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The Development of Applied Action Levels for Soil Contact: A Scenario for the Exposure of Humans to Soil in a Residential Setting

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The California Site Mitigation Decision Tree Manual, 1985, was developed by the California Department of Health Services to provide a detailed technical basis for managing uncontrolled hazardous waste sites. The Decision Tree describes a process that relies on criteria, Applied Action Levels (AALs) to evaluate and, if necessary, mitigate the impact of uncontrolled hazardous waste sites on the public health and the environment. AALs are developed for individual substances, species, and media of exposure. AALs have been routinely developed for the media of air and water; however, an approach for developing AALs for soil contact was lacking. Given that the air pathway for soil contact is addressed in AALs for air, two routes of exposure, ingestion and dermal contact, are addressed in developing AALs for soil contact. The approach assumes a lifetime of exposure to soil in a residential setting. Age-related changes in exposure are included in the scenario.

Exposure to soil due to ingestion and dermal contact are quantitated independently and then integrated in the final exposure scenario. A mass balance approach using four elements is employed to quantitate soil ingestion for a young child. Changes in soil ingestion with age are based on age-related changes in blood lead concentration and mouthing behavior. Dermal exposure to soil was determined from studies that reported skin soil load and from estimates of exposed skin surface area. Age-related changes in the dermal exposure to soil are also based on changes with age of blood lead concentration and mouthing behavior.

The estimates of exposure to soil due to ingestion and dermal contact are integrated, and an approach for developing AALs is advanced. AALs are derived by allocating the Maximum Exposure Level as described in the Decision Tree to the average daily exposure to soil. Toxicokinetic considerations for the two routes of exposure must be included in deriving AALs for the soil medium of exposure.

Introduction

Hazardous substances can cause adverse effects in humans only if exposure occurs. Exposure to toxic substances can occur due to contact with various contaminated media. Depending on the magnitude of the exposure to the contamination in the medium, adverse public health consequences could be realized.

Standards and other criteria have been developed to protect the public health from risks associated with the contamination of the environment by toxic substances. The California Site Mitigation Decision Tree Manual (*1*), which provides in detail an approach for managing uncontrolled hazardous waste sites, employs Applied Action Levels (AALs) to evaluate the implications of toxic substance contamination and to develop site-specific mitigation plans. AALs are developed for each medium of exposure. A methodology for developing AALs for drinking water and for ambient air is outlined in the California Site Mitigation Decision Tree Manual.

Soil as a Medium of Exposure

Soil has recently been recognized as a potentially important medium of exposure. A substantial body of evidence has linked risks of adverse health effects with the exposure of humans to contamination in soil. To date, there is no standard approach for evaluating risks associated with exposure to contaminated soils. Standards or other criteria for the evaluation of contamination in soil are lacking. Several approaches for evaluating the implications of soil contamination have been proposed (*2–7*). Unfortunately, these ap-

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proaches rely on assumptions that are at best extremely tenuous. Although there is no conventional mechanism available to develop soil criteria, there is little doubt that soil criteria are needed if the public health is to be protected.

Exposure Scenarios

Fundamental to developing a standard for any medium is to derive a reasonable exposure scenario. How much of a medium and by which routes will a human be exposed? For air, various agencies have employed 20 m^3/day as a reasonable estimate of exposure. For drinking water, a reasonable estimate of exposure for an adult is 2 L/day and for a young child, 1 L/day. Establishing a reasonable estimate of exposure to soil is requisite to the development of AALs for soil contact.

AALs for soil contact will be based on the following exposure scenario of human-to-soil in a residential setting. It should be understood that the approach will employ limited data and relay on certain assumptions when adequate data are not available. Clearly, much research is required to reduce the uncertainties associated with developing a soil exposure scenario.

Major Issues to be Addressed in Developing a Soil Exposure Scenario

Ample evidence is available that demonstrates contaminated air or water has resulted in significant exposure of humans to toxic chemicals. The evidence that contamination in soil can result in significant human exposure and thereby adversely impact the public health is much more limited. Studies that demonstrate significant exposure of humans to contamination in soil in residential settings are first reviewed. The evidence that soil can be an important medium of exposure stems almost exclusively from numerous studies of individuals residing in areas contaminated with lead. A series of investigations that focused on the exposure of children to lead are first reviewed, then studies that evaluated the exposure of adults to lead contamination are reviewed.

Once the need for soil AALs is demonstrated, methodology to quantify exposure to soil is presented. The quantification of exposure to soil is fundamental to developing health-based criteria for toxic contaminants in soil. An approach to estimate exposure to soil via ingestion is introduced. Measurements of four elements from two studies are employed in a mass-balance approach to estimate soil ingestion. A methodology to estimate exposure to soil via dermal contact is then presented. Measurements of soil loading on skin from three studies are employed to develop an estimate of dermal exposure to soil. An exposure scenario is advanced that integrates the estimate of exposure to soil via the oral and dermal routes. An equation to develop AALs for soil contact that are used in conjunction with the California Site Mitigation Decision Tree Manual (*1*) is based on the exposure scenario is then prescribed.

AALs for Soil Contact

The development of AALs for soil contact would be unnecessary if no significant exposure of humans to contaminants in soil occurred. Unfortunately, there is substantial evidence linking soil contamination with significant human exposure. Most of the available evidence is from studies that attempted to identify various sources of lead exposure.

It should be understood that the purpose of the following discussion is to demonstrate that there is substantial evidence linking soil contamination with human exposure. The discussion is not directed at comprehensively reviewing the scientific literature on lead exposure or determining the relative contributions of various sources of lead (Pb) exposure.

Evidence Linking Soil Contamination with Significant Human Exposure

At one time it was assumed that the elevated blood lead level (PbB) observed in children resulted from the ingestion of Pb-ladened paint chips. For children with very high PbB levels, particularly children with signs of Pb intoxication, the severe exposure to Pb probably results mainly from the ingestion of paint chips (*8*). However, for children with moderately elevated PbB levels, it is becoming clear that excessive lead exposure probably results from contact with contaminated air, house dust, or soil. The number of studies that demonstrate that these sources are important sources of Pb exposure is substantial.

Studies in Children

In a study of 51 inner-city and 51 suburban children between 9 months and 6 years of age, in Rochester, NY, house dust and towel wipes of children's hands were analyzed for Pb content (*9*). Dust samples from the residences of inner-city children, children with elevated PbB, contained significantly more Pb than dust from houses of children with low PbB levels. More Pb was also detected in the handwipe samples of children from inner-city homes.

The investigators segregated data from inner-city homes with high paint Pb levels into homes with peeling paint and homes where no peeling paint was observed. The Pb levels in house dust were essentially identical in these two groups of homes.

In a study of 377 children between 1 and 72 months of age in New Haven, CT, blood, soil, indoor and

outdoor paint, and house dust were analyzed for Pb content (*10*). Substantial amounts of Pb were detected in soil, paint, and house dust. The investigators observed that PbB levels in the children correlated with the following variables (in descending order of importance): (a) soil Pb levels next to the residence; (b) soil Pb levels not immediately adjacent to the residence; (c) Pb content of exterior house paint; and (d) Pb content of house dust.

House dust and handwipes of children from suburban and inner-city residences were analyzed for Pb content (*11*). Pb levels in the handwipe samples (micrograms of lead per hand towel) correlated closely with the level of Pb in the floor dust samples. Suburban residences had a lower range of Pb levels in floor dust than inner-city residences. Pb levels in older city residences were substantially higher than in newer residences. These findings mirror the pattern of elevated PbB levels in inner-city children and support the hypothesis that house dust is an important source of Pb exposure in small children.

PbB levels of 20 children of Pb workers, 1 to 6 years old were compared to 17 children of neighboring families where no family member worked in a Pb plant (*12*). Pb levels in house paint and house dust samples were also determined. Increased levels of PbB in children corresponded with increased levels of Pb in house dust in residences of Pb workers. No relationship between PbB levels and Pb in painted surfaces was observed.

In a study of three groups of about 35 children from different ethnic backgrounds in London whose ages ranged from 2.5 to 5 years, blood, diet, and tap water samples were analyzed for Pb content (*13*). Handwashing and the parents' smoking habits were also evaluated. Of the factors analyzed, handwashing and the parents smoking habits were correlated with PbB levels. Pb intake from the diet was similar in all three groups. These findings are consistent with dust and soil contamination on a child's hand being an important source of exposure to Pb.

A study conducted in the Netherlands investigated various media as sources of Pb exposure in children residing in the vicinity of a Pb smelter (*14*). The lead content of ambient air, soil, tap water, floor dust, dust fall (indoor and outdoor), and blood of 95 children (age range 1 year to 3 years) was determined. Other parameters that were investigated including mouthing behavior, time spent indoors and outdoors, and the dustiness of each house. A few old houses with peeling paint and high Pb levels in house dust were excluded from the study. PbB levels in the children were most closely correlated with: Pb in outdoor dust fall, soil Pb levels, Pb in indoor dust fall, dustiness of the home, and mouthing behavior.

Over 800 children between the ages of 1 and 18 years in Omaha, NE, participated in a study aimed at identifying sources of Pb exposure (*15*). Children from three neighborhoods, one in the vicinity of a battery plant, one adjacent to downtown Omaha, and one in a suburban location, were included in the study. Samples of blood, ambient air, cows' milk, tap water, house dust, dust fall, and soil were analyzed for Pb content. The mean concentration of PbB was highest in children from residences adjacent to the battery plant and lowest in residences in a suburban setting. The pattern of Pb content of air, dust fall, soil, and house dust in each of the areas corresponded to the pattern of PbB. For children aged 1 through 5 and 6 through 15 years, PbB levels correlated closest with soil and house dust Pb content. In children aged 6 to 15 years, PbB concentration correlated with Pb content of air, soil, and house dust.

In a large study of over 1000 children residing near a Pb smelter in Idaho, the relationships between PbB levels and a variety of parameters were investigated (*16*). The study grouped the children into age categories and into regions. The regions were based on the distance between the children's residences and the smelter. The parameters that were investigated included Pb levels in ambient air, soil, paint, the father's occupation, household dust levels, the prevalence of pica, and the sex of the child.

Of the parameters investigated, the children's PbB levels correlated with the Pb content of ambient air, soil, house dust levels, the occupation of the father, the prevalence of pica (at age 2), and sex of the child (at age 7 and 8). The strong correlation between PbB levels and Pb in ambient air, soil, and house dust could be related. Significant exposure to Pb may have occurred from direct contact with soil or house dust, with air acting as the source of Pb in these media. The Pb content in house paint was not a strong pedictor of PbB levels in children participating in this study.

Two groups of children from the same inner-city neighborhood in Rochester, NY, were the subject of a study aimed at identifying various sources of Pb exposure (*17*). One group of children ranging in age from 18 to 72 months had PbB levels between 40 and 79 μg/100 mL and a second group of children from the same age range had PbB levels below 30 μg/100 mL. Soil, paint, house dust, and handwipe samples were analyzed for Pb content. The prevalence of pica and mouthing behavior was also studied. The following parameters were associated with the higher level of PbB observed in the children: household dust Pb content, soil Pb levels, the Pb content of the handwipe samples, mouthing behavior, and pica. An analysis of the data by age group indicated that household dust and soil Pb content were the best predictors of PbB levels in children between the ages of 18 and 32 months. The results of this study indicate that soil and house dust are a significant source of Pb exposure in young children.

Four distinct environmental settings in Wales were evaluated for their impact on PbB levels in children aged 1 to 3 years (*18*). The children enrolled in this study resided in four areas: (a) areas with heavy vehicular traffic; (b) adjacent areas (50–250 m distance) to the areas of high vehicular traffic; (c) an area where

Pb was mined in the past (high soil lead content); and (d) a remote area with light vehicular traffic and no history of Pb mining. Indoor air, soil, pavement dust, tap water, handwipe, and blood samples were analyzed for Pb content. The prevalence of pica was also studied.

The PbB levels were elevated in children from the mining area but not from the other three environmental settings. House dust and soil Pb content were also elevated in the mining area. However, indoor air Pb concentration was highest at the two sites associated with high vehicular traffic. No consistent association between pica and PbB level was observed at any of the locations. Analysis of the data resulted in a good correlation between the Pb content on a child's hand in the mining area and child's PbB levels. This relationship was not observed in the other three areas.

More than 600 children in Australia were evaluated to determine the relationship between PbB and soil Pb levels (*19*). The study enrolled approximately 90% of the children born in the vicinity of a smelter. PbB concentration was determined at age 6 months, 15 months, 2, 3, and 4 years in each child who remained in the study. Soil PbB content was also determined. PbB levels were higher in children who resided in areas with elevated soil Pb content. This study was consistent with the hypothesis that soil contamination is an important source of Pb exposure for children.

The relationship between various types of housing and PbB content of children was evaluated in a study conducted in Cincinnati (*20*). Inner-city housing was categorized by its age, condition, and by public or private ownership. Paint, interior dust, interior dust fall, exterior dust scrapings, and handwipe samples from the children were analyzed for Pb content.

Elevated PbB levels in the children were associated with certain categories of housing. The relative concentrations of Pb detected in interior dust, dust fall, or on the children's hands mirrored the pattern of elevated blood Pb levels. Other parameters did not appear to be as closely associated with the PbB levels. The results of this study are consistent with the hypothesis that dust or soil are important media of exposure to Pb for young children. The investigators indicated that the lack of correlation between lead content of paint in the housing and PbB levels suggests that other sources of Pb in the immediate neighborhood are important.

Children aged 1 through 9 years who lived within 1.6 km of smelters in El Paso, TX, or Kellogg, ID, were grouped according to PbB concentrations (*21*). Samples of interior paint, air, soil, house dust, food, and water were analyzed for Pb content in order to identify significant sources of lead exposure. House dust and soil lead levels were elevated in areas where the PbB levels were high. The concentration of lead in air also correlated with elevated PbB levels. No relationship between the Pb content of paint and blood lead levels was observed.

Investigators employed a stable isotope ratio method to evaluate sources of Pb exposure in children from two residences in Oakland, CA (*22*). Various sources of Pb contain different ratios of stable Pb isotopes. By using lead isotopes, the study could identify likely sources of Pb exposure in the children. Samples of blood, air, exterior and interior paint, house dust, soil, and gasoline were analyzed for their Pb isotope content.

In one residence, blood samples from 10 children were analyzed. The ratio of Pb isotopes in blood was similar to that observed in house dust, soil near the residence, and in exterior house paint. The ratio of Pb isotopes in interior house paint, gasoline, and ambient air were substantially different than that observed in the blood samples.

In a second residence, the ratio of Pb isotopes in blood from twin boys was similar to the isotope ratio in soil where the children usually played. The isotope ratio was also similar to nearby exterior paint samples, indicating a possible source of the soil Pb. The ratio of Pb isotopes in interior dust was not similar to that observed in the children's blood. In children from both residences, the Pb isotope ratio in blood closely matched that of soil where the children played or of soil adjacent to their residence.

The identification of significant sources of Pb exposure was the aim of a study that enrolled 249 infants in the greater Boston area (*23*). The PbB levels of infants were monitored for 2 years after birth. In addition, samples of tap water, paint, indoor air, house dust, and soil were analyzed for Pb content. At certain ages, Pb levels in indoor air and paint correlated marginally with PbB levels. Pb in tap water was not predictive of PbB concentrations at any age. The highest correlations with PbB levels were the Pb content of house dust or soil. Pb content of soil and house dust were strongly intercorrelated. The findings of the study demonstrated a relationship between PbB concentration and the level of Pb in house dust and soil. Indoor paint did not appear to be a significant source of Pb exposure for this group of children.

A study of 18 children between the ages of 6 and 13 years evaluated the relationship of certain parameters and several indicators of Pb exposure (*24*). Blood, soil, and paint samples were analyzed for Pb content. Neither soil nor paint lead levels correlated with the PbB levels in these children. While these findings may be at variance with the results of other studies, an older age group of children was evaluated in this study. The older children probably ingest less foreign matter than do the young children.

In Los Angeles, 441 individuals from an area adjacent to a freeway were enrolled in a study that evaluated potential sources of Pb exposure (*25*). Also enrolled in the study were 442 individuals from a reference community, Lancaster, CA. The participants were grouped into three age ranges, 1 through 16 years, 17 through 34 years, and 35 years and older. Samples of blood, air, soil, and tap water were analyzed for Pb content. The PbB concentration of males between the ages of 1 and 34 years and all three female age groups were elevated in Los Angeles when compared to

individuals from the reference population. Pb content of air and soil, but not tap water, was also elevated in Los Angeles. The findings of this study are consistent with the view that soil and air are significant sources of exposure to Pb.

A study of school children indicated that an older age group (age range 10–14 years) could be affected by exposure to substances in soil (*26*). Three groups of children, one group living and attending school within 1 km of the smelter, a second group living 1.5 km and attending school 2.5 km from the smelter, and a rural control group were evaluated for exposure to Pb. The study was conducted in 1973 and then repeated in 1975. Airborne Pb concentrations in the vicinity of the smelter were monitored from 1973 to 1975.

PbB levels in children living within 1 km of the smelter were substantially elevated when compared to children living 2.5 km from the smelter or children from a rural environment. PbB levels measured in children living approximately 2.5 km from the smelter were significantly lower in 1975 than the levels observed in 1973. This observation corresponded to the decrease in airborne Pb levels during the same time period. However, PbB levels in children living within 1 km of the smelter were not significantly different in 1975 when compared to 1973 levels, even though airborne Pb levels were markedly lower in 1975.

These findings indicated that children who resided within 1 km of the smelter were exposed to an important source of Pb in addition to Pb in air. The investigators analyzed Pb in soil samples from the three areas. Pb levels in soil near the smelter were markedly elevated when compared to levels observed 5 km from the smelter or in the rural area. The authors suggest that ingestion of Pb in dust or dirt may represent a significant source of Pb exposure for children living within 1 km of the smelter.

Various sources of Pb exposure were evaluated in a study of older children, age 9 through 14, who attended school less than 1 km or 2.5 km from the Pb smelter in Belgium (*27*). A matched control group of rural children were also evaluated in the study. Ambient air, blood, playground soil, and samples collected from the hands of children were analyzed for their Pb content.

The blood, air, and soil Pb levels within 1 km of the smelter were substantially above levels observed at a distance of 2.5 km, which, in turn, were substantially above levels in the rural environment. The pattern of concentrations of Pb in the blood of the children at the three locations mirrored the pattern of Pb levels in air observed in the three environments. These investigators also determined Pb levels on the hands of children at playgrounds at the three schools. The pattern of Pb detected in playground soils and on the hands of children mirrored that observed for Pb concentration in blood from children participating in this study. Pb detected on a child's hands was more closely associated with elevated PbB levels than the Pb concentrations in the ambient air. In the vicinity of the smelter, air Pb contamination may cause elevated PbB levels in children indirectly through contamination of surface soils. The results of this study are consistent with soil being a significant medium of exposure to Pb for children.

Studies in Adults

Although the PbB levels in adults are generally much lower than those observed in children, elevated levels of Pb in soil have been associated with elevated adult PbB levels. In one study, mothers occupying the same dwelling had much lower PbB levels than those observed in children (*28*). However, the PbB levels in mothers from highly contaminated areas appeared to be elevated above PbB levels of mothers from areas of minimal lead contamination.

Mothers from residences in an area of high soil Pb levels were observed to have elevated PbB levels (*18*). However, the PbB concentrations of the mothers were much lower than that observed in children from the same area.

The findings of a study of residents occupying dwellings adjacent to a freeway indicated that adults could be subject to exposure to Pb via contaminated soil (*25*). The PbB levels of adult females, but not males, were significantly elevated in an area of substantial Pb contamination. However, PbB in both adult males and females were below that observed in children from the same area.

The low PbB levels in adults may not be entirely related to less exposure to contaminated media. Studies of Pb absorption in adults and children indicate that adults absorb approximately 10% of ingested Pb (*29*), whereas children absorb approximately 40 to 50% of ingested Pb (*30,31*). These observations could account for the small increases in PbB levels in adults from areas of high Pb contamination.

Summary

Children living in an environment contaminated by Pb often have significantly elevated PbB levels. Numerous studies of children residing adjacent to smelters or major highways, in old mining areas, or in old inner-city neighborhoods have consistently demonstrated increased PbB levels when compared to children from reference communities. These areas are characterized by elevated levels of Pb in the environment. Numerous studies have linked the PbB content of young children with the Pb content of soil or house dust (*9,10,12,14–23,25,26*). These studies indicate that exposure to Pb via contaminated soil or house dust is responsible for a significant amount of the Pb burden in children.

Pica or the mouthing of hands and foreign objects is very characteristic of children. Ingestion of Pb-contaminated soil or house dust that coats foreign objects of a child's hands appears to represent an important

source of Pb exposure. In a variety of studies, there is a strong correlation between PbB level and the amount of Pb detected on a child's hand (*9,17,18,20,27*). Pb contamination in soil or house dust is probably the source of the Pb found on the children's hands (*11,32*).

Pb-laden airborne particulates or outdoor weathered paint appear to be major sources of the Pb contamination in house dust or soil. A variety of studies have correlated PbB levels with airborne Pb or Pb in weathered paint (*15,16,21,22,26*). Several studies have indicated that the exposure to Pb occurred via contact with soil and not directly from air or paint. A reduction of airborne Pb levels did not result in appreciable reduction of PbB content in children living adjacent to a smelter (*26,27*). The Pb content of blood correlated with the Pb content of house dust, soil, or outdoor paint, but not with indoor paint (*10,22*).

Taken together, these studies that focused on identifying major sources of Pb exposure indicate that soil can be an important medium of human exposure. Exposure to soil that is contaminated with Pb can result in exposure of the public. Other toxic substances that persist in soil and to which exposure could occur may also present a threat to public health. The studies that focused on Pb exposure demonstrate a need to evaluate the potential impact of a variety of soil contaminants on the public health.

Relationship Between Age and Risk of Exposure to Soil Contamination

The amount of exposure to toxic substances in soil appears to be related to age. Children appear to undergo a higher level of exposure to toxic substance in soil than adults. This higher level of exposure of children to soil contaminants is consistent with studies of pica and mouthing behavior and is supported by the findings of investigators of age-related changes of PbB levels in children.

Relationship Between Age, Pica, and Mouthing Tendencies

Pica has been defined as an abnormal ingestion of substances not usually considered food (*8*). Other definitions include the ingestion of foreign substances by children that occurs during the course of normal development (*33*). In addition to pica, children also exhibit mouthing tendencies. Typically, children place their fingers, palms, and various objects into the mouth. This behavior allows exposure of the child to toxic substances that contaminate soil.

A large study conducted in Boston evaluated the prevalence of pica in children (*34*). Families were selected and either interviewed or sent a questionnaire in the mail. At age 1 year, approximately 40% of all children in this study exhibited pica. Less than 10% exhibited this behavior at age 4 or 5 years. Black children exhibited a substantially higher prevalence of pica than white children.

The prevalence of mouthing behavior was greater than the prevalence of pica. In one study, approximately 80% of all children at age 1 year exhibited mouthing tendencies. This behavior declined to approximately 40% of all children by age 4 years.

The prevalence of pica and mouthing tendencies was investigated in children from the Washington, DC area (*35*). The relationship between age, ethnic background, and the prevalence of pica was studied. Pica tendencies were noted in approximately 55% of black children and 28% of white children between the ages of 1 and 2 years. This behavior declined to approximately 20% of the black children and 2% of white children between the ages of 3 and 4 years, and 17% of black children and 2% of white children between the ages of 5 and 6 years. Approximately 80% of the white or black children exhibited mouthing behavior between the ages of 1 and 2 years. By age 6 years, approximately 40% of black children and 15% of white children exhibited mouthing behavior.

Relationship Between Age and PbB Content

A number of investigators have studied the relationship between the age of a child and PbB content. Sayre and co-workers examined the PbB concentration of children from inner-city or suburban households (*9*). The study was limited to children between the ages of 9 months and 6 years. These investigators reported that the mean PbB level of children from inner-city residences was elevated and remained relatively constant between ages 2 and 4 years. The mean PbB level then declined slightly by year 5, but continued to be elevated above the control group.

The relationship between age and the PbB content of children was investigated in various areas in the vicinity of a smelter in Idaho (*36*). In most of the areas that were studied, the mean PbB level of children peaked between the ages of 2 and 4 years and then declined steadily with age.

A study conducted in Cincinnati focused on the relationship between housing types, PbB content, and the age of children (*20*). In all housing types, the mean PbB level in children increased until the age of 18 months, the oldest age group of children reported in this study. The largest increase in PbB level occurred in children who resided in pre-World War II, deteriorated structures.

The relationship between age, sex, and PbB levels in children residing in the vicinity of a smelter in Port Pirie, Australia, was evaluated (*19*). In both boys and girls, the mean PbB levels peaked at 2 years of age and steadily declined in 3- and 4-year-old children.

Discussion

The findings of studies that investigated the relationship between age and PbB levels in children indicate that PbB concentration peaks between the ages of 2 and 4 years. A pattern of increasing Pb content has been observed by the first year and in certain environments remained elevated until 6 or 7 years of age.

These findings are consistent with the prevalence of pica or mouthing behavior in children. Pica and mouthing tendencies were substantial in 1-year-old children. Both pica and mouthing tendencies decline markedly by age 5. However, mouthing or thumb sucking behavior was still evident in some children at age 6 or 7 years.

Exposure Scenario for Soil Contact

A variety of studies have demonstrated that contaminated soil may result in substantial human exposure to toxic chemicals. Three routes of exposure, ingestion, inhalation, or dermal contact, could contribute to human exposure to toxic substance in soil. Any methodology designed to develop criteria or standards for soil as a medium of exposure should address these three routes of exposure. The evaluation of exposure to soil via the inhalation of soil particles is addressed employing AALs developed for air.

The development of AALs for soil contact will address exposure by the oral and dermal route. These routes of exposure will be evaluated separately and then integrated into a soil exposure scenario. The development of AALs for exposure to toxic substances in a soil medium will be based on this exposure scenario.

Oral Route of Exposure

Numerous studies that demonstrate age-related changes in pica or mouthing behavior and PbB concentrations support the conviction that soil exposure is a function of age. Therefore, soil exposure scenarios generally incorporate age-related changes in soil exposure (*2,5–7*). The following exposure scenario will incorporate an age-dependent variation for the oral route of exposure.

Ingestion of Soil by Children

A recent study by Binder and co-workers conducted in East Helena, MT, provides a reasonable approach for determining the average amount of soil ingested by a child (*37*). These investigators employed a mass-balance approach, based on the ingestion and excretion of three elements, aluminum (Al), silicon (Si), and titanium (Ti), to ascertain the quantity of soil ingested. Briefly stated, the amount of an element that is excreted into the feces should be approximately equivalent to the amount of the element ingested less the amount of the element absorbed from the gastrointestional tract. If an element is not appreciably absorbed, the amount ingested should be equivalent to the amount excreted. If it is assumed that all ingested Si, Al, or Ti is derived from ingested soil the average amount of soil ingested is derived by dividing the amount of the element ingested by its concentration in the soil.

Since there is evidence that some of these elements occur in the diet and that some of them are absorbed to some degree from the gastrointestional tract, the following analysis will attempt to take these factors into consideration.

Assuming the soil is the sole source of the element not obtained from the diet or drinking water provides the maximum estimate of soil ingestion. For the purpose of quantifying soil ingestion in children, it is assumed that the sole source of element not accounted for by the ingestion of food or drinking water is ingested soil. The following is an outline of a mass-balance approach to determine the average amount of daily soil ingestion by a young child:

$$\text{Soil ingestion (mg/day)} = (Fc \times F \div EF - DI) \div Sc$$

Where

FC = The concentration of the element in the feces (mg/g feces)

F = Daily feces output = 15 g/day (from 36, 37, and 38)

EF = Fraction of daily intake excreted into the feces

DI = Daily intake of the element from the diet and drinking water

Sc = Concentration of the element in the soil.

The mass-balance approach will be employed to analyze the results of the study of Binder and co-workers (*37*). Soil samples collected adjacent to the child's residence and feces from 59 children, 1 to 3 years of age, were analyzed for their Al, Si, and Ti content.

In addition to the efforts of Binder and co-workers, a study conducted in Detroit, MI (*41*), provides data that can also be evaluated by this mass-balance approach. Samples of feces were collected from 10 children (ages 1–3 years) "who lived in good housing." Soil samples adjacent to their residences were also obtained. These samples were analyzed for their ^{207}Pb and ^{210}Pb content.

Quantification Based on Pb (*41*)

Fecal Excretion of Ingested Pb. Two balance studies of Pb metabolism in infants have provided similar estimates of fecal excretion. In one study, the intake and excretion of Pb was determined in infants whose ages ranged from 14 to 746 days (*31*). Fecal excretion averaged 57% of the Pb intake of infants ingesting moderate quantities of Pb.

In another study, Pb intake and excretion were determined in eight children whose ages ranged from 3 months to 8.5 years (*30*). This investigator reported that on average, 48% of the Pb ingested by infants was excreted into the feces.

For the purpose of quantifying soil ingestion in young children, the arithmetic mean, 0.47, of these two determinations of the fecal excretion of lead will be employed.

Pb Intake from Food and Drinking Water. Investigators from the U.S. Food and Drug Administration (FDA) analyze samples of food from various geographic regions of the country on a yearly basis. These studies are designed to monitor the level of selected pesticides and other chemicals in the food supply (*42–44*).

The FDA also has established lists of various food commodities and the average amount of each commodity consumed by a typical American (*42,45,46*). These accounts of the average composition of the American diet have been developed for specific age groups. Estimates of the average intake of these food commodities are available for 2-year-old children. Employing the analyses of various food groups on these lists, the average daily intake of specific substances from the ingestion of drinking water and food commodities for a 2-year-old toddler has been estimated. The FDA determined that the average daily Pb intake from food and drinking water by a 2-year-old child from data collected in 1975 was 26 μg/day (*44*). No information that would allow the quantification of the dietary intake of Pb in toddlers could be located for the year 1974.

Quantification of soil ingestion using mass-balance approach of Pb:

FC = 4.1 μg/g feces
F = 15 g/day
EF = 0.53
DI = 26 μg/day
SC = 460 μg lead/g soil 2 ft from a brick house
SC = 178 μg lead/g soil 10 ft from a brick house
Daily soil ingestion = 0.20 g/day (based on 460 μg lead/g soil)
= 0.51 g/day (based on 178 μg lead/g soil)

For the purpose of quantifying the daily soil ingestion in a child, the mean of these two estimates, 0.35 g/day, will be employed.

Quantification Based on ^{210}Pb (*41*)

Fecal Excretion of Ingested ^{210}Pb. It will be assumed that the fecal excretion of ^{210}Pb and ^{207}Pb is equivalent. Information describing the amount of ^{207}Pb excreted will be employed in the following analysis.

^{210}Pb Intake from Food and Drinking Water. The average daily intake of ^{210}Pb from the ingestion of drinking water and food is derived by employing data from two sources: A study that quantified the ^{210}Pb content of various food commodities (*47*) and the FDA lists of food commodities that are employed in the annual total diet study. Two estimates of the daily dietary intake of ^{210}Pb will be derived by using two accounts of the average composition of a toddler's total diet. The old list (*42,46*) in addition to the new list (*45*) will be employed in the evaluation. Comparing the estimates of Pb intake based on the two lists yields information on the sensitivity of the analysis to changes in the dietary composition.

Various food commodities are assigned to food groups that coincided with the ^{210}Pb analysis (Tables 1

Table 1. Total diet commodities distributed into ^{210}Pb food groups.[a]

Food	Item no.	Quantity, g/day
Fresh fruit, 0.4 pCi/kg	078	16.715
	079	7.341
	080	10.858
	081	2.469
	083	2.964
	085	2.431
	086	1.209
	088	1.616
	089	0.701
	091	0.564
	092	0.843
	094	0.234
	095	1.158
	096	0.094
	097	0.112
	225	0.240
	226	0.147
	227	0.172
	228	0.257
	229	0.356
	233	0.402
	234	0.060
Total		50.944
Rice, 0.88 pCi/kg	050	12.931
Total		12.931
Dried beans, 0.76 pCi/kg	038	3.850
	040	1.094
	041	0.467
	043	0.584
	044	0.816
Total		6.811
Macaroni, 0.92 pCi/kg	142	16.410
	146	7.481
	149	4.689
	150	1.213
	151	1.137
	155	17.808
	069	1.409
	070	3.258
Total		53.405
Canned fruit, 2.0 pCi/kg	082	4.572
	084	5.687
	087	1.960
	090	0.913
	093	0.724
Total		13.856
Root vegetables, 0.21 pCi/kg	127	2.400
	128	0.221
	133	0.097
	047	2.911

(*Continued on next page*)

Table 1. (*Continued*)

Food	Item no.	Quantity, g/day
	048	0.237
Total		5.866
Fresh fish, 0.39 pCi/kg	031	2.261
	032	1.471
	034	1.156
Total		4.888
Shell fish, 3.4 pCi/kg	033	1.240
Total		1.240
Poultry, 0.45 pCi/kg	024	5.882
	025	4.802
	026	1.070
	154	0.5 × 1.692
	208	0.5 × 0.225
	212	0.5 × 0.054
Total		12.739
Eggs, 0.26 pCi/kg	035	13.088
	026	6.129
	037	2.891
Total		22.108
Potatoes, 1.5 pCi/kg	134	13.565
	135	9.879
	136	6.562
	137	3.178
	138	2.057
	139	2.049
	140	0.453
	141	0.355
Total		38.098
Bakery products, 1.8 pCi/kg	058	26.837
	059	4.104
	060	3.259
	061	2.582
	062	2.706
	063	1.288
	064	0.372
	065	0.687
	066	3.354
	067	1.376
	068	5.833
	147	0.5 × 4.574
	178	2.326
	179	3.170
	180	0.291
	181	2.365
	182	0.547
	183	7.635
	184	3.121
	185	1.693
	186	1.002
Total		76.835
Whole grain, 2.2 pCi/kg	051	12.159
	052	3.514
	053	5.117
	071	2.648
	072	2.739
	073	1.642
	074	1.158
	075	1.749
	076	0.285
	077	2.001
	144	0.5 × 3.198
	217	0.316
Total		34.927
Milk, 0.29 pCi/kg	001	249.285
	002	68.725
	003	10.673
	004	19.483
	005	0.488
	006	1.095
	007	1.286
	008	1.874
	009	1.749
	010	4.563
	011	1.470
	012	1.815
	144	0.5 × 3.198
	164	1.214
	167	0.541
	168	0.264
	174	9.579
	175	3.487
	176	1.164
	177	1.724
	202	0.682
	203	1.900
	204	0.147
	232	0.015
Total		385.722
Meat, 0.49 pCi/kg	013	10.973
	014	4.754
	015	0.549
	016	4.073
	017	2.785
	018	3.987
	019	2.232
	020	1.675
	021	1.599
	022	0.318
	023	0.212
	027	0.729
	028	8.583
	029	5.243
	030	0.442
	143	0.5 × 5.798
	145	0.5 × 2.226
	147	0.5 × 4.574
	148	3.983
	205	0.089
	206	0.030
	209	0.5 × 0.207
	211	0.5 × 0.147
	213	0.5 × 0.114
Total		64.835
Soda or water, 0.04 pCi/kg	191	43.668
	192	19.409
	193	78.505
	194	6.818
	195	2.162
	196	0.340
	197	31.637
	198	0.057
	201	321.000
Total		503.596
Juices, 0.23 pCi/kg	098	59.370
	099	17.594
	100	1.119
	101	8.784
	102	2.308
	103	0.296
	104	24.954
	105	4.641
	118	1.005
	230	0.940

(*Continued on next page*)

Table 1. (*Continued*)

Food	Item no.	Quantity, g/day
	231	0.604
Total		121.615
Fresh vegetables, 1.1 pCi/kg	057	0.697
	042	0.595
	046	0.666
	054	4.013
	107	0.586
	108	0.506
	109	2.619
	110	0.667
	111	0.347
	113	1.254
	114	0.173
	115	0.171
	116	0.387
	117	3.944
	121	0.905
	123	0.761
	124	0.403
	125	0.065
	126	0.226
	132	0.037
	143	0.5 × 5.798
	152	1.761
	153	0.809
	154	0.5 × 1.692
	161	0.578
	173	1.311
	208	0.5 × 0.225
	209	0.5 × 0.207
	211	0.5 × 0.147
	212	0.5 × 0.054
	213	0.5 × 0.114
	219	0.202
	221	0.128
	223	0.024
	220	0.060
Total		28.014
Canned vegetables, 0.44 pCi/kg	039	4.593
	045	3.192
	055	3.295
	056	1.697
	106	0.348
	112	0.064
	119	1.777
	120	0.152
	122	3.234
	129	1.182
	130	0.124
	131	0.337
	156	5.757
	157	16.196
	145	0.5 × 2.226
Total		43.061
Other	049	0.247
	158	1.049
	159	3.513
	160	0.467
	162	2.690
	163	0.938
	165	0.545
	166	0.498
	169	3.198
	170	2.454
	171	2.563
	172	0.347
	187	2.651
	188	1.879
	189	0.573
	190	5.016
Total		28.628

Table 1. (*Continued*)

[a]Various food groups from the revised total diet study food list (*45*) were allocated in accordance with the ^{210}Pb food groups (*48*). In certain instances a portion of the food commodity was allocated to two ^{210}Pb food groups. Commodities for which an assignment to a ^{210}Pb group was unclear were grouped in "Other."

Table 2. Total diet commodities grouped into ^{210}Pb food families.[a]

Item	Quantity, g/day	pCi/kg
Drinking water	341	0.04
Whole milk	511	0.29
Other dairy products	69	0.26
Meat, fish, and poultry	125	0.45
Grain and cereal products	118	2.0
Potatoes	36	1.5
Fresh vegetables	29[b]	1.1
Canned vegetables	43[b]	0.44
Fresh fruit	42[c]	0.4
Canned fruit	11[c]	2.0
Fruit juice	94[c]	0.23
Oils and fats	15	
Sugar	30	
Beverages	94	0.04

[a]Total diet constitutent from Duggan and McFarland (*46*) and Gartrell et al. (*42*).

[b]Distribution between fresh and canned vegetables in accordance to Pennington (*45*).

[c]Distribution of fresh fruit, canned fruit, and fruit juice in accordance with Pennington (*45*).

Table 3. ^{210}Pb daily intake.[a]

Item	pCi/day
Drinking water	0.014
Whole milk	0.148
Other dairy products	0.018
Meat, fish, and poultry	0.056
Grain and cereal products	0.236
Potatoes	0.054
Vegetables[b]	0.062
Fruit	0.060
Oils and fats	
Sugar	
Beverages	0.04
Total	0.69[d]

[a]Total diet constitutent from Duggan and McFarland (*46*) and Gartrell et al. (*43*).

[b]A distribution of 60% canned and 40% fresh, in accordance with Pennington (*45*).

[c]A distribution of 29% fresh, 7.4% canned, and 64% juice, in accordance with Pennington (*45*).

[d]Total adjusted to account for the daily intake of oils and fats and sugars. The adjustment, increasing the total by 2.8%, was based on the daily intake of oils, fats, and sugar/total daily intake.

and 2). These data were used to quantify the average ^{210}Pb intake from food and drinking water consumption by a 2-year-old toddler (Tables 3 and 4).

The daily intake of ^{210}Pb determined by employing the pre-1983 or the revised 1983 list of the composition of a toddler's diet were virtually identical. A mean of

Table 4. ^{210}Pb daily intake.[a]

Item	pCi/kg
Fresh fish	0.002
Shell fish	0.004
Poultry	0.006
Meat	0.032
Eggs	0.006
Fresh fruit	0.020
Canned fruit	0.028
Fresh vegetables	0.031
Canned vegetables	0.019
Root vegetables	0.001
Potatoes	0.057
Macaroni	0.049
Juice	0.028
Dried beans	0.005
Bakery goods	0.138
Whole grain	0.077
Milk	0.119
Soda and water	0.020
Rice	0.011
Total[b]	0.66

[a]Total diet from Pennington (*45*).
[b]Total adjusted to account for items classified in "other" group. The adjustment, an increase of 1.9%, was based on the daily intake of items in other group/total daily intake.

the two values, 0.68, will be employed for the purpose of quantifying soil ingestion by a child.

Quantification of soil ingestion using mass balance of ^{210}Pb:

FC = 0.044 pCi/g
F = 15 g/day
EF = 0.53
DI = 0.68 pCi/day
SC = 1.15 pCi/g soil
Daily soil ingestion = 0.49 g soil/day

Quantification Based on Si (*37*)

Fecal Excretion of Ingested Si. Information concerning the fecal excretion of Si from the gastrointestinal tract is very limited. The fraction of ingested Si absorbed in general is considered to be small (*48*).

In one study, a single oral dose at an aluminosilicate compound containing 1460 mg Si was ingested by four subjects (*49*). An additional 17 mg of Si compared to control, were detected in the urine of the four subjects. These data indicate that approximately 1% of the dose was absorbed from the gastrointestinal tract.

In another study, magnesium trisilicate, 5 g/day, was administered orally to humans for 4 consecutive days [9.2 g silicon dioxide (SiO_2)] (*50*). Urine was collected during and for 5 days after the last dose of the Si compound. Approximately 5% (484 mg) of the oral dose was detected in the urine. Si does not appear to be appreciably absorbed from the gastrointestinal tract in other species (*51,52*).

For the purposes of quantifying the amount of soil ingested by young children, 95% of ingested Si will be assumed to be excreted into the feces.

Si Intake from Food and Drinking Water. The average daily intake of Si from drinking water and food was determined by employing information from two sources: A study that measured the silicon content of various food commodities and water (*53,54*) and the FDA list of the typical composition of a toddler's diet (*45*). Using these data, various food commodities in the toddler's diet were placed into food groups that coincided with the results of the Si analyses (Table 5).

Table 5. Total diet commodities distributed into silicon food groups.

Food	Item no.	Quantity, g/day
Vegetables	057	0.697
	038	3.850
	040	1.094
	041	0.467
	043	0.584
	044	0.816
	145	0.5 × 2.226
	039	4.593
	045	3.192
	055	3.295
	056	1.697
	106	0.348
	112	0.064
	119	1.777
	120	0.132
	122	3.234
	129	1.182
	130	0.124
	131	0.337
	156	5.757
	157	16.196
	042	0.595
	046	0.666
	054	4.013
	107	0.586
	108	0.506
	109	2.619
	110	0.667
	111	0.347
	113	1.254
	114	0.173
	115	0.171
	116	0.387
	117	3.944
	121	0.905
	123	0.761
	124	0.403
	125	0.065
	126	0.226
	132	0.037
	143	0.5 × 5.798
	152	1.761
	153	0.809
	220	0.060
	154	0.5 × 1.692
	161	0.578
	173	1.311
	208	0.5 × 0.225
	209	0.5 × 0.207
	211	0.5 × 0.147
	212	0.5 × 0.054
	213	0.5 × 0.114
	219	0.202
	221	0.128
	223	0.024
Total		77.886
Root vegetables	127	2.400
	128	0.221

(*Continued on next page*)

Table 5. (*Continued*)

Food	Item no.	Quantity, g/day
	133	0.097
	134	13.565
	135	9.879
	136	6.562
	137	3.178
	138	2.057
	139	2.049
	140	0.453
	141	0.355
	147	2.911
	148	0.239
Total		43.996
Fats	049	0.247
	158	1.049
	159	3.513
	160	0.467
	162	2.690
	163	0.938
	165	0.345
	166	0.498
	169	3.198
	170	2.454
	171	2.563
	172	0.347
	187	2.051
	188	1.879
	189	0.573
	190	5.016
Total		27.83
Fish	034	1.156
	031	2.261
	032	1.471
	033	1.240
Total		6.128
Water and soft drinks	191	43.668
	192	19.409
	193	78.505
	194	6.818
	195	2.162
	196	0.340
	197	31.637
	198	0.057
	201	321.000
Total		503.596
Fruits	078	16.715
	079	7.341
	080	10.858
	081	2.469
	083	2.964
	085	2.431
	086	1.209
	088	1.616
	089	0.701
	091	0.564
	092	0.843
	094	0.235
	095	1.158
	096	0.094
	097	0.112
	225	0.240
	226	0.147
	227	0.172
	228	0.257
	229	0.356
	233	0.402
	234	0.060
	082	4.572
	084	5.687

Table 5. (*Continued*)

Food	Item no.	Quantity, g/day
	087	1.960
	090	0.913
	093	0.724
	098	59.370
	099	17.599
	100	1.119
	101	8.784
	102	2.308
	103	0.296
	104	24.954
	105	4.641
	118	1.005
	230	9.940
	231	0.604
Total		186.415
Meat	035	13.088
	026	6.129
	037	2.891
	013	10.973
	014	4.754
	015	0.549
	016	4.073
	017	2.785
	018	3.987
	019	2.232
	020	1.675
	021	1.599
	022	0.318
	023	0.212
	027	0.729
	028	8.583
	029	5.243
	030	0.442
	143	0.5 × 5.798
	145	0.5 × 2.226
	147	0.5 × 4.574
	148	3.983
	205	0.089
	206	0.030
	209	0.5 × 0.207
	211	0.5 ×0.147
	213	0.5 ×0.114
	024	5.882
	025	4.802
	026	1.070
	154	0.5 × 1.692
	208	0.5 × 0.225
	212	0.5 × 0.054
Total		93.686
Milk	231	0.015
	001	249.285
	002	68.725
	003	10.673
	004	19.483
	005	0.488
	006	1.095
	007	1.286
	008	1.874
	009	1.749
	010	5.463
	011	1.470
	012	1.815
	144	0.5 × 3.198
	164	1.214
	167	0.541
	168	0.264
	174	9.579
	175	3.487

(*continued*)

Table 5. (*Continued*)

Food	Item no.	Quantity, g/day
	176	1.164
	177	1.724
	202	0.682
	203	1.900
	204	0.147
Total		385.722
Cereal	058	26.837
	059	4.104
	060	3.259
	061	2.582
	062	2.706
	063	1.288
	064	0.372
	065	0.687
	066	3.354
	067	1.376
	068	5.833
	147	0.5 × 4.574
	178	2.326
	179	3.170
	180	0.291
	181	2.365
	182	0.547
	183	7.635
	184	3.121
	185	1.693
	186	1.002
	142	16.410
	146	7.481
	149	4.689
	150	1.218
	151	1.137
	155	17.808
	069	1.409
	070	3.258
	051	12.159
	052	3.514
	053	5.117
	071	2.648
	072	2.739
	073	1.642
	074	1.158
	075	1.749
	076	0.285
	077	2.001
	144	0.5 × 3.198
	217	0.316
	050	12.931
Total		165.167

The average quantity of silicon ingested from each food group was then derived (Table 6).

The average amount of silicon ingested daily in food and drinking water for a 2-year-old child is 18.0 mg/day. This value is similar to the intake reported by Bowen and Peggs (*53*) or Carlisle (*48*).

Quantification of soil ingestion using mass balance of Si:

FC = 2.56 mg/g feces
F = 15 g/day
EC = 0.95
DI = 18 mg/day
SC = 301.9 mg/g soil
Daily soil ingestion = 0.07 g soil/day

Table 6. Silicon daily intake.

Item	Food intake, g/day	Food content, mg Si/kg	Silicon intake, mg Si/day
Vegetable	77.886	8.8	0.7
Root vegetable	43.996	15.0	0.7
Fruit	186.415	1.0	0.2
Meat	93.686	3.0	0.3
Fish	6.228	1.3	0.0
Fat	27.83	1.6	0.1
Milk	385.722	3.4	1.3
Water and soft drinks	503.596	0.0025	1.3
Cereal	165.167	81.0	13.4
Total			18.0

Quantification Based on Al (*37*)

Fecal Excretion of Ingested Al. Very little Al appears to be absorbed from the gastrointestinal tract in healthy individuals (*55*). A balance study in six men indicated there was essentially no absorption of Al at levels that occur in the diet (*56*). When the diet was supplemented with an antacid containing large quantities of Al, net absorption of Al could be observed. Both plasma levels and urinary excretion of Al increased. Aluminum uptake did not exceed 25% of the oral dose of any individual receiving the antacid.

In another balance study in which eight healthy adults consumed a controlled diet or a diet supplemented with Al, almost all of the intake could be accounted for in the feces in individuals receiving controlled diet or those receiving an Al supplement (*57*).

Other studies that detected low levels of Al in the urine and plasma after the ingestion of substantial quantities of Al indicate that only a very small fraction of Al is absorbed from the gastrointestinal tract.

A recent study suggests that little Al is absorbed from the gastrointestinal tract of infants (*58*). The plasma and bone Al content of infants was comparable to that observed in adults and considerably below the levels observed in infants receiving prolonged intravenous feedings.

For the purposes of quantifying the amount of soil ingested by young children, all ingested Al will be assumed to be excreted into the feces.

Aluminum Intake from Food and Drinking Water. The average daily intake of Al from the ingestion of drinking water and food, 6.3 mg/day, was determined by investigators from the FDA (*59*).

Quantification of soil ingestion using mass balance of Al:

FC = 0.60 mg/g
F = 15 g/day
EF = 1.0
DI = 6.3 mg/day
SC = 66.03 mg/g soil
Daily soil ingestion = 0.04 g soil/day

Table 7. Gastrointestinal absorption of titanium.

Subject	Days on study	Mean titanium in diet, mg/day	Mean titanium in feces, mg/day	Fecal excretion, %
1	347	0.75	0.46	0.61
2	347	2.00	0.82	0.41
3	140	0.18	0.39	

Quantification Based on Ti (37)

Fecal Excretion of Ingested Ti. Information concerning the absorption of Ti is very limited. Very low concentrations of Ti are generally found in the urine. Based on an estimated intake of 300 μg/day, less than 5% of the oral uptake was accounted for in the urine (*60,61*).

The best information on the amount of Ti absorbed from the gastrointestinal tract comes from a balance study involving three males (*62*). Two subjects were enrolled in the study for 347 days; the third subject participated for 140 days. Appreciable absorption was observed in two of the subjects; a third subject appeared to be in a negative balance for titanium (Table 7).

For the purpose of quantifying the amount of soil ingested by children, a mean of the fecal excretion of subjects one and two will be employed (0.51).

Ti Intake from Food and Drinking Water. The average daily intake of Ti from the ingestion of food and drinking water was determined by employing data from two sources: A study that determined the titanium content of various food commodities (*63*) and the FDA list of the typical composition of a toddler's diet (*45*). Using these data, various food commodities in the average toddler's diet were assigned to food groups that corresponded with the results of the Ti analysis (Table 8). The average quantity of Ti ingested from each food group was then derived (Table 9). The average amount of Ti ingested

Table 8. Total diet constituents assigned to titanium food groups.

Food	Item no.	Quantity, g/day
Butter 2.49 μg/g	164	1.214
Total		1.214
Pork, 1.84 μg/g	17	2.785
	18	3.987
	19	2.232
	20	1.675
	21	1.599
	28	0.5 × 4.292
	206	0.03
	213	0.057
Total		14.511
Chicken, 0.16 μg/g	24	5.882
	25	4.802
	26	1.070
	207	.0
	208	0.5 × 0.225
	212	0.5 × 0.054
Total		11.894

Table 8. (*Continued*)

Food	Item no.	Quantity, g/day
Haddock, 0.78 μg/g	031	2.261
	032	1.471
Total		3.732
Rice, 0 μg/g	050	12.931
	075	1.749
Total		14.680
Cornflakes, 0.40 μg/g	071	2.648
Total		2.648
Oatmeal, 0.23 μg/g	051	12.159
	077	2.001
	217	0.5 × 0.316
Total		14.318
Beef and lamb, 0.01 μg/g	013	10.973
	014	4.754
	015	0.549
	016	4.073
	022	0.318
	023	0.212
	027	0.729
	028	0.5 × 4.292
	029	5.243
	030	0.442
	143	0.5 × 5.798
	145	0.5 × 2.226
	147	0.5 × 4.574
	148	3.983
	205	0.089
	211	0.5 × 0.142
	209	0.5 × 0.203
Total		39.988
Shrimp, 0.88 μg/g	033	0.124
Total		0.124
Halibut, 0.06 μg/g	034	1.156
Total		1.156
Corn meal, 0.06 μg/g	053	5.117
	060	3.259
	067	1.376
	063	1.288
Total		11.040
Rye, 0 μg/g	64	0.372
Total		0.372
Wheat, 0.41 μg/g	058	26.837
	059	4.104
	061	2.582
	062	2.706
	065	0.687
	066	3.354
	068	5.833
	069	1.409
	070	3.258
	072	2.739
	073	1.642
	076	0.285
	142	16.410
	146	7.481
	149	4.689
	150	1.213
	151	1.137
	155	17.808
	074	1.158
	178	2.326
	179	3.170
	180	0.291
	181	2.365
	182	0.547

(*continued*)

Table 8. (*Continued*)

Food	Item no.	Quantity, g/day
	183	7.635
	184	3.121
	185	1.693
	186	1.002
Total		127.662
Root vegetables, range: 0.02–0.31 μg/g median: 0.165 μg/g	134	13.565
	135	9.879
	136	6.562
	137	3.178
	138	2.057
	139	2.049
	140	0.453
	141	0.355
	127	2.400
	128	0.221
	133	0.097
	147	2.911
	148	0.239
Total		43.966
Fruit, range: 0.0-0.20 μg/g median: 0.10 μg/g	078	16.715
	079	7.341
	080	10.858
	081	2.469
	083	2.964
	085	2.431
	086	1.209
	088	1.616
	089	0.701
	091	0.564
	092	0.843
	094	0.235
	095	1.158
	096	0.094
	097	0.112
	225	0.240
	226	0.147
	227	0.172
	228	0.257
	229	0.356
	233	0.402
	234	0.060
	098	59.370
	099	17.594
	100	1.119
	101	8.784
	102	2.308
	103	0.296
	104	24.954
	105	4.641
	118	1.005
	082	4.572
	084	5.687
	087	1.960
	090	0.913
	093	0.724
	230	0.940
	231	0.604
Total		186.415
Vegetables, range: 0.0–2.42 μg/g; median: 1.21 μg/g	057	0.697
	042	0.595
	046	0.666
	054	4.013
	107	0.586
	108	0.506
	109	2.619
	110	0.667
	111	0.347
	113	1.254
	114	0.173
	115	0.171
	116	0.387
	117	3.944
	121	0.905
	123	0.761
	124	0.403
	125	0.065
	126	0.226
	132	0.037
	143	0.5 × 5.798
	152	1.761
	153	0.809
	154	0.5 × 1.692
	161	0.578
	173	1.311
	208	0.5 × 0.225
	209	0.5 × 0.207
	211	0.5 × 0.147
	212	0.5 × 0.054
	213	0.5 × 0.114
	219	0.202
	221	0.128
	223	0.024
	220	0.060
	039	4.593
	045	3.192
	055	3.295
	056	1.697
	106	0.348
	112	0.064
	119	1.777
	120	0.152
	122	3.234
	129	1.182
	130	0.124
	131	0.337
	156	5.757
	157	16.196
	038	3.850
	040	1.094
	041	0.467
	043	0.584
	044	0.816
	145	0.5 × 2.226
Total		77.886
Water and soft drinks, 0.5 μg/g	191	43.668
	192	19.409
	193	78.505
	194	6.818
	195	2.162
	196	0.340
	197	31.637
	198	0.57
	201	321.000
Total		503.596
Oil, 0.83 μg/g	163	0.938
	165	0.545
	166	0.498
Total		1.981
Corn oil/margarine, 1.8 μg/g	162	2.690
Total		2.690

Table 9. Titanium daily intake.

Food group	Amount ingested, g/day	Titanium content, μg/g	Titanium intake, μg/day
Dairy	–	0.0	0.0
Eggs	–	0.0	0.0
Butter	1.214	2.49	3.02
Beef and lamb	39.988	0.01	0.40
Pork	14.511	1.84	26.70
Chicken	11.894	0.16	1.90
Shrimp	0.124	0.88	0.11
Haddock	3.732	0.78	2.91
Halibut	1.156	0.06	0.07
Rice	–	0.0	0.0
Corn meal	11.040	0.06	0.66
Corn flakes	2.648	0.40	1.06
Wheat flour	127.662	0.41	52.34
Rye	–	0.0	0.0
Oatmeal	14.318	0.23	3.29
Fruit	186.415	0.10	18.64
Root vegetable	43.966	0.165	7.25
Vegetable	77.866	1.21	94.22
Water and soft drinks	503.596	0.50	251.8
Oil	1.981	0.83	1.64
Corn oil/margarine	2.690	1.80	4.84
Total			471.02

in food and drinking water for a 2-year-old child is 0.14 mg/day.

Quantification of soil ingestion using mass balance for Ti:

FC = 0.08 mg/g feces
F = 15 g/day
EF = 0.51
DI = 0.47 mg/day
SC = 2.99 mg/g soil
Daily soil ingestion = 0.64 g soil/day

Discussion

Five estimates of soil ingestion in children were ascertained by employing data from a variety of sources. The estimates, each of which was based on a different set of data, ranged from 0.04 g/day to 0.64 g/day. The arithmetic mean of the estimates is 0.33 g/day with a standard deviation of 0.26 g/day.

Considerable uncertainty surrounds the approach employed to derive the estimates of the amount of daily soil ingested. For instance, the daily dietary intake of each of the elements is based on a typical composition of an average toddlers' diet and on analyses of food at locations remote from the location of the two soil ingestion studies. It would have been desirable to have monitored the concentration of these elements in the food consumed by the children enrolled in these studies.

Given the many uncertainties associated with the approach employed to derive the estimates of soil ingestion, it is remarkable that the range of the estimates is so narrow. Since each estimate was arrived at by employing a separate set of data, the similarity of the estimates invites confidence that they reasonably reflect the amount of soil ingestion for this age group.

For the purpose of developing AAL for exposure to toxic substance via soil contact, the mean plus one standard deviation of the five estimates of soil ingestion will be employed as the average quantity of soil ingested by a child each day. This value provides a margin of safety in the development of AALs for soil contact.

Ingestion by Adults

No information could be identified that provides a sound technical basis for estimating the quantity of soil ingested by older children and adults. While other investigators have assumed that adults ingest a significant quantity of soil and employ an assumed rate of soil ingestion for adults, no basis for these assumptions could be identified (*2,4–6*). Investigators who have advanced exposure scenarios that include soil ingestion for adults assume that adults ingest less soil than children.

Exposure Scenario for Soil Ingestion

For the purpose of developing AALs for an exposure to toxic substance in soil via the route of ingestion, an estimate of the daily exposure to soil via ingestion for older children and adults will be based on age-related changes in PbB and prevalence of mouthing behavior.

Pb intoxication is observed in children but rarely in adults. Children appear to be more at risk in part because they are exposed to more Pb than adults. Soil contaminated with Pb appears to be a significant source of exposure for children. Studies aimed at identifying various sources of Pb exposure in children have correlated PbB levels in children with both the levels of Pb in soil and the level of Pb detected on the hand.

Mouthing behavior is highly prevalent in children. The mouthing of dirty hands would result in the ingestion of adhering soil. Since soil Pb levels have been correlated with PbB levels in children, age-related changes in PbB would appear to provide an indication of age-related changes in exposure to soil. Inasmuch as mouthing behavior appears to play a role in elevated Pb exposure in children, changes in its prevalence would appear to provide an indication of age-related changes in soil exposure.

A variety of studies have investigated age-related changes in PbB concentration and the prevalence of mouthing behavior in children. The results of these investigations will be employed to estimate age-related changes in soil exposure.

PbB levels of children between the ages of 2 and 9 years were determined in the area of a smelter in Idaho (*36*). Mean PbB levels were reported by age and by the distance that the children lived from the smelter. For the purpose of this analysis, the data were segregated by age only (Table 10).

The rate of the decline of PbB with age was determined by employing a curve-fitting program, which employed at least square fit to an exponential function. The analysis yielded the following expression:

$$y = 108\ e^{-0.0319x}$$

Where: y = the percent of the mean PbB concentration observed at age two, and x = age (years).

PbB levels were determined in a large cross-section of the U.S. population. The results of the lead survey portion of the Second National Health and Nutrition Examination Survey included PbB levels in various age, ethnic, economic, and geographical populations (*64*). PbB levels for all races within various age ranges are displayed in Table 11.

The average PbB level declined until the year 15. Blood levels then began to increase with age. The increases in PbB levels after age 15 is probably not due to increased ingestion of lead in contaminated soil. Therefore, the analysis will employ PbB levels through year 14.

The rate of the decline of PbB with age was determined by employing a curve-fitting program. The analysis yielded the following expression:

$$y = 1.0594\ e^{-0.0305x}$$

Where: y = the percent of the mean PbB concentration observed at age 2, and x = age (years).

PbB levels were determined in large surveys conducted in New York and Chicago (*65*). Mean PbB levels in both these cities were reported separately for the white and black population. The result of these surveys are displayed in Table 12.

The rate of the decline of PbB with age was determined by employing a curve-fitting program. The analysis yielded the following expressions:

New York

White: $y = 103.6\ e^{-0.021x}$

Black: $y = 104.2\ e^{-0.021x}$

Chicago

White: $y = 104.1\ e^{-0.023x}$

Black: $y = 104.2\ e^{-0.023x}$

where: y = the percentage of the mean PbB level observed at age 2, and x = age (years).

Table 10. Blood lead levels in children residing in the vicinity of a smelter.[a]

Age, years	Blood lead, μg/dL	Fraction of 2-year level, %
2	41	100
3	40	98
4	39	95
5	37	92
6	37	92
7	36	89
8	35	86
9	31	77

[a]From Yankel et al. (*36*).

Table 11. Blood lead levels of all races.[a]

Age, year	Blood lead level, μg/dL	Fraction of 2-year level, %
0.5–3	16.3	100
3–5	15.9	97.6
6–8	13.9	85.3
9–11	12.9	79.1
12–14	11.4	69.0
15–17	12.1	
18–24	13.1	
25–34	13.7	
35–44	14.6	
45–54	15.3	
55–64	14.6	
65–74	14.4	

[a]From Annest and Mahaffey (*64*).

Table 12. Blood lead levels in children from New York and Chicago.[a]

Age, years	Blood lead levels, μg/dL	Fraction of 2-year level, %
New York		
Whites		
2	17.2	100.0
3	16.7	97.1
4	16.4	95.4
5	15.9	92.4
6	15.9	92.4
Blacks		
2	18.6	100.0
3	18.1	97.3
4	17.7	95.2
5	17.2	92.5
6	17.2	92.5
Chicago		
Whites		
2	17.8	100.0
3	17.2	96.6
4	16.8	94.4
5	16.6	93.3
Blacks		
2	19.9	100.0
3	19.3	97.0
4	18.8	94.5
5	18.6	93.5

[a]From Billic (*65*).

For the purpose of developing AALs for soil contact, an arithmetic mean of the four exponents (–0.022) from the four populations studied will be employed.

In a large study conducted in New Haven, CT, PbB levels were determined in children between the ages of 1 and 72 months (*66*). The mean PbB level at various ages is displayed in Table 13.

The rate of the decline of PbB with age was determined by employing a curve-fitting program. The analysis yielded the following expressions:

$$y = 106.4\ e^{-0.0299x}$$

where: y = the percentage of the mean PbB level observed at 2 years of age, and x = age (years).

The prevalence of mouthing behavior at various

Table 13. Blood lead levels in New Haven children.[a]

Age, years	Blood lead levels, μg/dL	Fraction of 2-year level, %
2	28.7	100.00
3	27.8	96.9
4	27.4	95.5
5	26.1	90.9

[a]From Quah et al. (*66*).

Table 14. Prevalence of mouthing behavior in white children.[a]

Age, years	Prevalence, %	Fraction of 2-year level, %
1	83	163
2	51	100
3	25	49
4	19	37
5	16	31

[a]From Millican et al. (*35*).

Table 15. Prevalence of mouthing behavior in black children.[a]

Age, years	Prevalence, %	Fraction of 2-year level, %
1	77	128
2	60	100
3	39	65
4	45	75
5	38	65

[a]From Millican et al. (*35*).

ages was investigated in children from the Washington, DC area (*35*). Mothers of children were interviewed and the results of the survey reported for both white (Table 14) and black children (Table 15).

The rate of decline of the prevalence of mouthing behavior was determined by employing a curve-fitting program. The analysis yielded the following expressions:

$$\text{White children: } y = 226\ e^{-0.43x}$$
$$\text{Black children: } y = 137\ e^{-0.164x}$$

where: y = the percent of the prevalence of mouthing observed at 2 years of age, and x = age (years).

The prevalence of mouthing behavior was studied in children in the city of Boston (*34*). Either questionnaires were mailed to a family or interviews were conducted to obtain pertinent information. The prevalence of mouthing behavior in white children during a 14-day period in families contacted by mail is displayed in Table 16. The prevalence of mouthing behavior during a 14-day period for white and black children from families that were interviewed is displayed in Table 17.

The rate of decline of mouthing behavior was determined by employing a curve-fitting program. The analysis yielded the following expressions:

white and black children:

$$y = 124\ e^{-0.134x}$$

white children:

$$y = 111\ e^{-0.051x}$$

Table 16. Prevalence of mouthing behavior in white children.[a]

Age, years	Prevalence, %	Fraction of 2-year level, %
1	100	105
2	95	100
3	91	95
4	87	92
5	79	83
6	79	83

[a]Contacted by mail. From Barltrop (*34*).

Table 17. Prevalence of mouthing behavior in white and black children.[a]

Age, years	Prevalence, %	Fraction of 2-year level, %
1	82	114
2	72	100
3	56	78
4	42	58
5	51	71
6	42	58

[a]Parental interview. From Barltrop (*34*).

where: y = the percent of the observed prevalence of mouthing observed at 2 years of age, and x = age (years).

Soil Ingestion at Various Ages

For the purpose of developing AALs for soil contact, estimates of soil ingestion will be derived from the amount of soil ingested by a toddler (0.59 g/day) and estimates of soil ingestion at subsequent ages. These estimates will be derived by employing age-related changes in PbB levels and mouthing behavior as indicators of the change in soil ingestion with age.

Age-related changes in PbB levels and mouthing behavior are weighted equally in deriving an estimate of soil ingestion at various ages. The arithmetic mean of the four exponents of the equations describing the rate of decline mouthing behavior with age is –0.195. The arithmetic mean of the four exponents of the equations describing the rate of decline of PbB with age is –0.029. The mean of exponents from the PbB studies and the studies that evaluated mouthing behavior in children are weighted equally in deriving an expression for the decrease in soil ingestion with age.

Soil ingestion over a lifetime is derived by using the level of daily soil ingestion for toddlers derived from the studies of Binder and co-workers (*31*) and Ter Haar and Aronow (*41*) and the rate of decline of soil ingestion based on a mean of the exponents of equations that describe changes with age of PbB levels and the prevalence of mouthing behavior. For children from 1 to 3 years old, daily soil ingestion is 0.59 g/day. Soil in-

gestion between the ages of 3 and 19 years is derived as follows:

$$\text{Soil ingestion (g/day)} = 0.74 \text{ g/day } e^{-0.112x}$$

where x = age of child (years).

Age-related changes in soil ingestion would not be expected in adults (age 18–70 years). Therefore, the exposure scenario does not provide for changes in the amount of soil ingested after year 18.

For adults, daily soil ingestion (g/day) = $0.74\ e^{-0.112} \times 18$. Table 18 displays daily soil ingestion at successive ages. The average daily soil ingestion over a lifetime = 0.15 g/day.

Dermal Route of Exposure

Information concerning exposure to toxic chemicals in soil from contact with the skin is very limited. A variety of factors probably influence the level of soil exposure. The surface area of skin exposed to soil, the amount of soil that adheres to the exposed skin, the type of soil particles that adhere to skin, and the distribution of these particles in soil would be expected to influence the level of dermal exposure to toxic substances in soil. No information could be identified that provided a firm technical basis for addressing all these factors for the purpose of developing a soil exposure scenario.

Various investigators have proposed scenarios for evaluating exposure to toxic substances in soil via dermal contact (*2,6,7*). Elements of the approaches advanced by these investigators will be employed in developing a reasonable and conservative estimate of exposure to soil by the dermal route.

Table 18. Average amount of soil ingested.

Age range, years	Soil ingestion, g/day	Total exposure, g[a]
1-2	0.59	215.4
2-3	0.59	215.4
3-4	0.53	192.5
4-5	0.47	172.1
5-6	0.42	153.9
6-7	0.38	137.6
7-8	0.34	123.0
8-9	0.30	110.0
9-10	0.27	98.3
10-11	0.24	87.9
11-12	0.22	78.6
12-13	0.19	70.3
13-14	0.17	62.8
14-15	0.15	56.2
15-16	0.14	50.2
16-17	0.12	44.9
17-18	0.11	40.1
18-70	0.10	1865.9
Total		3775.1

[a]Total exposure = soil ingestion × number of days in the indicated age range.

Surface Area of Skin. The average surface area of the human body and various extremities has been determined at various ages (*67*). The surface area of the adult is greater than that of children and adolescents. Therefore, given that an equivalent load of soil occurs on an area of skin and that soil adheres to the same extremities, the total exposure to soil would be greater in the adult than in a child. For the purpose of developing AALs for exposure to toxic chemicals in soil by the dermal route, age-related increases in surface area will be incorporated into the exposure scenario.

Soil Adherence to Skin. Lepow and co-workers employed adhesive tape to sample 2.15 cm^2 of skin on an area of the hand to determine the amount of soil present (*68*). This method yielded approximately 11 mg of material on the skin surface. Assuming all the material recovered was soil and that the methodology yielded a substantial portion of the soil adhering to the skin, approximately 0.5 mg of soil was determined to be adhering to 1.0 cm of skin.

In another study, the amount of lead adhering to the hands of children (average age of 11 years) was determined while they played on school yards (*27*). The amount of Pb adhering to the hand was determined by pouring 500 mL of dilute nitric acid over the palm. The lead content of these hand rinse and of soil samples from the school yard was determined. Assuming the concentration of Pb in soil recovered from the hand and the school yard was equivalent, an estimate of the amount of soil on the hand can be ascertained (Table 19).

The average total surface area of an 11-year-old child is 12.300 cm^2 (*65*). The surface area of the hand on average is 2.5% of the total body surface area (69 and 70). Therefore, the average surface area of the hand of an 11 year old child is 307 cm^2. Assuming approximately 60% of the hand was sampled by the methodology employed by Roels et al. (*27*), 0.159 g of soil adhered to 185 cm^2 of skin (or 0.9 mg/cm^2).

Another group of investigators fractionated soil in ranges of particle sizes (*71*). For each range of particle sizes, the amount of particles that adhered to the palm of the hand of a small adult was determined.

Based on the following assumptions, a conservative estimate of the amount of soil adhering to the skin can be ascertained. Soil is composed of particles of the indicated diameters. All particle sizes continue to

Table 19. Soil adhering to the hand.[a]

Play yard location	Soil lead concentration, μg/g	Lead on the hand, μg	Soil on the hand, g[b]
Urban	112	20.4	0.182
Rural	114	17.0	0.149
2.5 km from a smelter	466	62.6	0.133
1.0 km from a smelter	2560	436.0	0.170
Arithmetic mean			0.159

[a]Adopted from Roels (*27*).

[b]Soil on the hand (g) = Lead on the hand (μg)/soil lead concentration (μg/g).

adhere to the skin to the degree observed in this study. An equivalent weight of particles of any diameter adhere to the same surface area of skin.

When these assumptions are used, on average, 31.2 mg of soil adheres to the palm of a small adult. When the surface area of the palm of a small adult is assumed to be approximately 160 cm^2 (16,000 $cm^2 \times 0.025 \times 0.40$) (*67,69,70*), 0.20 mg of soil adheres to 1 cm^2 of skin.

Skin Soil Load Estimate. For the purpose of developing AALs for exposure to toxic chemical in soil, an arithmetic mean of the three estimates, 0.5 mg/cm^2, of soil adhering to skin will be employed. A soil load of 0.5 mg/cm^2 on the hand should be considered to be near the maximum load of soil that could occur on the skin given the type of procedures employed to determine this measurement. Normally, it is very unlikely that most skin surfaces are covered with this amount of soil.

Exposed Surface Area. The surface area of skin exposed to soil will be influenced by the type of clothing worn by an individual. For the purpose of developing AALs for exposure to toxic chemicals in soil, it will be assumed that the head, neck, lower arms, hands, and feet are exposed to soil on a daily basis.

Relationship Between Age and Soil Exposure. In addition to the amount of surface area exposed, the magnitude of soil exposure would be expected to be influenced by behavior tendencies. Children who play outside would be expected to be exposed to more soil than sedentary adults. Therefore, for the purpose of developing AALs for soil contact, the exposure scenario to soil by dermal contact will address age-related behavior tendencies. Behavioral tendencies that influence the amount of soil ingested would also appear to influence dermal exposure to soil. Therefore, age-related changes in dermal exposure will mirror the age-related changes in soil ingestion that were described earlier. Soil load at various ages is derived according to the following algorithm:

Age 1 to 3 years:
Soil load = 0.5 g/cm^2

Age 3 to 18 years:
Soil load = $0.5\ g/cm^2 \times e^{-0.121 \times \text{years}}$

Age 19 to 70 years:
Soil load = $0.5\ g/cm^2 \times e^{-0.121 \times 18}$

Exposure Scenario for Dermal Route of Soil Exposure

For the purpose of developing AALs for exposure to toxic chemicals in soil it will be assumed that children and adults are exposed to soil on the head, neck, hands, feet, and lower arms. The relative distribution of the surface area of skin on various extremities at various ages is described by McDougal and co-workers (*69*) and Lund and Browder (*70*). The relative distribution changes slightly with age. The surface area of all the indicated extremities will be employed in the exposure scenario for dermal contact with soil.

The quantification of dermal exposure to soil is displayed in Tables 20 and 21. The exposed surface area is the area of skin assumed to be exposed to soil on a daily basis (Table 20). Exposed surface area is derived by multiplying total body surface area by the fraction of the body assumed to be exposed at the indicated ages.

Daily dermal exposure of the skin to soil (Table 21) is based on estimates of exposed surface area and soil load at the indicated ages. Daily dermal exposure is derived by multiplying exposed surface area by soil load at a given age. Total dermal exposure to soil at each indicated age range is determined by multiplying daily dermal exposure by the number of days in the

Table 20. Exposed surface area.

Age, years	Total surface area, cm^2 [a]	Exposed surface area, cm^2 [b]
2-3	6,030	2,050
3-4	6,640	2,258
4-5	7,310	2,485
5-6	7,930	2,379
6-7	8,660	2,598
7-8	9,360	2,808
8-9	10,000	3,000
9-10	10,700	3,210
10-11	11,800	3,304
11-12	12,300	3,444
12-13	13,400	3,752
13-14	14,700	4,116
14-15	16,100	4,186
15-16	17,000	4,420
16-17	17,600	4,224
17-18	18,000	4,320
18-70	19,400	4,656

[a]From EPA (*67*).
[b]Total surface area × fraction of total body surface area comprising the head, neck, hands, lower arms, and feet.

Table 21. Soil exposure via the dermal route.

Age, years	Daily dermal exposure, mg/day[a]	Total dermal exposure, g[b]
1-2	1,025	374
2-3	1,025	374
3-4	1,009	368
4-5	998	362
5-6	850	310
6-7	830	303
7-8	802	293
8-9	766	280
9-10	733	267
10-11	674	246
11-12	628	229
12-13	612	223
13-14	600	219
14-15	546	199
15-16	515	188
16-17	440	161
17-18	403	147
18-70	360	6,831
Total		11,376

[a]Exposure = exposed surface area (Table 19) × soil loading estimate for the indicated age range.
[b]Total exposure = daily dermal exposure × total number of days in the indicated age range.

indicated age range (Table 21). The average daily dermal soil exposure of 0.45 g/day was determined by cumulating total dermal exposure over a lifetime and then dividing the total exposure by the number of days in a lifetime.

AALs for Soil Contact

The California Site Mitigation Decision Tree Manual (*1*) outlines an approach for evaluation and mitigation of adverse health effects resulting from uncontrolled hazardous waste sites. A key element of the Decision Tree is the AAL. AALs are criteria that are employed to determine if the public health is or will be at risk from toxic substances emanating from an uncontrolled hazardous waste site. Because the Decision Tree employs a multimedia approach for evaluating impacts on the public health, AALs must be developed for various media of exposure.

Methods for developing AALs for air and water are outlined in the Decision Tree. The development of AALs for soil contact, when soil is a media of exposure, dictated the development exposure scenario for human contact to soil over a lifetime. Once an exposure scenario of humans to soil was formulated, AAL development for soil contact could proceed. The exposure scenario is based on the biological receptor exposed to soil in a residential setting for a 70-year lifetime.

Two routes of exposure, ingestion and dermal contact, are addressed in developing AALs for exposure to toxic chemicals when soil is the medium of exposure. A reasonable and conservative estimate of the average daily soil ingestion is 0.15 g/day. A reasonable and conservative estimate of the average daily soil exposure by dermal contact is 0.45 g/day.

In accordance with the methodology outlined in Chapter 4 of the California Site Mitigation Decision Tree Manual, May 1986, AALs are developed by allocating the Maximum Exposure Level (MEL), which is determined for each toxic substance to the estimated amount of medium to which a receptor is exposed daily. Toxicokinetic considerations are also addressed during the AAL development process. Toxicokinetic considerations would be expected to be a very important element in developing AALs for soil contact. Differences in the toxicokinetics of toxic substance that result from exposure by the ingestion of dermal route would be expected. Therefore, careful consideration of toxicokinetics should be an important element of developing AALs for soil contact. For the purpose of developing AALs for an exposure to toxic chemical by the medium of soil:

$$\text{AAL soil contact} = \text{MEL}/(\text{TF1} \times 0.15 \text{ g soil/day} + \text{TF2} \times 0.45 \text{ g soil/day})$$

Where TF1 and TF2 are toxicokinetic factors or considerations.

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