

D-8 TOXICITY PROFILES

D.8.1 ARSENIC

D.8.1.1 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.

D.8.1.2 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 produce 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).

D.8.1.3 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.

D.8.2 BARIUM

D.8.2.1 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.

D.8.2.2 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.

D.8.3 CADMIUM

D.8.3.1 PHARMACOKINETICS

Estimates of cadmium uptake by the respiratory tract range from 10 to 50 percent; uptake is greatest for fumes and small particles and least for large dust particles (Friberg et al., 1986; Goyer, 1991). GI absorption of ingested cadmium is ordinarily 5 to 8 percent, but may reach 20 percent in cases of serious dietary iron deficiency. Highest tissue levels are normally found in the kidneys followed by the liver, although levels in the liver may exceed those in the kidneys of persons suffering from cadmium-induced renal dysfunction. The half-life of cadmium in the kidneys and liver may be as long as 10-30 years. Fecal and urinary excretion of cadmium are approximately equivalent to normal humans exposed to small amounts. Urinary excretion increases markedly in humans with cadmium-induced renal disease.

F.7.3.2 NONCANCER TOXICITY

Acute inhalation exposure to fumes or particles of cadmium induces respiratory symptoms, general weakness, and, in severe cases, respiratory insufficiency, shock, and death (Friberg et al., 1986). Acute oral exposure induces GI disturbances. Chronic inhalation exposure induces pulmonary emphysema, and chronic exposure by either route consistently produces renal tubular disease in humans and laboratory animals. Proteinuria is a reliable early indicator of cadmium-induced kidney disease. The combination of pulmonary emphysema and renal tubular disease, if severe, may result in early mortality. Painful osteomalacia and osteoporosis may arise from altered metabolism of bone minerals secondary to renal damage. The combination of renal and skeletal damage is called itai-itai disease in Japan. Cadmium exposure has been associated with liver damage, but the liver appears to be less sensitive than the kidney. The kidney is the primary target organ of cadmium toxicity. The EPA (1998b) derived chronic oral RfD values of 0.5 ug/kg/day for cadmium ingested in water and 1 ug/kg/day for cadmium ingested in food, based on a toxicokinetic model that predicted NOAELs from renal cortical concentration of cadmium.

The different RfD values reflect assumed differences in GI absorption of cadmium from water (5 percent) and food (2.5 percent).

D.8.3.3 CARCINOGENICITY

Carcinogenicity data in humans consist of several occupational studies that associate cadmium exposure with lung cancer, but concomitant exposure to other carcinogenic chemicals and smoking were not adequately controlled. Other occupational studies reported significantly increased risk of prostatic cancer, but this effect was not observed in the largest occupational study of workers exposed to high levels (Thun et al., 1985). The animal data consist of an inhalation study in rats that showed a significant increase in lung tumors, and several parenteral injection studies that produced injection site tumors. No evidence of carcinogenicity, however, was observed in seven oral studies in rats and mice. The EPA (1998b) classifies cadmium a cancer weight-of-evidence Group B1 substance for inhalation exposure on the basis of limited evidence of carcinogenicity in humans and sufficient evidence in animals. The data were insufficient to classify cadmium as carcinogenic to humans exposed by the oral route. An inhalation unit risk of 0.0018 mg/m³, equivalent to 6.3 per mg/kg/day, was derived from the occupational exposure study by Thun et al. (1985) assuming an inhalation rate of 20 m³/day and a body weight of 70 kg for humans.

D.8.4 CHROMIUM

D.8.4.1 NONCANCER TOXICITY

In nature, chromium (III) predominates over chromium (VI) (Langård and Norseth, 1986). Little chromium (VI) exists in biological materials, except shortly after exposure, because reduction to chromium (III) occurs rapidly. Chromium (III) is considered a nutritionally essential trace element and is considerably less toxic than chromium (VI). No effects were observed in rats consuming 5% chromium (III)/kg/day in the diet for over two years (EPA, 1997). The NOEL of 5% Cr₂O₃ was the basis for a verified chronic oral RfD of 1.5 mg/kg/day (EPA, 1997). The same NOEL and an uncertainty factor of 1000 were the basis for a provisional subchronic oral RfD of 1 mg/kg/day (EPA, 1997).

Acute oral exposure of humans to high doses of chromium (VI) induced neurological effects, GI hemorrhage and fluid loss, and kidney and liver effects. Parenteral dosing of animals with chromium (VI) is selectively toxic to the kidney tubules. An NOAEL of 2.4 mg chromium (VI) /kg/day in a one-year drinking water study in rats and an uncertainty factor of 500 was the basis of a verified RfD of 0.003 mg/kg/day for chronic oral exposure (EPA, 1998b). The same NOAEL and an uncertainty factor of 100 were the basis of a provisional subchronic oral RfD of 0.02 mg/kg/day (EPA, 1997).

Occupational (inhalation and dermal) exposure to chromium (III) compounds induced dermatitis (ACGIH, 1991). Similar exposure to chromium (VI) induced ulcerative and allergic contact dermatitis, irritation of the upper respiratory tract including ulceration of the mucosa and perforation of the nasal septum, and possibly kidney effects. An inhalation RfC values was not located for chromium (III), however, EPA (1998b) presents an inhalation RfD of 0.03 ug/kg/day for chromium (VI).

A target organ was not identified for chromium (III). The kidney appears to be the principal target organ for repeated oral dosing with chromium (VI). Additional target organs for dermal and inhalation exposure include the skin and respiratory tract.

D.8.4.2 CARCINOGENICITY

Data were not located regarding the carcinogenicity of chromium (III). The EPA (1998b) classifies chromium (VI) in cancer weight-of-evidence Group A (human carcinogen), based on the consistent observation of increased risk of lung cancer in occupational studies of workers in chromate production or the chrome pigment industry. Parenteral dosing of animals with chromium (VI) compounds consistently induced injection-site tumors. There is no evidence that oral exposure to chromium (VI) induces cancer. An inhalation unit risk of 0.012 per mg/m^3 , equivalent to 41 per $\text{mg}/\text{kg}/\text{day}$, assuming humans inhale $20 \text{ m}^3/\text{day}$ and weigh 70 kg, was based on increased risk of lung cancer deaths in chromate production workers (EPA, 1997).

D.8.5 LEAD

D.8.5.1 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.

D.8.5.2 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.

D.8.5.3 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 chromatic 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).

D.8.6 THALLIUM

D.8.6.1 NONCANCER TOXICITY

Thallium is highly toxic; acute ingestion by humans or laboratory animals induced gastroenteritis, neurological dysfunction, and renal and liver damage (Kazantzis, 1986). Chronic ingestion of more moderate doses characteristically caused alopecia. Thallium was used medicinally to induce alopecia in cases of ringworm of the scalp, sometimes with disastrous results. In industrial (inhalation, oral, dermal) exposure, neurologic signs preceded alopecia, suggesting that the nervous system is more sensitive than the hair follicle. The EPA (1998b) presented verified chronic oral RfD values for several thallium compounds (thallium acetate, thallium acetate, thallium carbonate, thallium chloride, thallium nitrate, thallium sulfate, and thallic oxide) based on increased incidence of alopecia and increased serum levels of liver enzymes indicative of hepatocellular damage in rats treated with thallium sulfate for 90 days. EPA (1998b) presented a chronic oral RfD for thallium of 0.07 ug/kg/day.

D.8.6.2 CARCINOGENICITY

Thallium was classified as a cancer weight-of-evidence Group D substance (not classifiable as to carcinogenicity to humans) (EPA, 1998b).

D.8.7 ZINC

D.8.7.1 PHARMACOKINETICS

Zinc is a nutritionally required trace element. Estimates of the efficiency of GI absorption of zinc in animals range from <10 to 90 percent (Elinder 1986c). Estimates in normal humans range from approximately 20 to 77 percent (Elinder 1986c; Goyer 1991). The net absorption of zinc appears to be homeostatically controlled, but it is unclear whether GI absorption, intestinal secretion, or both are regulated. Distribution of absorbed zinc is primarily to the liver (Goyer 1991), with subsequent redistribution to bone, muscle, and kidney (Elinder 1986c). Highest tissue concentrations are found in the prostate. Excretion appears to be principally through the feces, in part from biliary secretion, but the relative importance of fecal and urinary excretion is species-dependent. The half-life of zinc absorbed from the GI tracts of humans in normal zinc homeostasis is approximately 162 to 500 days.

D.8.7.2 NONCANCER TOXICITY

Humans exposed to high concentrations of aerosols of zinc compounds may experience severe pulmonary damage and death (Elinder 1986c). The usual occupational exposure is to freshly formed fumes of zinc, which can induce a reversible syndrome known as metal fume fever. Orally, zinc exhibits a low order of acute toxicity. Animals dosed with 100 times dietary requirement showed no evidence of toxicity (Goyer 1991). In humans, acute poisoning from foods or beverages prepared in galvanized containers is characterized by GI upset (Elinder 1986c). Chronic oral toxicity in animals is associated with poor growth, GI inflammation, arthritis, lameness, and a microcytic, hypochromic anemia (Elinder 1986c), possibly secondary to copper deficiency (Underwood 1977). The EPA (1992b) presented a verified RfD of 0.3 mg/kg/day for chronic oral exposure to zinc, based on anemia in humans.

D.8.7.3 CARCINOGENICITY

The EPA (1993a) classifies zinc in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) based on inadequate evidence for carcinogenicity in humans and animals. The human data consist largely of occupational exposure studies not designed to detect a carcinogenic response, and of reports that prostatic zinc concentrations were lower in cancerous than in noncancerous tissue. The animal data consist of several dietary, drinking water, and zinc injection studies, none of which provided convincing data for a carcinogenic response.

D.8.8 POLYCHLORINATED BIPHENYLS

D.8.8.1 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.

D.8.8.2 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.

D.8.9 POLYAROMATIC HYDROCARBONS

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

D.8.9.1 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 tetrahydrotetrols 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.

D.8.9.2 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.

D.8.9.3 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).

D.8.10 BENZO(A)ANTHRACENE

D.8.10.1 NONCANCER TOXICITY

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

D.8.10.2 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.

D.8.11 BENZO [A]PYRENE (BAP)

D.8.11.1 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.

D.8.11.2 NONCANCER TOXICITY

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

D.8.11.3 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.

D.8.12 BENZO(B)FLUORANTHENE

D.8.12.1 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.

D.8.12.2 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.

D.8.13 DIBENZO[A,H]ANTHRACENE

D.8.13.1 NONCANCER TOXICITY

The oral RfD and inhalation RfC are not available.

D.8.13.2 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.

D.8.14 INDENO(1,2,3-CD)PYRENE

D.8.14.1 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.

D.8.14.2 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.

D.8.15 BENZENE

D.8.15.1 NONCANCER TOXICITY

In humans, short-term inhalation exposure to benzene induced CNS effects such as drowsiness, dizziness, and headaches; long-term exposure induced anemia (ACGIH, 1991). Oral dosing in animals induced hematopoietic effects (Agency for Toxic Substances and Disease Registry [ATSDR], 1995c). The EPA presents an oral RfD of 0.003 mg/kg/day and an inhalation RfD of 0.00171 mg/kg/day. The CNS and the hematopoietic system are the target organs of benzene.

D.8.15.2 CARCINOGENICITY

The EPA (1998b) classifies benzene in cancer weight-of-evidence Group A (human carcinogen) based on several studies of increased risk of nonlymphocytic leukemia associated with occupational exposure, supported by an increased incidence of neoplasia in rats and mice exposed by inhalation and gavage. A verified oral slope factor of 0.029 per mg/kg/day and inhalation unit risk of $8.3E-06$ ug/m³ is based on the increased incidence of leukemia in several occupational (inhalation exposure) studies. The inhalation unit risk is equivalent to 0.029 per mg/kg/day, assuming an inhalation rate of 20 m³/day and a body weight of 70 kg for humans.

D.8.16**CHLOROBENZENE**

One carcinogenicity study showed that chloroenezene caused neoplastic nodules in the liver of male rats but was not carcinogenic in female rats or in mice. Occupational studies suggest that chronic exposure to chlorobenzene vapor may cause blood dyscrasia, hyperlipidemia, and cardiac dysfunction in humans. Like many organic solvents, monochlorobenzene is a central nervous system depressant in overexposed humans, but no chronic neurotoxic effects have been reported.

D.8.17 DIELDRIN

D.8.17.1 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.

D.8.17.2 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.

D.8.18 HEPTACHLOR

Heptachlor is one of the chlorinated insecticides based on the cyclodiene ring structure; formerly widely used for the control of agricultural pests and for structural pest control. All of these pesticides are neurotoxicants, although the symptoms following exposure are quite different to those produced by DDT. Heptachlor is toxic by ingestion, inhalation, and dermal adsorption. Chronic toxicity is said to include carcinogenicity and/or tumor promotion. The mode of acute toxicity is now generally believed to involve effects on GABA transmitters in the brain. Cyclodienes are metabolized to their expoxides in vivo. Since the expoxides are approximately equitoxic and equally persistent with the parent compound, this is not a detoxication reaction.

Based on a review of a March 1999 version of information presented in the EPA's Integrated Risk Information System, toxicity criteria (reference doses and cancer slope factors) are available for this chemical based on the results of animal studies indicating affects to the liver.

D.8.19 ASBESTOS

D.8.19.1 NONCANCER TOXICITY

Data not available at this time.

D.8.19.2 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 um long and of fibers longer than 5 um. Comparison of PCM fiber counts and TEM counts of fibers >5 um show that the fraction of fibers detected by TEM that are also >0.4 um 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 a 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.

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D-9 RELATIVE ORDER OF POTENCY FOR PAHS

TABLE D-9

ESTIMATED ORDERS OF POTENTIAL POTENCY FOR CARCINOGENIC PAHs⁽¹⁾
OU#4, STRATFORD, CONNECTICUT

Chemical	Weight-of-Evidence	Order of Potential Potency
Benzo(a)anthracene	B2	0.1
Benzo(b)fluoranthene	B2	0.1
Benzo(k)fluoranthene	B2	0.01
Benzo(a)pyrene	B2	1.0
Chrysene	B2	0.001
Dibenzo(a,h)anthracene	B2	1.0
Indeno(1,2,3-cd)pyrene	B2	0.1

1 USEPA, July 1993g; USEPA Region I, 1994m.

D-10 RESIDENTIAL RISKS TO 0-4 FOOT SOILS

TABLE D-10.2
 OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
 RAYMARK OU4 - BALLFIELD

Scenario Timeframe: Future
 Medium Soil
 Exposure Medium: Soil
 Exposure Point: 0 to 4 feet

CAS Number	Chemical	Minimum Concentration ⁽¹⁾	Minimum Qualifier	Maximum Concentration ⁽¹⁾	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value ⁽²⁾	Screening Toxicity Value ⁽³⁾	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for Contaminant Deletion or Selection ⁽⁴⁾
78-93-3	2-Butanone	12	J	21		ug/kg	SO-426	5/21	8 - 25	21						
57-64-1	Acetone	15	J	180	J	ug/kg	SO-426	5/21	8 - 140	180		3.43E+06	80000	CTPMGB	NO	BSL
71-43-2	Benzene	0.8	J	0.8	J	ug/kg	SO-426	1/21	8 - 25	0.8		7.22E+05	140000	CTPMGB	NO	BSL
74-83-9	Bromomethane	1	J	1	J	ug/kg	SO-421	1/21	8 - 25	1		6.19E+02	200	CTPMGB	NO	BSL
75-15-0	Carbon Disulfide	0.6	J	4	J	ug/kg	SO-TP11	5/21	8 - 24	4		1.92E+03		CTPMGB	NO	BSL
108-90-7	Chlorobenzene	2	J	2	J	ug/kg	SO-421	2/21	8 - 25	2		1.74E+05	140000	CTPMGB	NO	BSL
57-66-3	Chloroform	0.8	J	2	J	ug/kg	SO-421	2/21	9 - 25	2		2.68E+04	20000	CTPMGB	NO	BSL
108-88-3	Toluene	8	J	8	J	ug/kg	SO-426	1/21	8 - 25	8		2.44E+02	1200	CTPMGB	NO	BSL
1330-20-7	Total Xylenes	6	J	6	J	ug/kg	SO-426	1/21	8 - 25	6		5.20E+05	67000	CTPMGB	NO	BSL
79-01-6	Trichloroethene	0.6	J	0.6	J	ug/kg	SO-421	1/21	8 - 25	0.6		2.10E+05	19500	CTPMGB	NO	BSL
106-46-7	1,4-Dichlorobenzene	24	J	24	J	ug/kg	SO-426	1/21	330 - 660	24		3.03E+03	15000	CTPMGB	NO	BSL
105-67-9	2,4-Dimethylphenol	81	J	630		ug/kg	SO-426	5/21	330 - 420	630		5.45E+05	28000	CTPMGB	NO	BSL
91-57-6	2-Methylnaphthalene	19	J	120	J	ug/kg	SO-424, SO-TP15	7/21	330 - 480	120			56000	CTPMGB	NO	BSL
95-48-7	2-Methylphenol	42	J	170	J	ug/kg	SO-426	4/21	330 - 660	170		1.36E+06	70000	CTPMGB	NO	BSL
91-94-1	3,3'-Dichlorobenzidine	100	J	100	J	ug/kg	SO-TP15	1/21	330 - 480	100		9.87E+02	16	CTPMGB	NO	BSL
106-44-5	4-Methylphenol	24	J	550		ug/kg	SO-421	5/21	330 - 420	550		1.36E+05	7000	CTPMGB	NO	BSL
83-32-9	Acenaphthene	18	J	190	J	ug/kg	SO-425	6/21	330 - 660	190		1.28E+06	84000	CTPMGB	NO	BSL
208-96-8	Acenaphthylene	20	J	540		ug/kg	SO-424	9/21	330 - 660	540			84000	CTPMGB	NO	BSL
120-12-7	Anthracene	29	J	900		ug/kg	SO-424	11/21	330 - 660	900		7.1E+06	400000	CTPMGB	NO	BSL
56-55-3	Benzo(a)anthracene	31	J	2600	J	ug/kg	SO-424	18/21	330 - 350	2600		5.57E+02	1000	CTPMGB	YES	ASL
50-32-8	Benzo(a)pyrene	24	J	2600	J	ug/kg	SO-424	20/21	330 - 330	2600		5.57E+01	1000	CTPMGB	YES	ASL
205-99-2	Benzo(b)fluoranthene	34	J	2200	J	ug/kg	SO-424	20/21	330 - 330	2200		5.57E+02	1000	CTPMGB	YES	ASL
191-24-2	Benzo(g,h,i)perylene	24	J	2700	J	ug/kg	SO-424	20/21	330 - 330	2700			40000	CTPMGB	NO	NTX
207-08-9	Benzo(k)fluoranthene	36	J	2100	J	ug/kg	SO-424	17/21	330 - 410	2100		5.57E+03	1000	CTPMGB	NO	BSL
117-81-7	bis(2-Ethylhexyl)phthalate	310	J	2100	J	ug/kg	SO-TP18	4/21	330 - 660	2100		3.17E+04	11000	CTPMGB	NO	BSL
85-68-7	Butylbenzylphthalate	30	J	64	J	ug/kg	SO-TP11	2/21	330 - 660	64		9.30E+05	200000	CTPMGB	NO	BSL
86-74-8	Carbazole	21	J	290	J	ug/kg	SO-425	8/21	330 - 660	290		2.22E+04	360	CTPMGB	NO	BSL
218-01-9	Chrysene	41	J	2800	J	ug/kg	SO-424	20/21	330 - 330	2800		5.57E+04	960	CTPMGB	NO	BSL
84-74-2	Di-n-Butylphthalate	20	J	570	J	ug/kg	SO-TP18	7/21	330 - 420	570		2.73E+06	140000	CTPMGB	NO	BSL
53-70-3	Dibenzo(a,h)anthracene	25	J	800	J	ug/kg	SO-424	10/21	330 - 660	800		5.57E+01	0.96	CTPMGB	YES	ASL
132-64-9	Dibenzofuran	19	J	200	J	ug/kg	SO-424	6/21	330 - 660	200		1.03E+05	5600	CTPMGB	NO	BSL
84-66-2	Diethylphthalate	20	J	120	J	ug/kg	SO-TP18	2/21	330 - 660	120		2.18E+07	1100000	CTPMGB	NO	BSL
206-44-0	Fluoranthene	18	J	4100		ug/kg	SO-424	20/21	460 - 460	4100		9.99E+05	56000	CTPMGB	NO	BSL
86-73-7	Fluorene	34	J	430		ug/kg	SO-424	6/21	330 - 660	430		8.94E+05	56000	CTPMGB	NO	BSL
193-39-5	Indeno(1,2,3-cd)pyrene	23	J	2200	J	ug/kg	SO-424	18/21	330 - 660	2200		5.57E+02	9.6	CTPMGB	YES	ASL
621-64-7	N-Nitroso-di-n-propylamine	44	J	44	J	ug/kg	SO-427	1/21	330 - 660	44		6.34E+01	1	CTPMGB	NO	BSL
86-30-6	N-Nitroso-diphenylamine	62	J	280	J	ug/kg	SO-426	3/21	330 - 420	280		9.06E+04	1400	CTPMGB	NO	BSL
91-20-3	Naphthalene	22	J	170	J	ug/kg	SO-424	8/21	330 - 420	170		2.74E+04	56000	CTPMGB	NO	BSL
87-86-5	Pentachlorophenol	53	J	53	J	ug/kg	SO-426	1/21	820 - 1700	53		2.53E+03	1000	CTPMGB	NO	BSL
85-01-8	Phenanthrene	18	J	2600		ug/kg	SO-424	19/21	340 - 360	2600			40000	CTPMGB	NO	NTX
108-95-2	Phenol	570		770		ug/kg	SO-426	2/21	330 - 660	770		1.64E+07	800000	CTPMGB	NO	BSL

TABLE D-10.2
 OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
 RAYMARK OU4 - BALLFIELD

Scenario Timeframe: Future
 Medium: Soil
 Exposure Medium: Soil
 Exposure Point: 0 to 4 feet

CAS Number	Chemical	(1) Minimum Concentration	Minimum Qualifier	(1) Maximum Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value (2)	Screening Toxicity Value (3)	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for Contaminant Deletion or Selection (4)	
129-00-0	Pyrene	18	J	7800	J*	ug/kg	SO-424	21/21	0 - 0	7800		7.42E+05	40000	CTPMGB	NO	BSL	
72-54-8	4,4'-DDD	0.24	J	42	J	ug/kg	RMF B+150	14/26	3.6 - 130	42		2.36E+03	29	CTPMGB	NO	BSL	
72-55-9	4,4'-DDE	0.21	J	710	J*	ug/kg	RMF C+250	23/27	3.6 - 75	710		1.66E+03	21	CTPMGB	NO	BSL	
50-29-3	4,4'-DDT	1	J	710	J	ug/kg	SO-TP18	21/27	3.8 - 130	710		1.66E+03	21	CTPMGB	NO	BSL	
319-84-6	alpha-BHC	1	J	8.8	J	ug/kg	SO-426	3/27	1.8 - 48	8.8		8.64E+01	1.1	CTPMGB	NO	BSL	
5103-71-9	alpha-Chlordane	0.6	J	130	J	ug/kg	SO-425	7/27	1.8 - 66	130		1.55E+03	66	CTPMGB	NO	BSL	
AROCLOLOR	Aroclor, Total	173.7	J	230000	J	ug/kg	SO-426	57/125	33 - 250	230000		1.98E+02		CTPMGB	YES	ASL	
12674-11-2	Aroclor-1016	200	J	200	J	ug/kg	SO-425	1/50	33 - 1300	200		1.98E+02		CTPMGB	YES	ASL	
11104-28-2	Aroclor-1221	22	J	27	J	ug/kg	SO-TP11	4/50	67 - 2600	27		1.98E+02		CTPMGB	NO	BSL	
53469-21-9	Aroclor-1242	13	J	3000	F	ug/kg	BF020	3/82	33 - 2000	3000		1.98E+02		CTPMGB	YES	ASL	
12672-29-6	Aroclor-1248	160	J	160	J	ug/kg	SO-425	1/82	33 - 1300	160		1.98E+02		CTPMGB	NO	BSL	
37324-23-5	Aroclor-1262	6.6	J	80000	*J	ug/kg	SO-426	24/82	33 - 750	80000		1.98E+02		CTPMGB	YES	ASL	
11100-14-4	Aroclor-1268	22	J	150000	*J	ug/kg	SO-426	52/125	33 - 10000	150000		1.98E+02		CTPMGB	YES	ASL	
319-85-7	beta-BHC	0.18	J	0.3	J	ug/kg	SO-427	2/27	1.8 - 66	0.3		3.02E+02	3.9	CTPMGB	NO	BSL	
60-57-1	Dieldrin	0.44	J	41	J	ug/kg	SO-TP18	5/27	3.6 - 130	41		2.78E+01	7	CTPMGB	YES	ASL	
659-96-8	Endosulfan I	0.093	J	14	J	ug/kg	SO-TP18	9/26	1.8 - 37	14		1.64E+05	8400	CTPMGB	NO	BSL	
1031-07-8	Endosulfan Sulfate	0.3	J	0.42	J	ug/kg	SO-423	2/26	3.6 - 130	0.42			8400	CTPMGB	NO	NTX	
72-20-8	Endrin	0.42	J	14	J	ug/kg	SO-TP18	6/27	3.6 - 130	14		8.18E+03	0	CTPMGB	NO	BSL	
53494-70-5	Endrin Ketone	1	J	30	J	ug/kg	SO-TP18	9/27	3.6 - 620	30			0	CTPMGB	NO	NTX	
58-89-9	gamma-BHC	0.082	J	1.1	J	ug/kg	SO-426	3/27	1.8 - 66	1.1		4.19E+02	40	CTPMGB	NO	BSL	
5103-74-2	gamma-Chlordane	0.079	J	220	J	ug/kg	SO-425	18/27	1.8 - 66	220		1.55E+03	66	CTPMGB	NO	BSL	
76-44-8	Heptachlor	0.058	J	130	J	ug/kg	SO-425	6/27	1.8 - 66	130		9.87E+01	13	CTPMGB	YES	ASL	
1024-57-3	Heptachlor Epoxide	0.73	J	38	J	ug/kg	SO-425	8/27	1.8 - 48	38		4.88E+01	20	CTPMGB	NO	BSL	
72-43-5	Methoxychlor	3.3	J	10	J	ug/kg	RMF G+050	2/26	1.8 - 660	10		1.36E+05	8000	CTPMGB	NO	BSL	
7429-90-5	Aluminum	4540	J	16200	J	mg/kg	RMF B+200	27/27	0 - 0	16200		3.75E+04		CTPMGB	NO	BSL	
7440-38-2	Arsenic	1.6	J	27	J	mg/kg	BF006	30/49	1.8 - 2.3	27		3.77E-01		CTPMGB	YES	ASL	
7440-39-3	Barium	30.5	J	18800	J	mg/kg	SO-TP15	32/49	40 - 40	18800		2.58E+03		CTPMGB	YES	ASL	
7440-41-7	Beryllium	0.23	J	1.2	J	mg/kg	RMF B+150	21/49	0.06 - 1	1.2		7.50E+01		CTPMGB	NO	BSL	
7440-43-9	Cadmium	0.47	J	5.3	J	mg/kg	SO-426	15/49	0.05 - 1	5.3		1.87E+01		CTPMGB	NO	BSL	
7440-70-2	Calcium	531	J	4590	J	mg/kg	SO-427	27/27	0 - 0	4590		3.01E+01		CTPMGB	YES	ASL	
7440-47-3	Chromium	11	J	234	J	mg/kg	SO-TP15	32/49	2 - 2	234		1.63E+03		CTPMGB	NO	BSL	
7440-48-4	Cobalt	3.1	J	39.5	J	mg/kg	SO-TP15	32/49	10 - 10	39.5		1.39E+03		CTPMGB	YES	ASL	
7440-50-8	Copper	9	J	45900	J	mg/kg	SO-426	31/49	5 - 73.3	45900		1.12E+04		CTPMGB	YES	ASL	
7439-89-6	Iron	9260	J	96200	J	mg/kg	SO-421	27/27	0 - 0	96200		2.00E+02		CTPMGB	YES	ASL	
7439-92-1	Lead	7.3	J	30700	J	mg/kg	SO-TP15	120/125	0.1 - 15.7	30700			114000	CTPMGB	NO	NTX	
7439-95-4	Magnesium	2130	J	114000	J	mg/kg	SO-TP15	27/27	0 - 0	114000		1.56E+03		CTPMGB	NO	BSL	
7439-96-5	Manganese	129	J	560	J	mg/kg	BF023A	32/49	3 - 3	560		1.10E+01		CTPMGB	NO	BSL	
7439-97-6	Mercury	0.057	J	6.2	J	mg/kg	SO-421	13/27	0.05 - 0.1	6.2		7.50E+02		CTPMGB	NO	BSL	
7440-02-0	Nickel	9	J	609	J	mg/kg	SO-426	32/49	8 - 8	609			3400	CTPMGB	NO	BSL	
7440-09-7	Potassium	147	J	3400	J	mg/kg	SO-426	24/27	287 - 409	3400					CTPMGB	NO	NTX
7782-49-2	Selenium	0.38	J	1.8	J	mg/kg	SO-TP18	4/27	0.1 - 1.3	1.8		1.87E+02		CTPMGB	NO	BSL	

TABLE D-10.2
 OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
 RAYMARK OU4 - BALLFIELD

Scenario Timeframe: Future
 Medium: Soil
 Exposure Medium: Soil
 Exposure Point: 0 to 4 feet

CAS Number	Chemical	Minimum Concentration ⁽¹⁾	Minimum Qualifier	Maximum Concentration ⁽¹⁾	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value ⁽²⁾	Screening Toxicity Value ⁽³⁾	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for Contaminant Deletion or Selection ⁽⁴⁾
7440-22-4	Silver	0.38	J	5.5		mg/kg	SO-426	9/21	0.25 - 1	5.5					NO	BSL
7440-23-5	Sodium	33.2	J	624		mg/kg	SO-TP15	24/26	28.8 - 65.8	624		1.87E+02		CTPMGB	NO	BSL
7440-28-0	Thallium	1	J	4.8		mg/kg	SO-421	3/27	0.18 - 85.6	4.8		2.51E+00		CTPMGB	NO	NTX
7440-62-2	Vanadium	12.9		93.6		mg/kg	SO-TP18	32/49	10 - 10	93.6		2.62E+02		CTPMGB	YES	ASL
7440-66-6	Zinc	28.8		9700		mg/kg	SO-426	32/49	4 - 4	9700		1.12E+04		CTPMGB	NO	BSL
ASBESTOS	Asbestos	0.9		50		%	SO-TP15	87/160	0.1 - 0.1	50		1.00E+00		CTPMGB	YES	ASL

(1) Minimum/maximum detected concentration

(2) N/A - Refer to supporting information for background discussion

(3) Region 9, Preliminary Remediation Goals, Residential Soil, May 1998

Non-cancer PRGs have been adjusted to a target Hazard Quotient of 0.5

PRG for Thallium was determined by TINUS using an adjusted RfD based on a molecular weight conversion from Thallium sulfate

(4) Rationale Codes Selection Reason

Infrequent Detection but Associated Historically (HIST)

Toxicity Information Available (1X)

Above Screening Levels (ASL)

cPAH family (CPAH)

No Toxicity Information (NTX)

Essential Nutrient (NUT)

Below Screening Level (BSL)

Deletion Reason

Definitions: N/A = Not Applicable

COPC = Chemical of Potential Concern

ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered

CTPMGB = Connecticut Pollutant Mobility Criteria for Soil in a GB area, Jan 1996

J = Estimated Value

F =

C = Carcinogenic

N = Non-Carcinogenic

TABLE D-10.3
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
RAYMARK OU4 - BALLFIELD

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Soil
Exposure Point: 0 to 4 feet

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
							Benzo(a)anthracene	ug/kg	360	600	2600	J
Benzo(a)pyrene	ug/kg	350	580	2600	J	ug/kg	700	95% UCL-L	W-Test()	700	95% UCL-L	W-Test()
Benzo(b)fluoranthene	ug/kg	340	530	2200	J	ug/kg	640	95% UCL-L	W-Test()	640	95% UCL-L	W-Test()
Benzo(k)fluoranthene	ug/kg	310	500	2100	J	ug/kg	480	95% UCL-L	W-Test()	480	95% UCL-L	W-Test()
Chrysene	ug/kg	440	710	2800	J	ug/kg	900	95% UCL-L	W-Test()	900	95% UCL-L	W-Test()
Dibenzo(a,h)anthracene	ug/kg	190	260	800	J	ug/kg	330	95% UCL-L	W-Test()	330	95% UCL-L	W-Test()
Indeno(1,2,3-cd)pyrene	ug/kg	290	500	2200	J	ug/kg	500	95% UCL-L	W-Test()	500	95% UCL-L	W-Test()
Aroclor, Total	ug/kg	5700	9500	230000		ug/kg	6300	95% UCL-L	W-Test()	6300	95% UCL-L	W-Test()
Dieldrin	ug/kg	8.9	14	41	J	ug/kg	14	95% UCL-L	W-Test()	14	95% UCL-L	W-Test()
Heptachlor	ug/kg	9.7	18	130		ug/kg	28	95% UCL-L	W-Test()	28	95% UCL-L	W-Test()
Arsenic	mg/kg	4.8	6.1	27		mg/kg	7	95% UCL-L	W-Test()	7	95% UCL-L	W-Test()
Barium	mg/kg	985	1770	18800	J	mg/kg	1130	95% UCL-L	W-Test()	1130	95% UCL-L	W-Test()
Chromium	mg/kg	27	39	234		mg/kg	76	95% UCL-L	W-Test()	76	95% UCL-L	W-Test()
Lead	mg/kg	1160	1780	30700		mg/kg	2880	95% UCL-L	W-Test()	2880	95% UCL-L	W-Test()
Thallium	mg/kg	2.2	5	4.8		mg/kg	2	95% UCL-L	W-Test()	2	95% UCL-L	W-Test()
Asbestos	%	3	4	50		%	4	95% UCL-L	W-Test()	4	95% UCL-L	W-Test()

For non-detects, 1/2 sample quantitation limit was used as a proxy concentration; for duplicate sample results, the maximum value was used in the calculation.
W-Test: Developed by Shapiro and Wilk, refer to Supplemental Guidance to RAGS: Calculating the Concentration Term, OSWER Directive 9285 7-081, May 1992.

Statistics: Maximum Detected Value (Max), 95% UCL of Normal Data (95% UCL-N), 95% UCL of Log-transformed Data (95% UCL-T), Mean of Log-transformed Data (Mean-T), Mean of Normal Data (Mean-N)

- (1) Shapiro-Wilk W-Test indicates data are log-normally distributed.
- (2) 95% UCL exceeds maximum detected concentration. Therefore, maximum concentration used for RME EPC, lesser of Mean-N or Max used for CTE EPC.
- (3) Shapiro-Wilk W Test indicates data are normally distributed.
- (4) < 20 sample results. Therefore, maximum concentration used for RME EPC, lesser of Mean-N or Max used for CTE EPC.

TABLE D-10.7a RME
 CALCULATION OF NON-CANCER HAZARDS - ADULT RESIDENT CONTACT WITH (0 - 4 FEET) SOIL
 REASONABLE MAXIMUM EXPOSURE
 RAYMARK OU-4 BALLFIELD

Scenario Timeframe: Future
 Medium Soil
 Exposure Medium: Soil
 Exposure Point: Contact with (0 - 4 feet) Soil
 Receptor Population: Resident
 Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient	
Ingestion	Arsenic	7.00E+00	mg/kg	7.00E+00	mg/kg	M	9.59E-06	mg/kg-day	3.00E-04	mg/kg-day	N/A	N/A	3.20E-02	
	Barium	1.13E+03	mg/kg	1.13E+03	mg/kg	M	1.55E-03	mg/kg-day	7.00E-02	mg/kg-day	N/A	N/A	2.21E-02	
	Cadmium	2.80E+00	mg/kg	2.80E+00	mg/kg	M	3.84E-06	mg/kg-day	1.00E-03	mg/kg-day	N/A	N/A	3.84E-03	
	Chromium	7.60E+01	mg/kg	7.60E+01	mg/kg	M	1.04E-04	mg/kg-day	3.00E-03	mg/kg-day	N/A	N/A	3.47E-02	
	Lead	2.88E+03	mg/kg	2.88E+03	mg/kg	M	3.95E-03	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Thallium	2.00E+00	mg/kg	2.00E+00	mg/kg	M	2.74E-06	mg/kg-day	7.00E-05	mg/kg-day	N/A	N/A	3.91E-02	
	Aroclor, Total	6.30E+03	ug/kg	6.30E+03	ug/kg	M	8.63E-06	mg/kg-day	2.00E-05	mg/kg-day	N/A	N/A	4.32E-01	
	Dieldrin	1.40E+01	ug/kg	1.40E+01	ug/kg	M	1.92E-08	mg/kg-day	5.00E-05	mg/kg-day	N/A	N/A	3.84E-04	
	Heptachlor	2.80E+01	ug/kg	2.80E+01	ug/kg	M	3.84E-08	ug/kg-day	5.00E-04	mg/kg-day	N/A	N/A	7.67E-05	
	Benz(a)anthracene	7.20E+02	ug/kg	7.20E+02	ug/kg	M	9.86E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Benzo(a)pyrene	7.00E+02	ug/kg	7.00E+02	ug/kg	M	9.59E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Benzo(b)fluoranthene	6.40E+02	ug/kg	6.40E+02	ug/kg	M	8.77E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Benzo(k)fluoranthene	4.80E+02	ug/kg	4.80E+02	ug/kg	M	6.58E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Chrysene	9.00E+02	ug/kg	9.00E+02	ug/kg	M	1.23E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Dibenz(a,h)anthracene	3.30E+02	ug/kg	3.30E+02	ug/kg	M	4.52E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Indeno(1,2,3-cd)pyrene (Total)	5.00E+02	ug/kg	5.00E+02	ug/kg	M	6.85E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Dermal	Arsenic	7.00E+00	mg/kg	7.00E+00	mg/kg	M	1.15E-06	mg/kg-day	3.00E-04	mg/kg-day	N/A	N/A	5.64E-01
		Barium	1.13E+03	mg/kg	1.13E+03	mg/kg	M	NA	mg/kg-day	4.90E-03	mg/kg-day	N/A	N/A	3.83E-03
Cadmium		2.80E+00	mg/kg	2.80E+00	mg/kg	M	1.53E-07	mg/kg-day	5.00E-05	mg/kg-day	N/A	N/A	NA	
Chromium		7.60E+01	mg/kg	7.60E+01	mg/kg	M	NA	mg/kg-day	7.50E-05	mg/kg-day	N/A	N/A	3.06E-03	
Lead		2.88E+03	mg/kg	2.88E+03	mg/kg	M	NA	mg/kg-day	--	mg/kg-day	N/A	N/A	NA	
Thallium		2.00E+00	mg/kg	2.00E+00	mg/kg	M	NA	mg/kg-day	7.00E-05	mg/kg-day	N/A	N/A	NA	
Aroclor, Total		6.30E+03	ug/kg	6.30E+03	ug/kg	M	4.82E-06	mg/kg-day	2.00E-05	mg/kg-day	N/A	N/A	2.41E-01	
Dieldrin		1.40E+01	ug/kg	1.40E+01	ug/kg	M	NA	mg/kg-day	5.00E-05	mg/kg-day	N/A	N/A	NA	
Heptachlor		2.80E+01	ug/kg	2.80E+01	ug/kg	M	NA	mg/kg-day	5.00E-04	mg/kg-day	N/A	N/A	NA	
Benz(a)anthracene		7.20E+02	ug/kg	7.20E+02	ug/kg	M	5.12E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Benzo(a)pyrene		7.00E+02	ug/kg	7.00E+02	ug/kg	M	4.97E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Benzo(b)fluoranthene		6.40E+02	ug/kg	6.40E+02	ug/kg	M	4.55E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Benzo(k)fluoranthene		4.80E+02	ug/kg	4.80E+02	ug/kg	M	3.41E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Chrysene		9.00E+02	ug/kg	9.00E+02	ug/kg	M	6.39E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Dibenz(a,h)anthracene		3.30E+02	ug/kg	3.30E+02	ug/kg	M	2.34E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Indeno(1,2,3-cd)pyrene (Total)		5.00E+02	ug/kg	5.00E+02	ug/kg	M	3.55E-07	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Total of Routes													8.12E-01	

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation

TABLE D-10.7b RME
 CALCULATION OF NON-CANCER HAZARDS - CHILD RESIDENT CONTACT WITH (0 - 4 FEET) SOIL
 REASONABLE MAXIMUM EXPOSURE
 RAYMARK OU-4 BALLFIELD

Scenario Timeframe: Future
 Medium: Soil
 Exposure Medium: Soil
 Exposure Point: Contact with (0 - 4 feet) Soil
 Receptor Population: Resident
 Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient	
Ingestion	Arsenic	7.00E+00	mg/kg	7.00E+00	mg/kg	M	8.95E-05	mg/kg-day	3.00E-04	mg/kg-day	N/A	N/A	2.98E-01	
	Banum	1.13E+03	mg/kg	1.13E+03	mg/kg	M	1.44E-02	mg/kg-day	7.00E-02	mg/kg-day	N/A	N/A	2.06E-01	
	Cadmium	2.80E+00	mg/kg	2.80E+00	mg/kg	M	3.58E-05	mg/kg-day	1.00E-03	mg/kg-day	N/A	N/A	3.58E-02	
	Chromium	7.60E+01	mg/kg	7.60E+01	mg/kg	M	9.72E-04	mg/kg-day	3.00E-03	mg/kg-day	N/A	N/A	3.24E-01	
	Lead	2.88E+03	mg/kg	2.88E+03	mg/kg	M	3.68E-02	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Thallium	2.00E+00	mg/kg	2.00E+00	mg/kg	M	2.56E-05	mg/kg-day	7.00E-05	mg/kg-day	N/A	N/A	3.65E-01	
	Aroclor, Total	6.30E+03	ug/kg	6.30E+03	ug/kg	M	8.05E-05	mg/kg-day	2.00E-05	mg/kg-day	N/A	N/A	4.03E+00	
	Dieldrin	1.40E+01	ug/kg	1.40E+01	ug/kg	M	1.79E-07	mg/kg-day	5.00E-05	mg/kg-day	N/A	N/A	3.58E-03	
	Heptachlor	2.80E+01	ug/kg	2.80E+01	ug/kg	M	3.58E-07	mg/kg-day	5.00E-04	mg/kg-day	N/A	N/A	7.16E-04	
	Benz(a)anthracene	7.20E+02	ug/kg	7.20E+02	ug/kg	M	9.21E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Benzo(a)pyrene	7.00E+02	ug/kg	7.00E+02	ug/kg	M	8.95E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Benzo(b)fluoranthene	6.40E+02	ug/kg	6.40E+02	ug/kg	M	8.18E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Benzo(k)fluoranthene	4.80E+02	ug/kg	4.80E+02	ug/kg	M	6.14E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Chrysene	9.00E+02	ug/kg	9.00E+02	ug/kg	M	1.15E-05	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Dibenz(a,h)anthracene	3.30E+02	ug/kg	3.30E+02	ug/kg	M	4.22E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	Indeno(1,2,3-cd)pyrene	5.00E+02	ug/kg	5.00E+02	ug/kg	M	6.39E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
	(Total)													5.26E+00
	Dermal	Arsenic	7.00E+00	mg/kg	7.00E+00	mg/kg	M	7.79E-06	mg/kg-day	3.00E-04	mg/kg-day	N/A	N/A	2.60E-02
		Banum	1.13E+03	mg/kg	1.13E+03	mg/kg	M	NA	mg/kg-day	4.90E-03	mg/kg-day	N/A	N/A	NA
Cadmium		2.80E+00	mg/kg	2.80E+00	mg/kg	M	1.04E-06	mg/kg-day	5.00E-05	mg/kg-day	N/A	N/A	2.08E-02	
Chromium		7.60E+01	mg/kg	7.60E+01	mg/kg	M	NA	mg/kg-day	7.50E-05	mg/kg-day	N/A	N/A	NA	
Lead		2.88E+03	mg/kg	2.88E+03	mg/kg	M	NA	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Thallium		2.00E+00	mg/kg	2.00E+00	mg/kg	M	NA	mg/kg-day	7.00E-05	mg/kg-day	N/A	N/A	NA	
Aroclor, Total		6.30E+03	ug/kg	6.30E+03	ug/kg	M	3.27E-05	mg/kg-day	2.00E-05	mg/kg-day	N/A	N/A	1.64E+00	
Dieldrin		1.40E+01	ug/kg	1.40E+01	ug/kg	M	NA	mg/kg-day	5.00E-05	mg/kg-day	N/A	N/A	NA	
Heptachlor		2.80E+01	ug/kg	2.80E+01	ug/kg	M	NA	mg/kg-day	5.00E-04	mg/kg-day	N/A	N/A	NA	
Benz(a)anthracene		7.20E+02	ug/kg	7.20E+02	ug/kg	M	3.47E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Benzo(a)pyrene		7.00E+02	ug/kg	7.00E+02	ug/kg	M	3.37E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Benzo(b)fluoranthene		6.40E+02	ug/kg	6.40E+02	ug/kg	M	3.08E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Benzo(k)fluoranthene		4.80E+02	ug/kg	4.80E+02	ug/kg	M	2.31E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Chrysene		9.00E+02	ug/kg	9.00E+02	ug/kg	M	4.34E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Dibenz(a,h)anthracene		3.30E+02	ug/kg	3.30E+02	ug/kg	M	1.59E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
Indeno(1,2,3-cd)pyrene		5.00E+02	ug/kg	5.00E+02	ug/kg	M	2.41E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--	
(Total)														1.68E+00
Total of Routes													6.94E+00	

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE D-10.8a RME
 CALCULATION OF CANCER RISKS - ADULT RESIDENT CONTACT WITH (0 - 4 FEET) SOIL
 REASONABLE MAXIMUM EXPOSURE
 RAYMARK OU-4 BALLFIELD

Scenario Timeframe: Future
 Medium Soil
 Exposure Medium Soil
 Exposure Point Contact with (0 - 4 feet) Soil
 Receptor Population Resident
 Receptor Age Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	7.00E+00	mg/kg	7.00E+00	mg/kg	M	3.29E-06	mg/kg-day	1.50E+00	1/(mg/kg-day)	4.93E-06
	Barium	1.13E+03	mg/kg	1.13E+03	mg/kg	M	5.31E-04	mg/kg-day	--	1/(mg/kg-day)	--
	Cadmium	2.80E+00	mg/kg	2.80E+00	mg/kg	M	1.32E-06	mg/kg-day	--	1/(mg/kg-day)	--
	Chromium	7.60E+01	mg/kg	7.60E+01	mg/kg	M	3.57E-05	mg/kg-day	--	1/(mg/kg-day)	--
	Lead	2.88E+03	mg/kg	2.88E+03	mg/kg	M	1.35E-03	mg/kg-day	--	1/(mg/kg-day)	--
	Thallium	2.00E+00	mg/kg	2.00E+00	mg/kg	M	9.39E-07	mg/kg-day	--	1/(mg/kg-day)	--
	Aroclor, Total	6.30E+03	ug/kg	6.30E+03	ug/kg	M	2.96E-06	mg/kg-day	2.00E+00	1/(mg/kg-day)	5.92E-06
	Dieldrin	1.40E+01	ug/kg	1.40E+01	ug/kg	M	6.58E-09	mg/kg-day	1.60E+01	1/(mg/kg-day)	1.05E-07
	Heptachlor	2.80E+01	ug/kg	2.80E+01	ug/kg	M	1.32E-08	mg/kg-day	4.50E+00	1/(mg/kg-day)	5.92E-08
	Benz(a)anthracene	7.20E+02	ug/kg	7.20E+02	ug/kg	M	3.38E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	2.47E-07
	Benzo(a)pyrene	7.00E+02	ug/kg	7.00E+02	ug/kg	M	3.29E-07	mg/kg-day	7.30E+00	1/(mg/kg-day)	2.40E-06
	Benzo(b)fluoranthene	6.40E+02	ug/kg	6.40E+02	ug/kg	M	3.01E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	2.19E-07
	Benzo(k)fluoranthene	4.80E+02	ug/kg	4.80E+02	ug/kg	M	2.25E-07	mg/kg-day	7.30E-02	1/(mg/kg-day)	1.65E-08
	Chrysene	9.00E+02	ug/kg	9.00E+02	ug/kg	M	4.23E-07	mg/kg-day	7.30E-03	1/(mg/kg-day)	3.09E-09
	Dibenz(a,h)anthracene	3.30E+02	ug/kg	3.30E+02	ug/kg	M	1.55E-07	mg/kg-day	7.30E+00	1/(mg/kg-day)	1.13E-06
	Indeno(1,2,3-cd)pyrene (Total)	5.00E+02	ug/kg	5.00E+02	ug/kg	M	2.35E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	1.71E-07
Dermal	Arsenic	7.00E+00	mg/kg	7.00E+00	mg/kg	M	3.94E-07	mg/kg-day	1.50E+00	1/(mg/kg-day)	5.90E-07
	Barium	1.13E+03	mg/kg	1.13E+03	mg/kg	M	NA	mg/kg-day	--	1/(mg/kg-day)	--
	Cadmium	2.80E+00	mg/kg	2.80E+00	mg/kg	M	5.25E-08	mg/kg-day	--	1/(mg/kg-day)	--
	Chromium	7.60E+01	mg/kg	7.60E+01	mg/kg	M	NA	mg/kg-day	--	1/(mg/kg-day)	--
	Lead	2.88E+03	mg/kg	2.88E+03	mg/kg	M	NA	mg/kg-day	--	1/(mg/kg-day)	--
	Thallium	2.00E+00	mg/kg	2.00E+00	mg/kg	M	NA	mg/kg-day	--	1/(mg/kg-day)	--
	Aroclor, Total	6.30E+03	ug/kg	6.30E+03	ug/kg	M	1.65E-06	mg/kg-day	2.00E+00	1/(mg/kg-day)	3.31E-06
	Dieldrin	1.40E+01	ug/kg	1.40E+01	ug/kg	M	NA	mg/kg-day	1.60E+01	1/(mg/kg-day)	NA
	Heptachlor	2.80E+01	ug/kg	2.80E+01	ug/kg	M	NA	mg/kg-day	4.50E+00	1/(mg/kg-day)	NA
	Benz(a)anthracene	7.20E+02	ug/kg	7.20E+02	ug/kg	M	1.75E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	1.28E-07
	Benzo(a)pyrene	7.00E+02	ug/kg	7.00E+02	ug/kg	M	1.71E-07	mg/kg-day	7.30E+00	1/(mg/kg-day)	1.24E-06
	Benzo(b)fluoranthene	6.40E+02	ug/kg	6.40E+02	ug/kg	M	1.56E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	1.14E-07
	Benzo(k)fluoranthene	4.80E+02	ug/kg	4.80E+02	ug/kg	M	1.17E-07	mg/kg-day	7.30E-02	1/(mg/kg-day)	8.54E-09
	Chrysene	9.00E+02	ug/kg	9.00E+02	ug/kg	M	2.19E-07	mg/kg-day	7.30E-03	1/(mg/kg-day)	1.60E-09
	Dibenz(a,h)anthracene	3.30E+02	ug/kg	3.30E+02	ug/kg	M	8.04E-08	mg/kg-day	7.30E+00	1/(mg/kg-day)	5.87E-07
	Indeno(1,2,3-cd)pyrene (Total)	5.00E+02	ug/kg	5.00E+02	ug/kg	M	1.22E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	8.89E-08
											2.13E-05

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation

Total of Routes 2.13E-05

TABLE D-10.8b RME
 CALCULATION OF CANCER RISKS - CHILD RESIDENT CONTACT WITH (0 - 4 FEET) SOIL
 REASONABLE MAXIMUM EXPOSURE
 RAYMARK OU-4 BALLFIELD

Scenario Timeframe: Future
 Medium Soil
 Exposure Medium: Soil
 Exposure Point: Contact with (0 - 4 feet) Soil
 Receptor Population: Resident
 Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk	
Ingestion	Arsenic	7.00E+00	mg/kg	7.00E+00	mg/kg	M	7.67E-06	mg/kg-day	1.50E+00	1/(mg/kg-day)	1.15E-05	
	Barium	1.13E+03	mg/kg	1.13E+03	mg/kg	M	1.24E-03	mg/kg-day	--	1/(mg/kg-day)	--	
	Cadmium	2.80E+00	mg/kg	2.80E+00	mg/kg	M	3.07E-06	mg/kg-day	--	1/(mg/kg-day)	--	
	Chromium	7.60E+01	mg/kg	7.60E+01	mg/kg	M	8.33E-05	mg/kg-day	--	1/(mg/kg-day)	--	
	Lead	2.88E+03	mg/kg	2.88E+03	mg/kg	M	3.16E-03	mg/kg-day	--	1/(mg/kg-day)	--	
	Thallium	2.00E+00	mg/kg	2.00E+00	mg/kg	M	2.19E-06	mg/kg-day	--	1/(mg/kg-day)	--	
	Aroclor, Total	6.30E+03	ug/kg	6.30E+03	ug/kg	M	6.90E-06	mg/kg-day	2.00E+00	1/(mg/kg-day)	1.38E-05	
	Dieldrin	1.40E+01	ug/kg	1.40E+01	ug/kg	M	1.53E-08	mg/kg-day	1.60E+01	1/(mg/kg-day)	2.45E-07	
	Heptachlor	2.80E+01	ug/kg	2.80E+01	ug/kg	M	3.07E-08	mg/kg-day	4.50E+00	1/(mg/kg-day)	1.38E-07	
	Benz(a)anthracene	7.20E+02	ug/kg	7.20E+02	ug/kg	M	7.89E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	5.76E-07	
	Benzo(a)pyrene	7.00E+02	ug/kg	7.00E+02	ug/kg	M	7.67E-07	mg/kg-day	7.30E+00	1/(mg/kg-day)	5.60E-06	
	Benzo(b)fluoranthene	6.40E+02	ug/kg	6.40E+02	ug/kg	M	7.01E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	5.12E-07	
	Benzo(k)fluoranthene	4.80E+02	ug/kg	4.80E+02	ug/kg	M	5.26E-07	mg/kg-day	7.30E-02	1/(mg/kg-day)	3.84E-08	
	Chrysene	9.00E+02	ug/kg	9.00E+02	ug/kg	M	9.86E-07	mg/kg-day	7.30E-03	1/(mg/kg-day)	7.20E-09	
	Dibenz(a,h)anthracene	3.30E+02	ug/kg	3.30E+02	ug/kg	M	3.62E-07	mg/kg-day	7.30E+00	1/(mg/kg-day)	2.64E-06	
	Indeno(1,2,3-cd)pyrene	5.00E+02	ug/kg	5.00E+02	ug/kg	M	5.48E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	4.00E-07	
	(Total)											3.55E-05
	Dermal	Arsenic	7.00E+00	mg/kg	7.00E+00	mg/kg	M	6.67E-07	mg/kg-day	1.50E+00	1/(mg/kg-day)	1.00E-06
		Barium	1.13E+03	mg/kg	1.13E+03	mg/kg	M	NA	mg/kg-day	--	1/(mg/kg-day)	--
		Cadmium	2.80E+00	mg/kg	2.80E+00	mg/kg	M	8.90E-08	mg/kg-day	--	1/(mg/kg-day)	--
Chromium		7.60E+01	mg/kg	7.60E+01	mg/kg	M	NA	mg/kg-day	--	1/(mg/kg-day)	--	
Lead		2.88E+03	mg/kg	2.88E+03	mg/kg	M	NA	mg/kg-day	--	1/(mg/kg-day)	--	
Thallium		2.00E+00	mg/kg	2.00E+00	mg/kg	M	NA	mg/kg-day	--	1/(mg/kg-day)	--	
Aroclor, Total		6.30E+03	ug/kg	6.30E+03	ug/kg	M	2.80E-06	mg/kg-day	2.00E+00	1/(mg/kg-day)	5.61E-06	
Dieldrin		1.40E+01	ug/kg	1.40E+01	ug/kg	M	NA	mg/kg-day	1.60E+01	1/(mg/kg-day)	NA	
Heptachlor		2.80E+01	ug/kg	2.80E+01	ug/kg	M	NA	mg/kg-day	4.50E+00	1/(mg/kg-day)	NA	
Benz(a)anthracene		7.20E+02	ug/kg	7.20E+02	ug/kg	M	2.97E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	2.17E-07	
Benzo(a)pyrene		7.00E+02	ug/kg	7.00E+02	ug/kg	M	2.89E-07	mg/kg-day	7.30E+00	1/(mg/kg-day)	2.11E-06	
Benzo(b)fluoranthene		6.40E+02	ug/kg	6.40E+02	ug/kg	M	2.64E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	1.93E-07	
Benzo(k)fluoranthene		4.80E+02	ug/kg	4.80E+02	ug/kg	M	1.98E-07	mg/kg-day	7.30E-02	1/(mg/kg-day)	1.45E-08	
Chrysene		9.00E+02	ug/kg	9.00E+02	ug/kg	M	3.72E-07	mg/kg-day	7.30E-03	1/(mg/kg-day)	2.71E-09	
Dibenz(a,h)anthracene		3.30E+02	ug/kg	3.30E+02	ug/kg	M	1.36E-07	mg/kg-day	7.30E+00	1/(mg/kg-day)	9.95E-07	
Indeno(1,2,3-cd)pyrene		5.00E+02	ug/kg	5.00E+02	ug/kg	M	2.07E-07	mg/kg-day	7.30E-01	1/(mg/kg-day)	1.51E-07	
(Total)												1.03E-05
Total of Routes											4.58E-05	

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation

TABLE D-10.8c RME
 CALCULATION OF CANCER RISKS - LIFETIME RESIDENT CONTACT WITH (0 - 4 FEET) SOIL
 REASONABLE MAXIMUM EXPOSURE
 RAYMARK OU-4 BALLFIELD

Scenario Timeframe: Future
 Medium Soil
 Exposure Medium: Soil
 Exposure Point: Contact with (0 - 4 feet) Soil
 Receptor Population: Resident
 Receptor Age: Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk	
Ingestion	Arsenic	7.00E+00	mg/kg	7.00E+00	mg/kg	M	1.10E-05	mg/kg day	1.50E+00	1/(mg/kg day)	1.64E-05	
	Barium	1.13E+03	mg/kg	1.13E+03	mg/kg	M	5.31E-04	mg/kg day	--	1/(mg/kg day)	--	
	Cadmium	2.80E+00	mg/kg	2.80E+00	mg/kg	M	1.32E-06	mg/kg day	--	1/(mg/kg day)	--	
	Chromium	7.60E+01	mg/kg	7.60E+01	mg/kg	M	3.57E-05	mg/kg day	--	1/(mg/kg day)	--	
	Lead	2.88E+03	mg/kg	2.88E+03	mg/kg	M	1.35E-03	mg/kg day	--	1/(mg/kg day)	--	
	Thallium	2.00E+00	mg/kg	2.00E+00	mg/kg	M	9.39E-07	mg/kg day	--	1/(mg/kg day)	--	
	Aroclor, Total	6.30E+03	ug/kg	6.30E+03	ug/kg	M	9.86E-06	mg/kg day	2.00E+00	1/(mg/kg day)	1.97E-05	
	Dieldrin	1.40E+01	ug/kg	1.40E+01	ug/kg	M	2.19E-08	mg/kg day	1.60E+01	1/(mg/kg day)	3.51E-07	
	Heptachlor	2.80E+01	ug/kg	2.80E+01	ug/kg	M	4.38E-08	mg/kg day	4.50E+00	1/(mg/kg day)	1.97E-07	
	Benz(a)anthracene	7.20E+02	ug/kg	7.20E+02	ug/kg	M	1.13E-06	mg/kg day	7.30E-01	1/(mg/kg day)	8.23E-07	
	Benzo(a)pyrene	7.00E+02	ug/kg	7.00E+02	ug/kg	M	1.10E-06	mg/kg day	7.30E+00	1/(mg/kg day)	8.00E-06	
	Benzo(b)fluoranthene	6.40E+02	ug/kg	6.40E+02	ug/kg	M	1.00E-06	mg/kg day	7.30E-01	1/(mg/kg day)	7.31E-07	
	Benzo(k)fluoranthene	4.80E+02	ug/kg	4.80E+02	ug/kg	M	7.51E-07	mg/kg day	7.30E-02	1/(mg/kg day)	5.49E-08	
	Chrysene	9.00E+02	ug/kg	9.00E+02	ug/kg	M	1.41E-06	mg/kg day	7.30E-03	1/(mg/kg day)	1.03E-08	
	Dibenz(a,h)anthracene	3.30E+02	ug/kg	3.30E+02	ug/kg	M	5.17E-07	mg/kg day	7.30E+00	1/(mg/kg day)	3.77E-06	
	Indeno(1,2,3-cd)pyrene	5.00E+02	ug/kg	5.00E+02	ug/kg	M	7.83E-07	mg/kg day	7.30E-01	1/(mg/kg day)	5.71E-07	
	(Total)											5.07E-05
	Dermal	Arsenic	7.00E+00	mg/kg	7.00E+00	mg/kg	M	1.06E-06	mg/kg day	1.50E+00	1/(mg/kg day)	1.59E-06
		Barium	1.13E+03	mg/kg	1.13E+03	mg/kg	M	NA	mg/kg day	--	1/(mg/kg day)	--
		Cadmium	2.80E+00	mg/kg	2.80E+00	mg/kg	M	5.25E-08	mg/kg day	--	1/(mg/kg day)	--
Chromium		7.60E+01	mg/kg	7.60E+01	mg/kg	M	NA	mg/kg day	--	1/(mg/kg day)	--	
Lead		2.88E+03	mg/kg	2.88E+03	mg/kg	M	NA	mg/kg day	--	1/(mg/kg day)	--	
Thallium		2.00E+00	mg/kg	2.00E+00	mg/kg	M	NA	mg/kg day	--	1/(mg/kg day)	--	
Aroclor, Total		6.30E+03	ug/kg	6.30E+03	ug/kg	M	4.46E-06	mg/kg day	2.00E+00	1/(mg/kg day)	8.91E-06	
Dieldrin		1.40E+01	ug/kg	1.40E+01	ug/kg	M	NA	mg/kg day	1.60E+01	1/(mg/kg day)	--	
Heptachlor		2.80E+01	ug/kg	2.80E+01	ug/kg	M	NA	mg/kg day	4.50E+00	1/(mg/kg day)	--	
Benz(a)anthracene		7.20E+02	ug/kg	7.20E+02	ug/kg	M	4.73E-07	mg/kg day	7.30E-01	1/(mg/kg day)	3.45E-07	
Benzo(a)pyrene		7.00E+02	ug/kg	7.00E+02	ug/kg	M	4.60E-07	mg/kg day	7.30E+00	1/(mg/kg day)	3.30E-06	
Benzo(b)fluoranthene		6.40E+02	ug/kg	6.40E+02	ug/kg	M	4.20E-07	mg/kg day	7.30E-01	1/(mg/kg day)	3.07E-07	
Benzo(k)fluoranthene		4.80E+02	ug/kg	4.80E+02	ug/kg	M	3.15E-07	mg/kg day	7.30E-02	1/(mg/kg day)	2.30E-08	
Chrysene		9.00E+02	ug/kg	9.00E+02	ug/kg	M	5.91E-07	mg/kg day	7.30E-03	1/(mg/kg day)	4.31E-09	
Dibenz(a,h)anthracene		3.30E+02	ug/kg	3.30E+02	ug/kg	M	2.17E-07	mg/kg day	7.30E+00	1/(mg/kg day)	1.58E-06	
Indeno(1,2,3-cd)pyrene		5.00E+02	ug/kg	5.00E+02	ug/kg	M	3.28E-07	mg/kg day	7.30E-01	1/(mg/kg day)	2.40E-07	
(Total)												1.64E-05
Total of Routes											6.70E-05	

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation

TABLE D-10.9a RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT RESIDENT EXPOSURE TO (0 - 4 FEET) SOIL
REASONABLE MAXIMUM EXPOSURE
RAYMARK OU-4 BALLFIELD

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient							
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total			
Soil	Soil	Contact with (0 - 4 feet) Soil	Arsenic	4.93E-06	--	5.90E-07	5.52E-06	Arsenic	Skin	3.20E-02	--	3.83E-03	3.58E-02			
			Barium	--	--	--	--	Barium	Cardiovascular/Kidney	2.21E-02	--	NA	2.21E-02			
			Cadmium	--	--	--	--	Cadmium	Kidney	3.84E-03	--	3.06E-03	6.90E-03			
			Chromium	--	--	--	--	Chromium	Kidney	3.47E-02	--	NA	3.47E-02			
			Lead	--	--	--	--	Lead	N/A	--	--	--	--			
			Thallium	--	--	--	--	Thallium	Blood	3.91E-02	--	NA	3.91E-02			
			Aroclor, Total	5.92E-06	--	3.31E-06	9.22E-06	Aroclor, Total	Skin/Eye	4.32E-01	--	2.41E-01	6.73E-01			
			Dieldrin	1.05E-07	--	NA	1.05E-07	Dieldrin	Liver	3.84E-04	--	NA	3.84E-04			
			Heptachlor	5.92E-08	--	NA	5.92E-08	Heptachlor	Liver	7.67E-05	--	NA	7.67E-05			
			Benz(a)anthracene	2.47E-07	--	1.28E-07	3.75E-07	Benz(a)anthracene	N/A	--	--	--	--			
			Benzo(a)pyrene	2.40E-06	--	1.24E-06	3.64E-06	Benzo(a)pyrene	N/A	--	--	--	--			
			Benzo(b)fluoranthene	2.19E-07	--	1.14E-07	3.33E-07	Benzo(b)fluoranthene	N/A	--	--	--	--			
			Benzo(k)fluoranthene	1.65E-08	--	8.54E-09	2.50E-08	Benzo(k)fluoranthene	N/A	--	--	--	--			
			Chrysene	3.09E-09	--	1.60E-09	4.69E-09	Chrysene	N/A	--	--	--	--			
			Dibenz(a,h)anthracene	1.13E-06	--	5.87E-07	1.72E-06	Dibenz(a,h)anthracene	N/A	--	--	--	--			
			Indeno(1,2,3-cd)pyrene	1.71E-07	--	8.89E-08	2.60E-07	Indeno(1,2,3-cd)pyrene	N/A	--	--	--	--			
			(Total)	1.52E-05	--	6.07E-06	2.13E-05	(Total)		5.64E-01	--	2.48E-01	8.12E-01			
							Total Risk Across Soil					Total Hazard Index Across Soil				
										2.13E-05						8.12E-01
							Total Risk Across All Media and All Exposure Routes					Total Hazard Index Across All Media and All Exposure Routes				
							2.13E-05						8.12E-01			

Total Blood HI =	3.91E-02
Total Cardiovascular HI =	2.21E-02
Total Eye HI =	6.73E-01
Total Kidney HI =	6.37E-02
Total Liver HI =	4.60E-04
Total Skin HI =	7.08E-01

TABLE D-10.9b RME
 SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD RESIDENT EXPOSURE TO (0 - 4 FEET) SOIL
 REASONABLE MAXIMUM EXPOSURE
 RAYMARK OU-4 BALLFIELD

Scenario Timeframe: Future
 Receptor Population: Resident
 Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient						
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total		
Soil	Soil	Contact with (0 - 4 feet) Soil	Arsenic	1.15E-05	--	1.00E-06	1.25E-05	Arsenic	Skin	2.98E-01	--	2.60E-02	3.24E-01		
			Barium	--	--	--	--	Barium	Cardiovascular/Kidney	2.06E-01	--	NA	2.06E-01		
			Cadmium	--	--	--	--	Cadmium	Kidney	3.58E-02	--	2.08E-02	5.66E-02		
			Chromium	--	--	--	--	Chromium	Kidney	3.24E-01	--	NA	3.24E-01		
			Lead	--	--	--	--	Lead	N/A	--	--	--	--		
			Thallium	--	--	--	--	Thallium	Blood	3.65E-01	--	NA	3.65E-01		
			Aroclor, Total	1.38E-05	--	5.61E-06	1.94E-05	Aroclor, Total	Skin/Eye	4.03E+00	--	1.64E+00	5.66E+00		
			Dieldrin	2.45E-07	--	NA	2.45E-07	Dieldrin	Liver	3.58E-03	--	NA	3.58E-03		
			Heptachlor	1.38E-07	--	NA	1.38E-07	Heptachlor	Liver	7.16E-04	--	NA	7.16E-04		
			Benzo(a)anthracene	5.76E-07	--	2.17E-07	7.93E-07	Benzo(a)anthracene	N/A	--	--	--	--		
			Benzo(a)pyrene	5.60E-06	--	2.11E-06	7.71E-06	Benzo(a)pyrene	N/A	--	--	--	--		
			Benzo(b)fluoranthene	5.12E-07	--	1.93E-07	7.05E-07	Benzo(b)fluoranthene	N/A	--	--	--	--		
			Benzo(k)fluoranthene	3.84E-08	--	1.45E-08	5.29E-08	Benzo(k)fluoranthene	N/A	--	--	--	--		
			Chrysene	7.20E-09	--	2.71E-09	9.91E-09	Chrysene	N/A	--	--	--	--		
			Dibenz(a,h)anthracene	2.64E-06	--	9.95E-07	3.64E-06	Dibenz(a,h)anthracene	N/A	--	--	--	--		
			Indeno(1,2,3-cd)pyrene	4.00E-07	--	1.51E-07	5.51E-07	Indeno(1,2,3-cd)pyrene	N/A	--	--	--	--		
			(Total)	3.55E-05	--	1.03E-05	4.58E-05	(Total)		5.26E+00	--	1.68E+00	6.94E+00		
Total Risk Across Soil							4.58E-05	Total Hazard Index Across Soil							6.94E+00
Total Risk Across All Media and All Exposure Routes							4.58E-05	Total Hazard Index Across All Media and All Exposure Routes							6.94E+00

Total Blood HI =	3.65E-01
Total Cardiovascular HI =	2.06E-01
Total Eye HI =	5.66E+00
Total Kidney HI =	5.87E-01
Total Liver HI =	4.30E-03
Total Skin HI =	5.99E+00

TABLE D-10.9c RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - LIFETIME RESIDENT EXPOSURE TO (0 - 4 FEET) SOIL
REASONABLE MAXIMUM EXPOSURE
RAYMARK OU-4 BALLFIELD

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Soil	Contact with (0 - 4 feet) Soil	Arsenic	1.64E-05	--	1.59E-06	1.80E-05	Arsenic	N/A	N/A	--	N/A	--
			Barium	--	--	--	--	Barium	N/A	N/A	--	N/A	--
			Cadmium	--	--	--	--	Cadmium	N/A	N/A	--	N/A	--
			Chromium	--	--	--	--	Chromium	N/A	N/A	--	N/A	--
			Lead	--	--	--	--	Lead	N/A	N/A	--	N/A	--
			Thallium	--	--	--	--	Thallium	N/A	N/A	--	N/A	--
			Aroclor, Total	1.97E-05	--	8.91E-06	2.86E-05	Aroclor, Total	N/A	N/A	--	N/A	--
			Dieldrin	3.51E-07	--	--	3.51E-07	Dieldrin	N/A	N/A	--	N/A	--
			Heptachlor	1.97E-07	--	--	1.97E-07	Heptachlor	N/A	N/A	--	N/A	--
			Benzo(a)anthracene	8.23E-07	--	3.45E-07	1.17E-06	Benzo(a)anthracene	N/A	N/A	--	N/A	--
			Benzo(a)pyrene	8.00E-06	--	3.36E-06	1.14E-05	Benzo(a)pyrene	N/A	N/A	--	N/A	--
			Benzo(b)fluoranthene	7.31E-07	--	3.07E-07	1.04E-06	Benzo(b)fluoranthene	N/A	N/A	--	N/A	--
			Benzo(k)fluoranthene	5.49E-08	--	2.30E-08	7.79E-08	Benzo(k)fluoranthene	N/A	N/A	--	N/A	--
			Chrysene	1.03E-08	--	4.31E-09	1.46E-08	Chrysene	N/A	N/A	--	N/A	--
			Dibenz(a,h)anthracene	3.77E-06	--	1.58E-06	5.35E-06	Dibenz(a,h)anthracene	N/A	N/A	--	N/A	--
			Indeno(1,2,3-cd)pyrene	5.71E-07	--	2.40E-07	8.11E-07	Indeno(1,2,3-cd)pyrene	N/A	N/A	--	N/A	--
			(Total)	5.07E-05	--	1.64E-05	6.70E-05	(Total)	--	--	--	--	--
				Total Risk Across Soil			6.70E-05	Total Hazard Index Across Soil					--
				Total Risk Across All Media and All Exposure Routes			6.70E-05	Total Hazard Index Across All Media and All Exposure Routes					--

TABLE D-10.10b RME
 RISK ASSESSMENT SUMMARY - CHILD RESIDENT EXPOSURE TO (0 - 4 FEET) SOIL
 REASONABLE MAXIMUM EXPOSURE
 RAYMARK OU-4 BALLFIELD

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Soil	Contact with (0 - 4 feet) Soil						Arsenic	Skin	2.98E-01	--	2.60E-02	3.24E-01
			(Total)	--	--	--	--	Aroclor, Total (Total)	Skin/Eye	4.03E+00	--	1.64E+00	5.66E+00
				Total Risk Across Soil					Total Hazard Index Across Soil				
				Total Risk Across All Media and All Exposure Routes				--	Total Hazard Index Across All Media and All Exposure Routes				
									5.99E+00				
									5.99E+00				

Total Eye HI =	5.66E+00
Total Skin HI =	5.99E+00

TABLE D-10.11
SUMMARY OF IEUBK LEAD MODELING RESULTS
CHILD (AGE 1 - 6) EXPOSURE TO SOIL
RAYMARK OU-4 BALLFIELD SITE

MEDIA	LEAD CONC. (MG/KG)	PERCENT OF POPULATION WITH BLOOD-LEAD > 10UG/DL
Soil (0 - 4 Feet)	1160	61.13

Model Assumptions:

Receptor: Child, Ages 12 to 84 months
RME Exposure point concentration: Arithmetic Mean
CTE Exposure point concentration: Arithmetic Mean
Drinking water lead concentration: 4 ug/L (model default concentration)

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
 Indoor AIR Pb Conc: 30.0 percent of outdoor.

Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
 WATER Consumption: DEFAULT

SOIL & DUST:

Soil: constant conc.
 Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	1160.0	1160.0
1-2	1160.0	1160.0
2-3	1160.0	1160.0
3-4	1160.0	1160.0
4-5	1160.0	1160.0
5-6	1160.0	1160.0
6-7	1160.0	1160.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
 Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	12.7	24.54	22.14
1-2:	14.7	36.49	33.67
2-3:	13.9	38.21	34.96
3-4:	13.4	39.51	36.22
4-5:	11.3	32.48	29.00
5-6:	9.6	30.71	26.90
6-7:	8.6	30.00	25.82

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
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0.5-1:	2.07	0.30	0.00	0.02
1-2:	2.07	0.72	0.00	0.03
2-3:	2.41	0.77	0.00	0.06
3-4:	2.41	0.82	0.00	0.07
4-5:	2.50	0.92	0.00	0.07
5-6:	2.72	1.00	0.00	0.09
6-7:	3.06	1.03	0.00	0.09

