PUBLIC HEALTH CONSULTATION

DONA PARK NEIGHBORHOOD

CITY OF CORPUS CHRISTI

NUECES COUNTY, TEXAS

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Prepared by

The Texas Department of Health (TDH)
Under a Cooperative Agreement with
The Agency for Toxic Substances and Disease Registry (ATSDR)

BACKGROUND AND STATEMENT OF ISSUES

The Texas Department of Health (TDH) was asked by the Agency for Toxic Substances and Disease (ATSDR) to review environmental data associated with the operation of a former American Smelting and Refining Co. (ASARCO) facility. Specifically, the TDH was asked to comment on the public health significance of the environmental contamination. The facility operated on a 86-acre site at 5500 Up River Road, Corpus Christi, Nueces County, Texas. At this facility, ASARCO operated a zinc refinery and smelter from 1942 until 1982 and oxide and sulfide plants from 1953 until 1982. The sulfide plant processed zinc sulfide concentrates by roasting, filtering, purifying, and electrolyzing procedures. Through the processes used, zinc sulfite concentrates yielded residues in the form of lead, zinc, and cadmium. The oxide plant processed zinc oxide through procedures of crushing, drying, leaching, filtering, purifying, and electrolyzing. These processes yielded lead, zinc, and cadmium. Since 1988, Encycle/Texas, Inc., a wholly owned subsidiary of ASARCO Inc., has operated a hazardous waste management facility at the site. Encycle has a permit to receive and store hazardous wastes and nonhazardous industrial solid wastes from offsite sources.

The former ASARCO facility is bounded on the north by the Corpus Christi Inner Harbor, to the west by the Interstate Grain Port Terminal, and to the east by a Coastal Refining and Marketing facility. Approximately 200 yards south of the facility, across Up River Road, is the Dona Park/Manchester Place neighborhood (Figure 1). There are approximately 150 residential properties in this neighborhood. In February 1994, at the request of the Corpus Christi City Manager, the Texas Natural Resource Conservation Commission (TNRCC) Region 14 office collected soil samples from around this industrial area. The City Manager and local residents had concerns of potential soil contamination caused by activities of the surrounding industries. Two samples, obtained from vacant lots in the Dona Park and Manchester Place neighborhoods, had lead levels greater than 500 mg/kg.

In April 1994, the TNRCC Region 14 Office collected approximately 150 additional samples from the Dona Park and Manchester Place neighborhoods as well as along Up River Road. Each sample was a four part composite collected from two adjacent properties and was analyzed for cadmium, lead, and zinc. Summary results from this sampling event are presented in Table 1.

Table 1
Summary Results - April 1994, TNRCC Sampling Event

Contaminant	Concentration Range (mg/kg)	
	Dona Park / Manchester Place	Up River Road
Zinc	379 - 7,130	11,000 - 15,800
Lead	30 - 895	690 - 1,180
Cadmium	<5 - 84	185 - 268

In June 1994, to further characterize individual properties, Dames & Moore, consultants to ASARCO, collected soil samples from 126 residential lots closest to the former ASARCO facility in the Dona Park/Manchester Place neighborhood. Two composite samples were collected from each residential lot, one sample from the front yard and one sample from the back yard. Each sample was a four-part composite collected away from the drip line of the house or other structures, away from the driveway or other area where vehicles are parked, and away from any other man-made or natural feature that could effect sample results. Samples were collected from the upper one inch of soil. On lots with vegetative cover, a six-inch by six-inch area of sod was peeled back to allow access to the underlying soil. In addition to surface soil samples, ten core samples to a depth of 12 inches were collected from areas that exhibited high concentrations of metals during previous sampling episodes. All samples were analyzed for total cadmium, lead, and zinc.

Zinc concentrations in the surface soil ranged from 33 to 8,500 mg/kg; lead concentrations ranged from 10 to 1,000 mg/kg; and cadmium concentrations ranged from less than one to 170 mg/kg. For the neighborhood as a whole, soil concentrations for all three contaminants were lognormally distributed with geometric mean concentrations of 1,314 mg/kg, 133 mg/kg, and 14 mg/kg for zinc, lead, and cadmium respectively (Figure 2).

Because cadmium can translocate into home grown vegetables, the TDH recommended and the TNRCC concurred that garden soil and homegrown vegetables should be collected and analyzed for metal content. Fifteen composite soil samples were collected from neighborhood gardens and cadmium concentrations were measured in 12 tomatoes, one squash, two cucumbers, one green onion, one radish, one bell pepper, two serrano peppers, one chili pepper, and one carrot. The concentration of cadmium in garden soil ranged from below detection limit to 31 mg/kg; cadmium concentration in the vegetables ranged from below detection limit to 11 mg/kg. There was a positive correlation between the concentration of cadmium in the soil and tomatoes; the vegetable for which there the most data was available (Figure 3).

In response to specific citizen concerns about the lead, cadmium, and zinc in the neighborhood, the TDH recommended biological screening for all residents in this area. The TDH recommended sampling blood for lead levels and sampling urine for cadmium and creatinine levels. It was decided that screening for zinc was not warranted because the significance of the information would be difficult to interpret; relationships between zinc exposure and serum and/or urine levels have not been established. ASARCO agreed to fund urine screening for cadmium and creatinine for all residents, and blood lead testing for those residents who did not have their blood lead tested during the Corpus Christi/Nueces County Health District blood lead testing effort in March, 1994.

Blood lead levels were determined for 405 individuals (137 children and 268 adults; March 1994 and February 1995) and urinary cadmium levels were determined for 95 people (February 1995). All adults had blood lead levels less than 25 microgram per deciliter (µg/dL), while eight children (5.8%) had blood levels greater than 10 µg/dL. The maximum cadmium level measured in the urine was 3 micrograms (µg) of Cadmium per gram (Cd/g) creatinine.

DISCUSSION

In a previous health consultation, the TDH determined that the levels of zinc found in the Dona Park neighborhood did not present a threat to public health [1]; therefore, a discussion of zinc will not be included in this health consultation. The primary route of exposure to the soil contaminants in this neighborhood is through ingestion of contaminated soil; however, since cadmium can translocate into plants, eating vegetables grown in contaminated soil also can be a source of exposure to this toxicant. The degree to which cadmium will translocate into plants is dependent upon factors such as the concentration of the cadmium in the soil, the pH of the soil, and the type of plant. The public health significance of exposure to lead and cadmium are discussed below. Site specific conclusions are provided where appropriate.

Lead

Lead In The Environment

Lead is naturally present in most soils. The natural lead content of soil derived from crustal rock typically ranges from <10 to 30 parts lead per million parts soil (ppm). Lead is widespread in the human environment as a result of industrialization. It is generally found in higher concentrations in urban environments, principally as a result of automobile emissions and the use of lead-based paint. Concentrations of lead in the top layers of soil varies widely due to deposition and accumulation of atmospheric particulates from numerous human activities associated with lead pollution, including driving automobiles. For example, concentrations of lead in the upper layer of soil next to roadways are typically 30 to 2,000 ppm higher than natural levels. These levels drop drastically with increasing distance from the road [2].

Routes Of Exposure To Lead

Because of the prevalence of lead in the environment, humans are exposed to lead through a variety of media including air, water, and soil, as well as through diet. The relative contribution of each of these sources to total lead intake varies with age and is dependent on site-specific characteristics.

Infants often are born with lead in their bodies due to their mother's past exposure to lead. Infants and children are exposed to lead mainly through diet and ingestion of non-food materials associated with normal early hand-to-mouth behavior. The degree to which hand-to-mouth behavior contributes to blood lead levels depends on the levels of lead in house dust, soil, and paint. In the United States, leaded paint continues to cause most of the severe lead poisoning in young children because it is the most widespread source and has the highest concentration of lead per unit of weight [3].

Most adults are exposed to lead from dietary sources. In some instances, occupational sources also are a significant source of exposure. A great deal of information on the health effects of lead has been obtained through years of medical observation and scientific research.

Susceptibility To The Effects Of Lead

Preschool-age children and fetuses are usually the most vulnerable segments of the population for exposures to lead. This increased vulnerability results from a combination of factors which include the following: 1) the developing nervous system of fetuses and neonates are more susceptible to the neurotoxic effects of lead; 2) young children are more likely to play in dirt and to place their hands and other objects in their mouths, increasing the opportunity for soil ingestion, (pica, the eating of dirt and other non-food items, also is more likely to occur in children); 3) the efficiency of lead absorption from the gastrointestinal tract is greater in children than in adults; and 4) nutritional deficiencies of iron or calcium, which are prevalent in children, may facilitate lead absorption and exacerbate the toxic effects of lead [3].

Effects Of Acute High Dose Exposure

The most serious effects of acute high dose lead exposure is encephalopathy, characterized initially by headache and drowsiness, and in more severe cases by coma, convulsions, and death. Virtually all children who recover from acute lead encephalopathy exhibit residual reduction in intelligence and behavioral dysfunction. Acute encephalopathy is usually associated with high blood lead levels (over 150 μg/dL). Another effect of acute high dose lead exposure is the Fanconi syndrome; an acute injury to the renal tubules that is characterized by spillage of glucose, protein, amino acids, and phosphates into urine.

Effects Of Chronic Low Dose Exposure

Chronic exposure to lead principally affects three organ systems: the hematologic system (red blood cells and their precursors), the central and peripheral nervous system, and the kidneys. Lead also has been shown to have adverse effects on the reproductive system in both males and females. Lead is especially harmful to unborn children. Exposure to lead during pregnancy has been correlated with premature births, low birth weight infants, and spontaneous abortions. While the impact of maternal and cord blood lead levels below 10 μg/dL have not been well-defined, reduced gestational age and reduced birthweight have been associated with blood lead levels of 10 to 15 μg/dL [3]. In addition, lead has been found to lower intelligence quotient (I.Q.) scores, slow growth, and cause hearing problems in children. These adverse effects can persist and lead to decreased performance in school.

Carcinogenicity Of Lead

Lead has not been shown to be carcinogenic in humans; however, high doses of lead have been found to produce kidney tumors in laboratory studies of rats and mice. The extremely high cumulative doses of lead used in animal studies are difficult to extrapolate to low-level exposure in humans, and do not provide a sufficient basis for quantitative risk assessment. Based on animal data, EPA currently classifies lead as a B2 carcinogen (probable human carcinogen).

Indices of Toxicity

Although no threshold level for adverse health effects has been established, evidence suggests that adverse effects can occur at blood lead levels at least as low as 10 μg/dL. The Centers for

Disease Control and Prevention (CDC) has determined that a blood lead level greater than or equal to 10 μg/dL in children indicates excessive lead absorption and constitutes the grounds for intervention. The 10 μg/dL level is based on observations of enzymatic abnormalities in the red blood cells at blood levels below 25 μg/dL and observations of neurologic and cognitive dysfunction in children with blood lead levels between 10 and 15 μg/dL [4].

Blood Lead/Soil Lead Relationship

A number of studies are available relating blood lead levels in children to levels of lead in the environment [5]. In general, blood lead levels rise 3-7 μg/dL for every 1,000 mg/kg increase in soil or dust lead concentration; however, this relationship is affected by factors such as the age of the children in the population at risk, the physical availability of the soil or dust to the children, the bioavailability of the lead in the soil, and differences in individual behavioral patterns. Various approaches used to estimate a "safe" level of lead in soil yield results ranging from approximately 285 mg/kg to over 1,000 mg/kg [5,6]. The wide range of estimated "safe" levels may be attributed to differences in: background blood lead levels, exposure to lead from other sources, the degree of protection considered adequate, and the soil lead level/blood lead level relationship used in the calculations. Additionally, the "target" blood lead level must also be considered.

Site-Specific Conclusions

At any given site, the concentration of lead in soil that would be considered protective of public health may depend on site specific factors; however, in Texas, it is generally accepted that except in the most extreme cases (ie., frequent contact by children exhibiting pica behavior) soil levels at or below 500 mg/kg provide a sufficient margin of safety to ensure that blood lead levels remain below 10 μg/dL. During investigative work, EPA has recommended a soil lead level of 400 mg/kg as a screen to decide if further investigation is necessary.

Most of the properties in this neighborhood do not have elevated soil-lead levels. Based on the distribution of the soil-lead concentrations, 97 percent of the samples collected were below 500 mg-lead/kg-soil. Except for a few select properties, potential exposure to lead in soil in this neighborhood is minimal.

Less than six-percent of the children tested had blood lead levels greater than 10 μg/dL. This percent is lower than what we might expect based on previous experience with data from the Early Periodic Screening Diagnosis and Treatment (EPSDT) program where approximately 10% of the blood lead levels measured are above 10 μg/dL. We were not able to correlate the elevated blood lead levels with specific soil lead levels; however, because of the ubiquitousness of lead, reasons for the elevated blood lead levels may be different for each individual.

Cadmium

Cadmium in the Environment

Cadmium is a naturally occurring metallic element that is present in small amounts in virtually all soils and rocks of the earth's crust. It is also present in coal and in both mineral and municipal sludge fertilizers. In its pure form, cadmium is a soft, silver-white metal that is easily cut with a knife. However, cadmium is not usually found in the environment as a pure metal, but instead is combined with other elements such as oxygen, chlorine, or sulfur. Cadmium concentrations in non-polluted soil are highly variable, depending upon sources of minerals and organic materials. The mean level of cadmium in uncontaminated topsoil in the U.S. is approximately 0.25 ppm. The presence of cadmium in air, water, or food cannot be detected by smell or taste, because it does not have a definite taste or odor.

Production and Uses: Cadmium (as cadmium oxide) is obtained primarily as a byproduct of the smelting of zinc-bearing ores and also from the refining of lead and copper from sulfide ores. Major uses of cadmium include nickel-cadmium batteries (35%), metal plating (30%), pigments (15%), plastics and synthetics (10%), and alloys and other miscellaneous uses (10%).

Sources of Environmental Contamination: Most cadmium in the environment is released by human activities such as mining and smelting operations, fuel combustion, disposal of metal-containing products, and application of phosphate fertilizer or sewage sludge. Cadmium is extracted from natural materials during the production of other metals including lead, zinc, or copper [7]. Soil becomes contaminated primarily through the deposition of airborne cadmium, land spreading of municipal sludge, and the application of phosphate fertilizers. Cadmium enters the environment through the air as a result of burning coal and household waste, and metal mining and refining processes.

Routes of Exposure to Cadmium

Ingestion: Food is the major sources of human exposure to cadmium in the general, non-smoking population. Average cadmium levels in U.S. food range from 1 to 42 parts of cadmium per billion parts of food (1-42 ppb). Adults consume approximately 30 μg of cadmium from food sources each day, absorbing approximately 1 to 3 μg. Of all food items, vegetables generally contain the highest levels of cadmium, particularly potatoes and leafy vegetables with levels of 42 ppb and 33 ppb respectively; grain and cereal products also contain elevated levels, approaching 24 ppb. Meats, fish, and poultry generally contain relatively low levels of cadmium (less than 6 ppb), except for organ meats such as kidney and liver, which, of course, concentrate cadmium and may have levels 10 to 100 times higher [7]. The uptake of cadmium into plants depends on the availability of soluble metal from the soil; this, in turn, is dependent upon the concentration of cadmium, the acidity of the soil, and the organic matter content. Cadmium concentrations of 800 mg/kg (lettuce leaves) and 1600 mg/kg (chard leaves) have been reported in vegetables grown in acid soil containing cadmium at a concentration of 320 mg/kg [13]. Thus, for populations surrounding hazardous waste

sites, increased ingestion exposure can result from eating fruits and vegetables grown in cadmium-contaminated soil as well as from direct ingestion of cadmium-contaminated soil present on food or hands. Consequently, in highly polluted areas, the daily oral intake of cadmium can be as high as 400 μg per day [7].

Inhalation: The inhalation route is another major source of cadmium exposure. Average concentrations in air range from less than 1 ng/m^3 in remote areas to 5 to 40 ng/m^3 in U.S. urban areas, with isolated measurements to 7,000 ng/m^3 in highly contaminated industrial areas [7]. Assuming an average air cadmium concentration of 10 ng/m^3 for indoor and outdoor air combined and a daily inhalation rate of 16 m^3 for an adult, the average cadmium intake by inhalation is 0.16 μg/day, of which about 25% or 0.04 μg/day will be absorbed. However, populations surrounding industrial sources or hazardous waste sites can be exposed to higher levels of cadmium through inhalation of fugitive emissions and/or blowing dust from cadmium-contaminated soil. In highly polluted areas, the amount of cadmium absorbed through inhalation can be as high as 2 μg/day. Another major source of inhalation exposure affects smokers, who absorb an additional 1 to 3 μg of cadmium per day for each pack of cigarettes smoked [7].

Toxicity and Target Organs

The toxic effects of chronic cadmium exposure occur primarily in the lungs and in the kidneys. Pulmonary effects are associated solely with inhalation exposures, while the kidney effects may occur after either oral or inhalation exposures [7,8].

Renal Effects: It has been hypothesized that there is a critical concentration of cadmium in the kidney, above which, cadmium-induced nephropathy will occur. Most cadmium-induced renal toxicity is probably associated with cadmium not bound to metallothionein. However, brush border membranes of the renal tubule may be damaged by metallothionein-bound cadmium. Damage is thought to occur when the renal cortical cadmium concentration exceeds the "critical" level of 200 μg/g wet weight [7,8]. At these levels, the amount of cadmium not bound to metallothionein becomes high enough to begin causing tubular damage. However, other researchers have proposed that, for the general population, the amount of cadmium accumulated in the renal cortex should not exceed 50 μg/g, a level corresponding to a urinary excretion of 2 μg of cadmium per 24-hours [9]. Long-term exposure to excessive cadmium can effect the kidneys, causing proximal tubular necrosis, lesions in the renal cortex, and kidney dysfunction. Common laboratory findings include the presence of protein, amino acids, and glucose in the urine. Average kidney cadmium levels in non-occupationally exposed 50 year-olds are approximately 15-30 μg Cd/g-wet-weight [10]. Cigarette smoking can double renal cortical cadmium concentrations. Because of the high amount of cadmium ingested through diet, the margin of safety for exposure to cadmium from other sources may be relatively small, particularly for smokers.

Gastrointestinal Effects: Following high-level, acute, oral exposure (doses above 0.07 mg/kg in humans), the main symptoms are nausea, vomiting, and abdominal pain. Because of the

size of the dose required for acute toxicity, gastrointestinal effects are not likely to occur from environmental exposures to cadmium.

Hematologic Effects: Because cadmium interferes with the uptake of iron from the diet, excessive cadmium exposure can cause anemia in humans and animals.

Musculoskeletal Effects: There is evidence that cadmium exposure may affect the metabolism of vitamin D in the kidney and cause disturbances in calcium balance and bone density. This suggests that accumulation of cadmium in the kidney may be partially responsible for the decreased bone density (osteoporosis), which is particularly common in elderly women.

Carcinogenic Effects: Chronic cadmium exposure has been weakly associated with lung cancer in humans and somewhat more strongly with lung cancer in rats. However, in the human studies, subjects were also exposed to other known carcinogens, such as arsenic and nickel, and smoking as a risk factor was not controlled. There is further evidence of a weak association between cadmium exposure and cancer of the prostate, kidney, and stomach, although these associations, likewise, are not well established. The EPA has classified cadmium as a probable human carcinogen by inhalation (Group B1) based on positive responses in rats and possible positive responses in humans [11]; however, inhalation is not the primary route of exposure at this site.

Recommended Exposure Limits

Studies on humans orally exposed to cadmium in cadmium-polluted areas of Japan report a No Observable Adverse Effects Level (NOAEL) for kidney toxicity (proteinuria) of 0.0021 mg/kg/day [12]. Using an uncertainty factor of 3, ATSDR has used this NOAEL to establish a chronic oral MRL of 0.0007 mg/kg/day [7]. The EPA used a toxicokinetic model to determine the level of chronic human oral exposure which would result the highest renal cadmium level not associated with significant proteinuria (200 μg/g wet weight). Assuming 2.5% absorption of cadmium from food and 5% from water, the model predicts a NOAEL for chronic cadmium exposure of 0.01 mg/kg/day from food and 0.005 mg/kg/day from water. Based on these NOAELs and an uncertainty factor of 10, the EPA calculated a chronic oral reference dose (RfD) of 0.001 mg/kg/day for food and an equivalent RfD for cadmium in water of 0.0005 mg/kg/day [11].

Considerations in Determining the Public Health Significance of Cadmium in Soil

Because of a number of substance-specific factors, determining public health significance for cadmium in soil using standard methodology may be inappropriate. These factors include:

1. Both the MRL and RfD consider only the total daily amount of cadmium intake, not the source of the cadmium. When assessing the hazard and risk of cadmium from a particular source, it is imperative that this be done in the context of total cadmium exposure. This means that to determine the public health significance of a particular soil level, the assessment must consider other sources of exposure, including dietary

intake and water ingestion, in addition to the various soil-related exposures such as soil ingestion, dust inhalation, and skin contact. Some consideration should be given to smoking; however, this is voluntary exposure that is avoidable.

2. Cadmium is a cumulative toxicant. The critical effect, kidney toxicity, occurs only after cadmium has accumulated in the renal cortex to a level of approximately 200 μg/g wet weight. This is thought to occur only after many years of exposure. Both the MRL and RfD are estimates of a "safe" daily intake occurring over a lifetime of exposure. Thus, estimating the public health significance of particular soil levels should also consider intake occurring over a lifetime of exposure.

3. In their calculation of the RfD, the EPA assumes a value of 2.5% absorption of cadmium from food and 5% absorption from water. The absorption of cadmium from the gastrointestinal tract varies and is dependent upon many factors. Limited observations on humans indicate an average absorption of about 5% [13]. However, absorption rates as high as 20% have been observed. Women may have higher absorption rates than men due to chronic, borderline anemia. Additionally, while there is little information regarding the rates at which children absorb cadmium from various media, other data would indicate that children tend to absorb some metals (such as lead) at rates 3-4 times greater than those observed for adults [2].

4. The daily intake of cadmium is greatly affected by the concentration of cadmium in food. Vegetables grown on cadmium-contaminated soil may contain higher levels of cadmium than generally reported for those food items. Thus, persons living in cadmium-contaminated areas who ingest home grown vegetables could be at greater risk.

5. Because a number of exposure and dosage factors are highly dependent upon age, simple examination of cadmium intake in terms of the RfD and a 30-year or 50-year exposure period (even when broken down into two periods such as 0-6 years and 6-30 years) may not adequately show what happens to kidney cortical concentrations of cadmium over a lifetime of exposure.

Determining the Public Health Significance of Cadmium in the Soil

To determine the public health significance of the cadmium in the soil, we developed a toxicokinetic model which employs all of the information known about cadmium intake, absorption, distribution, and excretion. We implemented the model in an Excel 5.0 spreadsheet, specifically designed for this analysis. Rather than dichotomizing the calculations only into two age groups (0-6 years and 6-30 years) and assuming that the various body parameters remain relatively constant during these periods, we utilized 46 different age groups from 0 to 100 years with the largest age group size being 5 years from age 30 to 100 years. Three to six month intervals were used from birth to 4 years of age, one year intervals were used from 4 to 22 years of age, and two to three year intervals were used from 22 to 30 years of age. Age-group and sex-

appropriate values for dietary intake, water intake, air inhalation, rates, body weights, heights, body surface area, soil ingestion rates, dietary absorption factors, and kidney weights were used in all calculations. In addition to normal dietary intake of cadmium, we used site-specific soil/vegetable cadmium data in conjunction with the toxicokinetic model to determine cadmium uptake from the consumption of home grown produce.

This model estimates total cadmium uptake (µg Cd/day) in humans from all sources including diet, drinking water, smoking, inhalation and ingestion of soil, and absorption of cadmium through the skin from soil contact. The total quantity of absorbed cadmium, less the excreted quantity in each age group, combined with published values for percent of total cadmium body burden in the kidneys and values for cadmium concentration in the kidney cortex with respect to concentration on the total kidney, is then used to predict the net concentration of cadmium in the kidney cortex (in µg Cd/g cortex wet weight) as a function of age. Additional details pertaining to the model are included in Attachment A.

Site-Specific Conclusions

To determine the public health significance of the cadmium in the soil, we used the model to determined the soil concentration which resulted in a kidney cortex concentration less than 40 µg Cd/kg wet weight. This kidney cortical concentration was used because it was 20% less than the 50 µg Cd/kg wet weight recommended by Laurwerys [9]. For non-smoking females who eat home grown produce the model predicts that a cadmium soil concentration of 49 mg/kg would result in a kidney cortex concentration of 39 µg Cd/kg wet weight (Figure 4). Based on the results of the model, cadmium soil concentrations greater than 49 mg/kg could pose a public health threat; however, the actual public health significance of these concentrations will depend upon residence-specific exposure factors. These include: the actual soil concentration, the presence of ground cover, the presence of a vegetable garden, the quantity and type of homegrown produce eaten, and the duration of exposure.

Urinary cadmium levels were determined for 95 individuals. In general, when urinary cadmium levels are less than 10 µg/g creatinine, renal dysfunction is considered unlikely [14]. We used the more stringent level of 5 µg/g creatinine as the level of potential health concern. The highest reported level measured was 3 µg/g creatinine; thus, for the people tested, excess exposure to cadmium was not indicated.

CONCLUSIONS AND RECOMMENDATIONS

1. Approximately six percent of the children in this neighborhood had blood lead levels greater than 10 µg/dL; however, it is not unusual for a small percentage of children in a neighborhood with older housing stock to have elevated blood lead levels. Children identified with blood lead levels greater than 10µg/dL should receive the appropriate follow-up as per the Centers for Disease Control and Prevention (CDC) guidance.

In general, the State of Texas does not consider soil lead levels less than 500 mg/kg to be a threat to public health. Except under the most extreme exposure conditions, soil lead levels below 500 mg/kg contribute little to children's blood lead levels. Approximately 97% of the soil samples from this neighborhood had lead levels less than 500 mg/kg; therefore, for the neighborhood as a whole we would not expect the soil to have a significant impact on blood lead levels. Conversely, soil lead concentrations greater than 500 mg/kg could represent a public health concern. The magnitude of the concern depends on site specific exposure factors such as the actual soil lead concentration, the available vegetative cover, the absence or presence of children on the property, and characteristics specific to the individuals living on the property. In general, many of the properties in this neighborhood had good ground cover, thus providing a barrier to most exposures. The potential for children to come into contact with lead levels greater than 500 mg-lead/kg-soil should be determined and efforts to minimize these exposures should be taken.

2. Excess exposure to cadmium was not indicated for the 95 individuals for whom urinary cadmium levels were determined. However, using a multi-source model, we estimated that soil cadmium levels greater than 49 mg/kg could pose a risk to public health. This conclusion is based on a conservative estimate of the maximum amount of cadmium that should be allowed to accumulate in the kidney cortex. The soil concentration of 49 mg/kg only applies when consideration is given to the concomitant ingestion of homegrown produce. We would not expect the cadmium concentrations measured in this neighborhood to present a significant public health threat to those individuals who do not eat homegrown produce.

   Based on the distribution of soil cadmium levels, approximately seven percent of the samples exceed 49 mg/kg. The actual public health significance of soil levels greater than 49 mg/kg depends upon factors such as the actual soil cadmium concentrations, the presence of a vegetable garden, the types of vegetables grown in the garden, the amount of cadmium in the vegetables, and the average daily amount and type homegrown produce consumed. Unless additional data indicate otherwise it would not be advisable to regularly eat vegetables grown in soil with cadmium levels greater than 49 mg/kg.

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CERTIFICATION

The Dona Park Neighborhood Health Consultation was prepared by the Texas Department of Health under the a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the Health Consultation was initiated.

Technical Project Officer, SPS, SSAB, DHAC

The Division of Health Assessment and Consultation, ATSDR, has reviewed this Health Consultation and concurs with its findings.

Chief, SPS, SSAB, DHAC, ATSDR

Figure 1

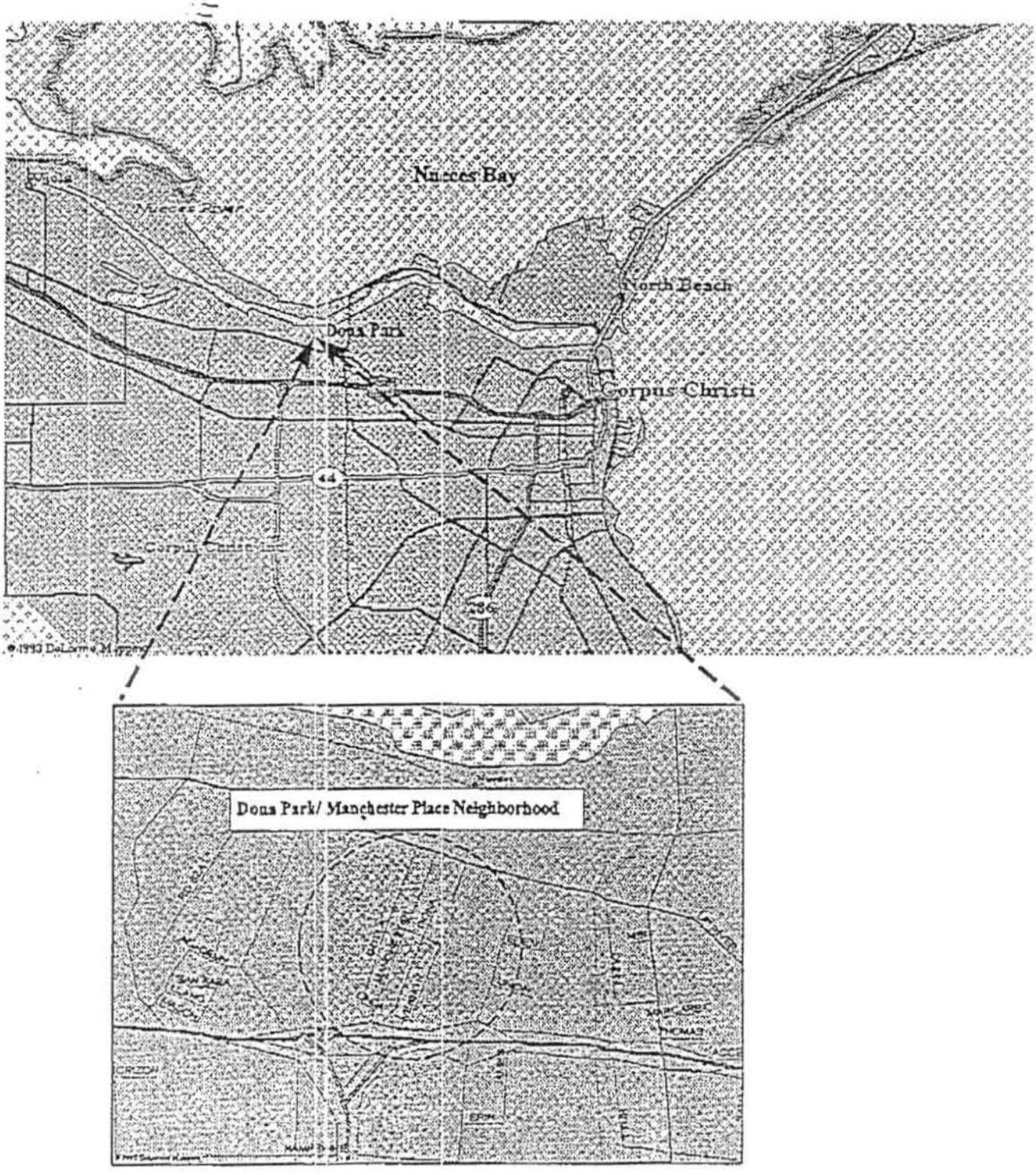
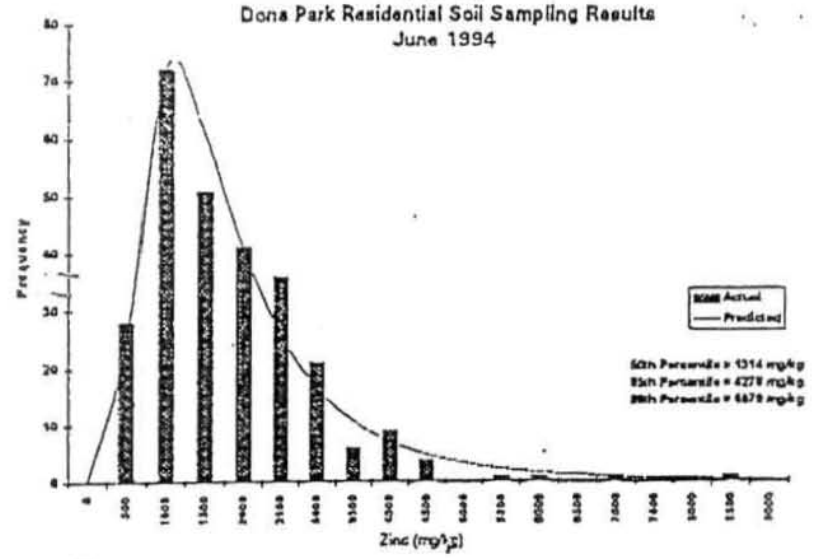
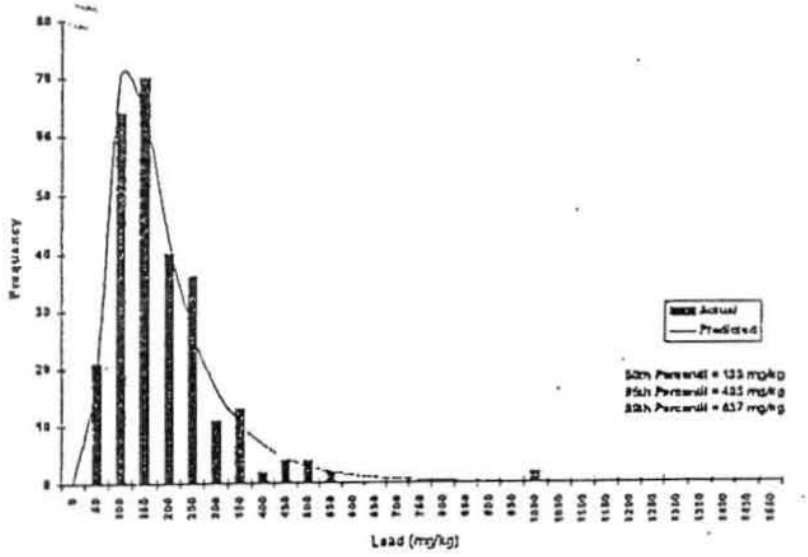

Nueces Bay
Nueces River
North Beach
Doña Park
Corpus Christi
44
Corpus Christi
Doña Park/ Manchester Place Neighborhood

Figure 2
Dona Park Residential Soil Sampling Results
June 1994





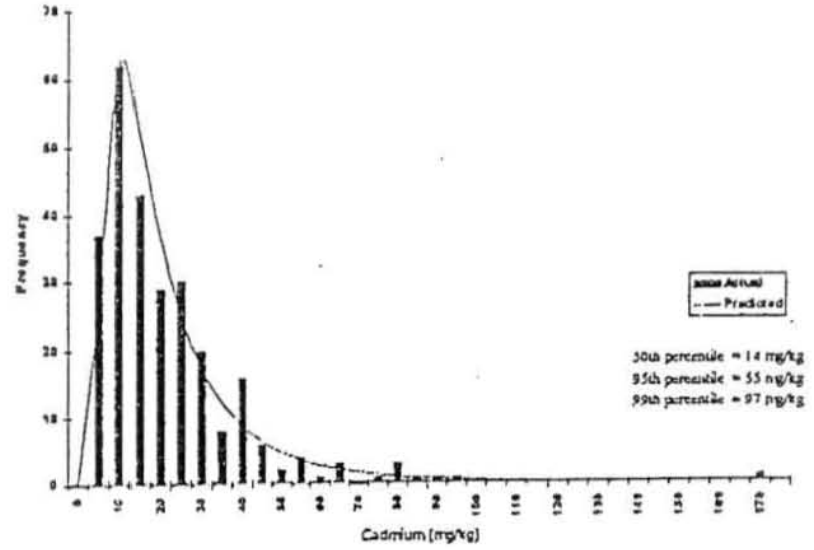


Figure 3

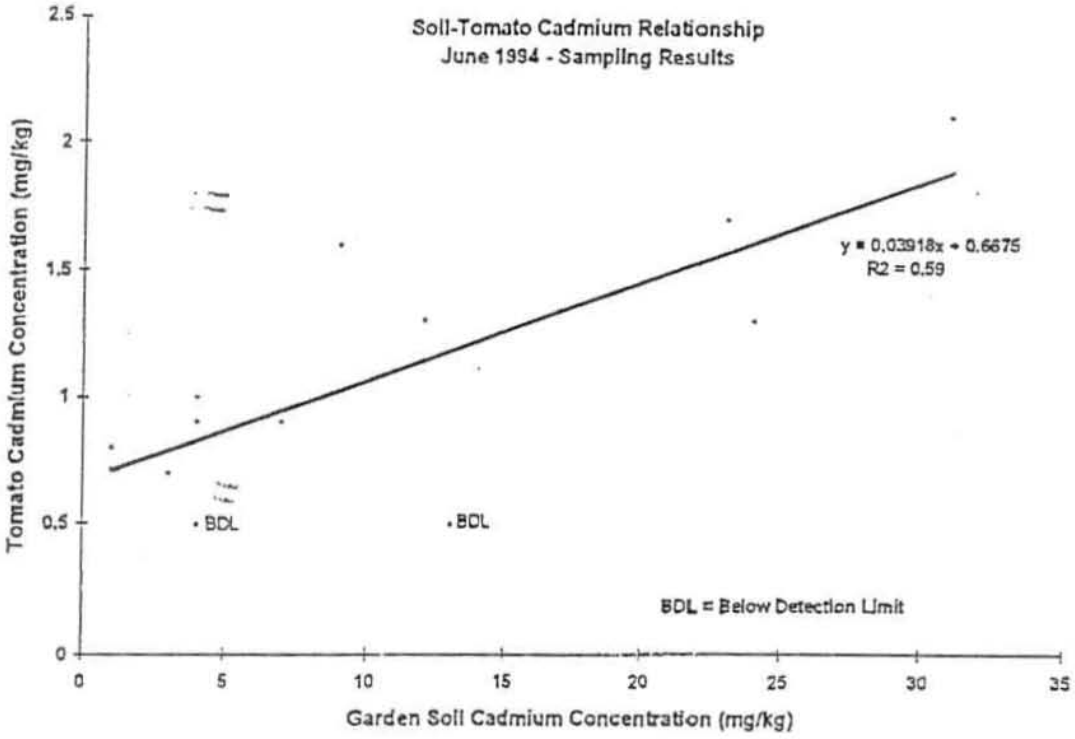
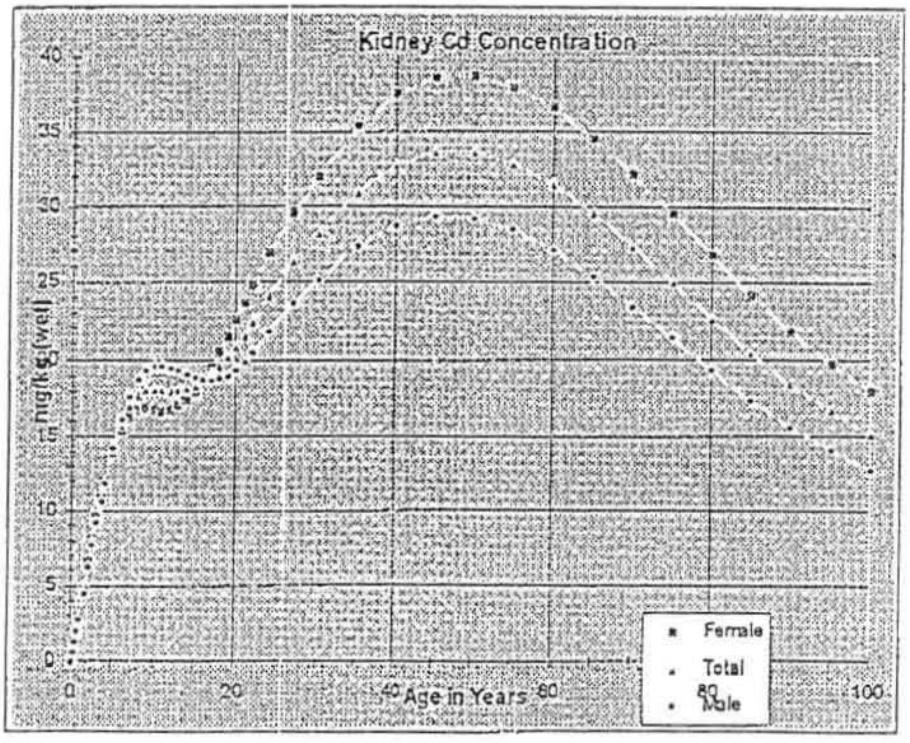


Figure 4
Predicted Lifetime Kidney Cortex Cadmium Concentrations
Non-Smoking Females Who Eat Homegrown Vegetables



Attachment A

Cadmium Model Details

Data Sources

Body weights, heights, and daily caloric intakes were derived from data published by the National Center for Health Statistics (NCHS). Combined kidney weights were obtained from Documenta Geigy Scientific Tables (Diem and Lentner, 1970). Body surface areas were calculated from mean height and weight data by the method outlined in the EPA's Exposure Factors Handbook (EPA, 1989). The estimated daily dietary intake of cadmium was obtained from the Toxicological Profile for Cadmium (ATSDR, 1993) and from the Environmental Health Criteria 134: Cadmium (WHO, 1992). Baseline kidney cadmium levels in the general population, dietary cadmium absorption fractions, biological half-life for cadmium in the body, percent of total body burden of c stored in kidney, cadmium concentration in the cortex with respect to cadmium concentration in the total kidney, the cadmium content of the average cigarette, percent of cigarette Cadmium inhaled during smoking, and percent absorbed were all obtained from WHO (1992). The number of cigarettes consumed per capita and the prevalence of smoking in the general population for the U.S. and Texas were obtained from Novotny et al. (1992). For consistency, all data items which vary significantly with age, such as body weight, height, surface area, respiratory daily volume, caloric intake, water intake, soil ingestion rate, and combined kidney weight were smoothed and values for intermediate ages were derived through least squares cubic spline interpolation.

Calibration of the Model

Kjellström (1979) in WHO (1992) reported kidney cortical cadmium concentrations for men from Japan, U.S., and Sweden. These results came from the general population without regard to smoking history. For men from the U.S., cadmium levels in the kidney cortex peaked at approximately 23 μg Cd/g cortex wet-weight at 45-50 years of age (Figure 1). In the U.S., the adult per capita cigarette consumption is approximately 2,920 per year, and the prevalence of cigarette smoking among adults 20 years and older is approximately 25.5% (Novotny et al., 1992). This amounts to a cigarette consumption of approximately 0.40 packs per day for the general population, where the "general population" consists of both smokers and non-smokers combined. Since only 25.5% of the population smokes, an average smoker, therefore, consumes approximately 1.5 packs of cigarettes per day.

Calibration of the model was achieved by setting the dietary intake to the reported U.S. average of 30 μg/day, the smoking level to 0.40 packs per day, and the soil level to 0 mg/kg. The model parameters were then adjusted to give a peak level of cadmium in the kidney cortex of approximately 23 μg Cd/g wet-weight at 45-50 years of age. Other baseline data (soil cadmium concentration of zero) were determined for males and females from four other distinct smoking categories. Peak renal cortex cadmium concentrations for each of the above smoking categories are presented in Table 1. From the table we see that smoking one pack of cigarettes per day approximately doubles the peak concentration of cadmium in the renal cortex (16.1 μg/g going to 35.2 μg/g in males and 21.4 μg/g going to 44.0 μg/g in females). This finding is consistent with published information (WHO, 1992).

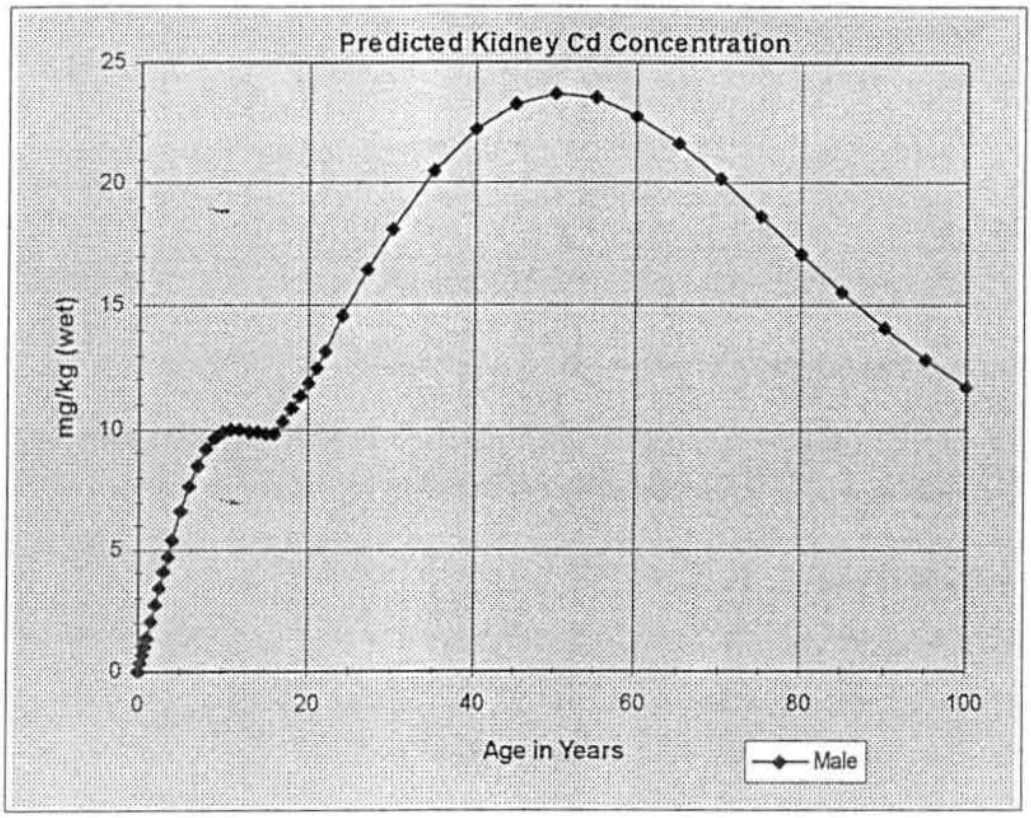
Assumptions Used in Baseline Calculations

Average Cd content in food per kcal of intake = .009906 μg/kcal

(Produces a maximum Cd intake of 30.0 μg/day for a 20-22 year old male;
an average Cd intake of 26.0 μg/day for males from age 0-50; and
an average Cd dose of 0.0004188 mg/kg/day for males from age 0-50)

Percent of dietary Cd absorbed from GI tract	=	5.0% absorbed
Additional absorption factor for newborn child	=	1.50 x
Additional absorption factor back to 1.0 @ age	=	10.0 years
Additional absorption factor applied to women over 20	=	1.50 x
Estimated max Cd concentration in local drinking water	=	1.00 μg/l
Percent of water Cd absorbed from GI tract	=	5.0% absorbed
Additional daily water ingestion factor (M&F)	=	1.00 x
Added daily water ingestion factor (F only)	=	0.90 x
Average Cd concentration present in local soil & dust	=	0.00 μg/g
Percent of soil Cd absorbed from GI tract	=	5.0% absorbed
Additional daily soil ingestion factor (M&F)	=	2.00 x
Added daily soil ingestion factor (F only)	=	0.90 x
Percent of soil Cd absorbed through skin	=	0.1% absorbed
Percent of total body surface area exposed	=	25.0% exposed
Amount of soil adhering per cm^2 exposed skin	=	0.200 mg/cm^2
Local levels of PM10 airborne particulates	=	25.0 μg/m^3
Percent of inhaled Cd absorbed through lungs	=	90.0% absorbed
Enter % time spent at heavy activity level	=	3.0% heavy
Enter % time spent at moderate activity level	=	7.0% moderate
Enter % time spent at light activity level	=	25.0% light
Remainder of time is spent at resting level	=	65.0% resting
Added respiratory daily vol factor (F only)	=	0.90 x
Average Cd content per cigarette	=	1.50 μg/cig
% Cd content inhaled when cigarette is smoked	=	10.0% inhaled
% inhaled Cd absorbed from cigarette smoke	=	66.7% absorbed
Age when person starts smoking	=	16.00 years
Number of packs per day person smokes	=	0.40 pk/day
Age when person stops smoking	=	100.00 years
Percent of total body burden of Cd in kidney	=	33.0% in kidney
Relative Cd concentration cortex/tot kidney	=	125.0% in cortex
Average biological ½-life, age 0-50	=	17.0 years

Figure 1



Predicted kidney cortex cadmium concentration accumulated over a lifetime for males. Assumptions include a soil cadmium concentration of zero and 0.4 packs per day smoked (population average for smokers and non-smokers combined).

Table 1
Predicted Peak Cadmium Concentrations in the Renal Cortex, Males and Females,
Dietary Cadmium Intake = 30 μg/day, Baseline Data (Soil Cadmium = 0 mg Cd/kg soil)

Smoking Category (packs/day)	Soil Cadmium Concentration (mg Cd/kg soil)	Peak Cadmium Concentration in the Renal Cortex (μg Cd/g wet weight)		Age at Which Peak Level Occurs (years)	
		Males	Females	Males	Females
0.00	0.0	16.1	21.4	45	50
0.40	0.0	23.7	30.3	50	50
1.00	0.0	35.2	44.0	55	55
1.50	0.0	45.0	55.5	55	55
2.00	0.0	54.7	67.0	55	55

Uncertainties in the TDH Toxicokinetic Model

There is a certain amount of uncertainty inherent to using this (or any other) model to determine the public health significance of cadmium levels for soil. While many of the assumptions used are based on scientifically verifiable findings, professional judgement and probabilities have also been utilized as well. Some of the potential sources of uncertainty are discussed below:

1. The critical value of 200 μg Cd/g wet weight is generally accepted as the lowest observable effects levels (LOAEL) for kidney damage. However, some have suggested that for the general public subtle effects may occur at kidney cortex cadmium concentrations as low as 50 μg Cd/g wet weight (Lauwerys et al., 1993).

2. The absorption factor distributions have been approximated from best available evidence and sound professional judgement. A somewhat higher absorption factor was used for children (7.5%) than for adults (5%), and a somewhat higher absorption factor was used for females over 20 (7.5%) than for males (5%).

3. Since using a constant value for the half-life, such as 17 years or 25 years, predicted an increasing kidney cadmium level even at 100 years of age, professional judgement was used in selecting a non-linear biologic half-life distribution. This distribution was selected to give an average half-life of 17 years from 0-50 years of age (consistent with values reported by Tsuchiya & Sugita, 1971 in WHO, 1992) and a kidney cadmium level that peaked at approximately 23 μg Cd/g wet weight at 45-50 years of age, consistent with values reported by Kjellström, (1979 in WHO, 1992). This ensured that the model, in the baseline state, was calibrated to acurately predict kidney cadmium levels that were consistent with data reported in the literature. While there may be other explanations for the lower kidney cadmium levels observed in older individuals, we chose a biological half-life distribution to fit the available data.

4. There is considerable variation in dietary cadmium intake. The values used in this model vary with caloric consumption and are within the range of values reported in the literature.

5. Dermal absorption of cadmium has been demonstrated in animal studies in which a 2-3% solution of cadmium chloride was painted on the bare skin of guinea pigs. Although no data has been reported to indicate that humans may absorb cadmium directly out of cadmium-contaminated soil which may adhere to skin, the model was designed to assume 0.1% absorption of the total cadmium present in soil adhering to skin. Daily exposure with 25% of the total body surface area uniformly coated with 0.20 mg soil/cm^2 of skin was assumed. We expect these assumptions to considerably overestimate the contribution of dermal absorption to overall cadmium exposure. Even with these very conservative assumptions, dermal absorption contributed only a minor

amount (approximately 10%) to all soil-related exposures combined.

6. Likewise, inhalation of cadmium as predicted by this model is felt to very likely overestimate actual cadmium inhalation exposure. The model assumes a constant, daily PM10 airborne particulate level of 25 μg/m^3 (all coming from cadmium-contaminated dust) and a 90% absorption of total cadmium out of inhaled dust. Under these very conservative assumptions, dust inhalation still contributes only a very minor amount (approximately 5%) to all soil-related exposures combined.

7. The soil ingestion rates used in this analysis range from 100 mg soil/day for adults to a peak of 400 mg soil/day for a 2-3 year old child. The time-weighted-average soil ingestion rate for all ages combined was 114 mg soil/day. Constant, daily exposure to zero percent ground cover soil was assumed, and all soil ingested was assumed to be contaminated at the "average cadmium concentration present in local soils." Under these assumptions cadmium absorption from ingested soil contributes approximately 85% to all soil-related exposures combined. Although actual soil ingestion rates may vary considerably from one individual to another, the assumptions used in this analysis are felt to be conservative with respect to protecting public health.

REFERENCES

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4. Lauwerys RR, Bernard AM, Buchet JP, Roels HA. 1993. Assessment of the health impact of environmental exposure to cadmium: Contribution of the epidemiologic studies carried out in Belgium. Environmental Research 62:200-206.

5. Novotny TE, Romano RA, Davis RM, Mills SL. 1992. The public health practice of tobacco control: Lessons learned and directions for the states in the 1990s. Annu Rev Publ Health 13:287-318.

6. WHO. 1992. Environmental Health Criteria 134: Cadmium. Geneva, World Health Organization.