}} \times 1 \times 52 \frac{\text{days}}{\text{year}} \times 10 \text{ years}}{51 \text{ kg} \times 25550 \text{ days}} \\ &= \boxed{4.0 \text{E-7 mg/kg-day RME}} \checkmark \end{aligned}$$

Then Incremental Cancer Risk (ICR) and Hazard Quotient are calculated via the following:

$$\text{ICR (unitless)} = \frac{\text{Carcinogenic Intake (mg/kg-day)}}{\text{Cancer Slope Factor (kg/mg-day)}}$$

$$\text{HQ (unitless)} = \frac{\text{Noncarcinogenic Intake (mg/kg-day)}}{\text{Reference Dose (RFD) (mg/kg-day)}}$$

where the CSF for Arsenic = 1.5 and the RFD = 3.0E-4

$$\text{ICR RME Arsenic (unitless)} = \frac{4 \text{E-7 mg/kg-day}}{1.5 \text{ kg/mg-day}} = \boxed{6.0 \text{E-7 RME}} \checkmark$$

$$\text{HQ RME Arsenic (unitless)} = \frac{2.8 \text{E-6 mg/kg-day}}{3.0 \text{E-4 mg/kg-day}} = \boxed{9.3 \text{E-3 RME}} \checkmark$$

CLIENT	Raymark - Ferry Creek		JOB NUMBER	7491
SUBJECT	Accidental Ingestion of Soil - Adolescent trespasser			
BASED ON	RAGS 1984		DRAWING NUMBER	
BY	KAC	CHECKED BY	RJJ	APPROVED BY
				DATE 9/24/99

Intake of Arsenic at a concentration of 9.9 mg/kg for area A1 surface soil under a central tendency exposure (CTE) assumptions are calculated:

$$\begin{aligned} \text{CTE Noncancer} &= \frac{9.9 \frac{\text{mg}}{\text{kg}} \times 50 \frac{\text{mg}}{\text{day}} \times 1 \text{E-}6 \frac{\text{kg}}{\text{mg}} \times 1 \times 52 \frac{\text{day}}{\text{year}} \times 5 \text{ years}}{51 \text{ kg} \times 1825 \text{ days}} \\ \text{Intake (mg/kg-day)} &= \boxed{1.4 \text{E-}6 \text{ mg/kg-day CTE}} \checkmark \end{aligned}$$

$$\begin{aligned} \text{CTE Cancer} &= \frac{9.9 \frac{\text{mg}}{\text{kg}} \times 50 \frac{\text{mg}}{\text{day}} \times 1 \text{E-}6 \frac{\text{kg}}{\text{mg}} \times 1 \times 52 \frac{\text{day}}{\text{year}} \times 5 \text{ years}}{51 \text{ kg} \times 25550 \text{ days}} \\ \text{Intake (mg/kg-day)} &= \boxed{9.9 \text{E-}8 \text{ mg/kg-day CTE}} \checkmark \end{aligned}$$

Then ICR and HQ are calculated for arsenic via the following:

$$\begin{aligned} \text{ICR CTE Arsenic} &= \frac{9.9 \text{E-}8 \text{ mg/kg-day}}{\text{mg/kg-day}} \times 1.5 \frac{\text{kg/mg-day}}{\text{kg/mg-day}} = \boxed{1.5 \text{E-}7 \text{ CTE}} \checkmark \\ (\text{unitless}) & \end{aligned}$$

$$\begin{aligned} \text{HQ CTE Arsenic} &= \frac{1.4 \text{E-}6 \text{ mg/kg-day}}{3 \text{E-}4 \text{ mg/kg-day}} = \boxed{4.6 \text{E-}3 \text{ CTE}} \checkmark \\ (\text{unitless}) & \end{aligned}$$

Reference

USEPA Dec. 1989. Risk Assessment Guidance for Superfund Volume 1 Human Health Evaluation Manual (Part A). OSWER EPA/540/1-89/002.

CLIENT	Rainmark Ferry Creek	JOB NUMBER	7491
SUBJECT	Direct Dermal Contact with Soil - Adolescent trespasser		
BASED ON	RAGS 1989	DRAWING NUMBER	
BY	KAC	CHECKED BY	RJS
		APPROVED BY	
		DATE	9/24/99

Purpose: To calculate carcinogenic and noncarcinogenic risk for the adolescent trespasser with exposure via direct dermal contact with soil.

Relevant Equations:

$$\text{Absorbed Dose} = \frac{C_s \times CF \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

(mg/kg-day)

Where:

- C_s = Chemical concentration in soil (mg/kg)
 CF = Conversion Factor (kg/mg)
 SA = Skin surface available for contact (cm²/event)
 AF = Soil to skin adherence factor (mg/cm²)
 ABS = Absorption factor (unitless)
 EF = Exposure Frequency (events/year)
 ED = Exposure Duration (years)
 BW = Body weight (kg)
 ATC = Averaging Time for carcinogenic (days)
 ATN = Averaging Time for noncarcinogenic (days)

	Reasonable Maximum Exposure	Central Tendency Exposure
C_s	Chemical specific (mg/kg)	Chemical specific (mg/kg)
CF	1E-6 kg/mg	1E-6 kg/mg
SA	3500 cm ² /event	3500 cm ² /event
AF	0.07 mg/cm ²	0.01 mg/cm ²
ABS	Chemical-specific (unitless)	Chemical-specific (unitless)
EF	52 events/year	52 events/year
ED	10 years	5 years
BW	51 kg	51 kg
ATN	3650 days	1825 days
ATC	25550 days	25550 days

CLIENT	Raymark - Ferry Creek	JOB NUMBER	7491
SUBJECT	Direct Dermal Contact with Soil - Adolescent Trespasser		
BASED ON	RAGs 1989	DRAWING NUMBER	
BY	KAC	CHECKED BY	RJS
		APPROVED BY	
		DATE	9/24/99

The absorbed dose of arsenic at a concentration of 9.9 mg/kg for Area A1 surface soil under the reasonable maximum exposure (RME) assumptions is calculated:

$$\begin{aligned} \text{RME Noncancer Absorbed Dose (mg/kg-day)} &= \frac{9.9 \frac{\text{mg}}{\text{kg}} \times 1 \text{E-}6 \frac{\text{kg}}{\text{m}^2} \times 3500 \frac{\text{cm}^2}{\text{event}} \times 0.07 \frac{\text{m}^2}{\text{cm}^2} \times 0.03 \times 52 \frac{\text{events}}{\text{year}} \times 10 \text{ years}}{51 \text{ kg} \times 3650 \text{ days}} \\ &= \boxed{2.03 \text{E-}7 \text{ mg/kg-day RME}} \quad \checkmark \end{aligned}$$

$$\begin{aligned} \text{RME Carcinog Absorbed Dose (mg/kg-day)} &= \frac{9.9 \frac{\text{mg}}{\text{kg}} \times 1 \text{E-}6 \frac{\text{kg}}{\text{m}^2} \times 3500 \frac{\text{cm}^2}{\text{event}} \times 0.07 \frac{\text{m}^2}{\text{cm}^2} \times 0.03 \times 52 \frac{\text{events}}{\text{year}} \times 10 \text{ yr}}{51 \text{ kg} \times 25550 \text{ days}} \\ &= \boxed{2.9 \text{E-}8 \text{ mg/kg-day RME}} \quad \checkmark \end{aligned}$$

Then Incremental Cancer Risk (ICR) and Hazard Quotient (HQ) are calculated via the following equations:

$$\text{ICR (unitless)} = \frac{\text{Carcinogenic Absorbed Dose (mg/kg-day)}}{\text{Cancer Slope Factor (CSF) (kg/mg-day)}}$$

$$\text{HQ (unitless)} = \frac{\text{noncarcinogenic Absorbed Dose (mg/kg-day)}}{\text{Reference Dose (RfD) (mg/kg-day)}}$$

where the CSF for arsenic = 1.5 and the RfD = 3.0E-4

$$\text{RME ICR Arsenic (unitless)} = 2.9 \text{E-}8 \text{ mg/kg-day} \times 1.5 = \boxed{4.4 \text{E-}8 \text{ RME}} \quad \checkmark$$

$$\text{RME HQ Arsenic (unitless)} = \frac{2.03 \text{E-}7 \text{ mg/kg-day}}{3.0 \text{E-}4 \text{ mg/kg-day}} = \boxed{6.8 \text{E-}4 \text{ RME}} \quad \checkmark$$

CLIENT	Raymark - Ferry Creek		JOB NUMBER	7491
SUBJECT	Direct Dermal Contact with Soil - Adolescent trespasser			
BASED ON	RAGS 1989		DRAWING NUMBER	
BY	KAC	CHECKED BY	RJJ	APPROVED BY
				DATE 9/24/99

The absorbed dose of arsenic at a concentration of 9.9 mg/kg for Urea A1 surface soil under the central tendency exposure (CTE) assumptions are calculated:

$$\text{CTE Noncanc. Absorbed Dose (mg/kg-day)} = \frac{9.9 \frac{\text{mg}}{\text{kg}} \times 1E-6 \frac{\text{kg}}{\text{cm}^2} \times 3500 \frac{\text{cm}^2}{\text{event}} \times 0.01 \frac{\text{mg}}{\text{cm}^2} \times 0.03 \times 52 \frac{\text{events}}{\text{yr}} \times 5 \text{yr}}{51 \text{ kg} \times 1825 \text{ days}} = \boxed{2.9E-8 \text{ mg/kg-day CTE}} \checkmark$$

$$\text{CTE Canc. Absorbed Dose (mg/kg-day)} = \frac{9.9 \frac{\text{mg}}{\text{kg}} \times 1E-6 \frac{\text{kg}}{\text{cm}^2} \times 3500 \frac{\text{cm}^2}{\text{event}} \times 0.01 \frac{\text{mg}}{\text{cm}^2} \times 0.03 \times 52 \frac{\text{event}}{\text{yr}} \times 5 \text{yr}}{51 \text{ kg} \times 25550 \text{ days}} = \boxed{2.1E-9 \text{ mg/kg-day CTE}} \checkmark$$

Then ICR and HQ for arsenic are calculated via the following:

$$\text{ICR CTE Arsenic} = \frac{2.1E-9 \text{ (mg/kg-day)}}{3E-4 \text{ (mg/kg-day)}} \times 1.5 = \boxed{3.15E-9 \text{ CTE}} \checkmark$$

$$\text{HQ CTE Arsenic} = \frac{2.9E-8 \text{ mg/kg-day}}{3E-4 \text{ mg/kg-day}} = \boxed{9.7E-5 \text{ CTE}} \checkmark$$

Reference

USEPA Dec. 1989, Risk Assessment Guidance for Superfund Volume 1 Human Health Evaluation Manual (Part A), OSWER EPA/540/1-89/002,

TETRA TECH NUS, INC. CALCULATION WORKSHEET

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CLIENT Raymark	JOB NUMBER		
SUBJECT Direct Dermal Contact with Surface Water			
BASED ON RAGs and Draft Dermal Guidance		DRAWING NUMBER	
BY KAC	CHECKED BY R/Q	APPROVED BY	DATE 7/22/99

Purpose: To calculate carcinogenic and noncarcinogenic risk via dermal exposure to surface water.

Relevant Equations:

$$\text{Absorbed Dose} = \frac{D_{\text{event}} \times EV \times EF \times ED \times SA}{BW \times AT}$$

For Inorganic Compounds:

$$D_{\text{event}} = K_p \times C_w \times t_{\text{event}} \times CF$$

For Organic Compounds:

If $t_{\text{event}} \leq t^*$ then,

$$D_{\text{event}} = 2 \times K_p \times C_w \times CF \times \sqrt{\frac{6 \times \tau \times t_{\text{event}}}{\pi}}$$

If $t_{\text{event}} > t^*$ then,

$$D_{\text{event}} = K_p \times C_w \times CF \times \left[\frac{t_{\text{event}}}{1+B} + 2 \times \tau \times \frac{(1+3B+3B^2)}{(1+B)^2} \right]$$

where: If $B \leq 0.6$ then $t^* = 2.4 \times \tau_{\text{au}}$

but if $B > 0.6$ then $t^* = \frac{(b - \sqrt{b^2 - c^2}) \times 0.000001}{D_{\text{sc}}}$

$$\text{where } B = K_p \times \frac{MW}{2.6}$$

$$C = \frac{1 + 3B + 3B^2}{3(1+B)}$$

$$b = \left(\frac{2(1+B)^2}{\pi} \right) - C$$

$$D_{\text{sc}} = 0.001 \times 10^{(-2B - 0.0056 \times MW)}$$

$$\tau_{\text{au}} = 0.105 \times 10^{(0.0056 \times MW)}$$

CLIENT Raymark		JOB NUMBER	
SUBJECT Direct Dermal Contact with Surface Water			
BASED ON RAGs and Draft Dermal Guidance		DRAWING NUMBER	
BY KAC	CHECKED BY RJG	APPROVED BY	DATE 7/22/99

the organic compound
 Values for **Trichloroethene** in surface water at Area A-3
 for the Adult Frequent Recreational User the following exposure
 assumptions are true:

Where:	SA =	8,600	Skin surface available for contact (cm ²)
	DA _{event} =		Chemical specific absorbed dose per event (mg/cm ² -event)
	EV =	1	Event frequency (events/days)
	EF =	90	Exposure frequency (days/year)
	ED =	24	Exposure duration (years)
	BW =	70	Body weight (kg)
	AT _c =	26,550	Averaging time for carcinogenic exposures (days)
	AT _n =	8,760	Averaging time for noncarcinogenic exposures (days)
	CF =	0.001	Conversion Factor (L/m ³)
	K _p =	2.90E-2	Chemical specific permeability coefficient (cm/hr) trichloroethene
	C _w =	0.078	Concentration of chemical in water (mg/L) trichloroethene
	t _{event} =	1	duration of event (hr/event)
	tau =		Chemical specific lag time (hr)
	t* =		Chemical specific time it takes to reach steady state (hr)
	B =		Chemical specific dimensionless constant
	D _{sc} =		Effective diffusivity for chemical transfer through skin (cm ² /hr)
	b, c =		chemical specific constants

MW = 112.6 Molecular Weight for trichloroethene

$$\tau = 0.105 \times 10^{(0.0056 \times 112.6)} = \boxed{4.48E-1 \text{ hr}}$$

$$B = 2.90E-2 \times \frac{\sqrt{112.6}}{2.6} = \boxed{1.18E-1}$$

$$C = \frac{1 + 3(1.18E-1) + 3(1.18E-1)^2}{3(1 + 1.18E-2)} = \boxed{4.16E-1}$$

$$b = \left(\frac{2(1 + 1.18E-1)}{\pi} \right)^2 - 4.16E-1 = \boxed{3.79E-1}$$

$$D_{sc} = 0.001 \times 10^{(-2.8 - 0.0056 \times 112.6)} = \boxed{3.71E-7 \text{ cm}^2/\text{hr}}$$

And $B = 1.18E-1$ which is less than 0.6 hence

$$t^* = 2.4 \times \tau \quad ; \quad \boxed{t^* = 2.4 \times 4.48E-1 = 1.08 \text{ hr}}$$

TETRA TECH NUS, INC. CALCULATION WORKSHEET

CLIENT	Raymark		JOB NUMBER	
SUBJECT	Direct Dermal Contact with Surface Water			
BASED ON	RAGs and Draft Dermal Guidance		DRAWING NUMBER	
BY	KAC	CHECKED BY	RJP	APPROVED BY
				DATE 7/20/99

And t^* (1.08) is greater than t_{event} (1.0) therefore

$$DA_{event} = 2 \times 2.9E-2 \times 0.078 \times 0.001 \times \sqrt{\frac{6 \times 4.48E-1 \times 1.0}{\pi}}$$

$DA_{event} = 4.2E-6$ mg/cm²-event
trichloroethene

absorbed dose = $\frac{4.2E-6 \times 1 \times 90 \times 24 \times 6600}{70 \times 8760} = 9.7E-5$ mg/kg-day
noncarcinogen

absorbed dose = $\frac{4.2E-6 \times 1 \times 90 \times 24 \times 6600}{70 \times 25550} = 3.3E-5$ mg/kg-day
carcinogen

The cancer slope factor (CSF) for Trichloroethene = $1.1E-2$ kg/mg-day

The reference dose (RfD) for trichloroethene = $6.0E-3$ mg/kg-day

Lifetime Cancer Risk = $\frac{1.1E-2 \times 3.3E-5}{(kg/mg-day) (mg/kg-day)} = 3.6E-7$

Hazard Quotient = $\frac{\text{absorbed dose noncarcinogen mg/kg-day}}{RfD \text{ mg/kg-day}} = \frac{9.7E-5}{6.0E-3} = 1.6E-2$

Values for the inorganic compound Arsenic in surface water at Area A3 for the adult Frequent Recreational Use were also calculated for which the following assumptions are true:

- $K_p = 1.0E-3$ cm/hr permeability coefficient for arsenic
- $C_w = 0.0751$ mg/L concentration of Arsenic in surface water
- $CF = 0.001$ L/m³ Conversion Factor
- $t_{event} = 1.0$ hr/event duration of event

TETRA TECH NUS, INC. CALCULATION WORKSHEET

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CLIENT Raymark	JOB NUMBER		
SUBJECT Direct Dermal Contact with Surface Water	DRAWING NUMBER		
BASED ON RAGs and Draft Dermal Guidance	APPROVED BY		DATE 7/22/99
BY KAC	CHECKED BY RJF		

For Anorganics $DA_{event} = K_p \times C_w \times CF \times t_{event}$

$$= 1E-3 \times 0.0751 \times 0.001 \times 1 = 7.51E-8 \frac{mg}{cm^2 \cdot event}$$

absorbed dose = $\frac{7.51E-8 \times 1 \times 90 \times 24 \times 6600}{70 \times 0.760} = 1.75E-6 \text{ mg/kg-day}$
noncarcinogen

absorbed dose = $\frac{7.51E-8 \times 1 \times 90 \times 24 \times 6600}{70 \times 25550} = 5.99E-7 \text{ mg/kg-day}$
Carcinogen

The cancer slope factor (CSF) for Arsenic = 1.5 kg/mg-day
The reference dose (RfD) for Arsenic = $3.0E-4 \text{ mg/kg-day}$

Lifetime Cancer Risk = $\frac{1.5 \text{ kg/mg-day}}{CSF} \times \frac{5.99E-7 \text{ mg/kg-day}}{\text{absorbed dose carcinogen}} = 9.0E-7$

Hazard Quotient = $\frac{\text{absorbed dose noncarcinogen mg/kg-day}}{RfD \text{ mg/kg-day}} = \frac{1.75E-6}{3E-4} = 5.8E-3$