HEALTH CONSULTATION

Exposure Investigation
Herculaneum Lead Smelter Site
(aka: Doe Run Lead Smelter)
Herculaneum, Jefferson County, Missouri

EPA Facility ID: MOD006266373

June 10, 2005

Prepared by

U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry
and
Missouri Department of Health and Senior Services

Table of Contents

Abbreviations and Acronyms

ATSDR	Agency for Toxic Substances and Disease Registry
ACGIH	American Conference of Governmental Industrial Hygienists
CDC	Centers for Disease Control and Prevention
EI	exposure investigation
EPA	U.S. Environmental Protection Agency
FDA	Food and Drug Administration
G	grams
MDHSS	Missouri Department of Health and Senior Services
NCEH	National Center for Environmental Health (CDC)
mg/kg/day	milligrams per kilogram (of body weight) per day
mL	milliliter
MRL	minimal risk level
ppm	parts per million
PTTIL	Provisional Total Tolerable Intake Level
USDA	U.S. Department of Agriculture
µg/L	micrograms per liter

Executive Summary

The Herculaneum lead smelter is an active facility that has been operating since 1892. The Doe Run Company currently owns and operates the smelter. The facility is located approximately 25 miles south of St. Louis, Missouri, on the Mississippi River. A lead ore concentrate, consisting of approximately 80% lead sulfide, is processed at the smelter. The ore is transported by truck from eight lead mines operated by the company near Viburnum, Missouri, approximately 75 miles south-southwest of Herculaneum.

Environmental sampling has shown lead contamination throughout the community and high prevalence rates of elevated blood lead levels in children less than 6 years of age (ATSDR, February 2002). Several exposure reduction activities have been implemented including smelter emission and fugitive dust reductions, soil yard replacements, and in-house dust removals (ATSDR, April 2002).

A November 2002 health consultation by the Missouri Department of Health and Senior Services (MDHSS) and the Agency for Toxic Substances and Disease Registry (ATSDR) concluded that information was not available to determine if past exposure to cadmium from air, soil, and dust was a health hazard; and therefore, categorized these possible exposures as an "indeterminate" health hazard. Uptake of heavy metals (cadmium, lead, and arsenic) by locally grown and consumed garden vegetables was also a route of exposure on which no data was available and therefore categorized as an indeterminate health hazard. However, current arsenic levels in air, soil, and dust do not pose a health hazard.

In the summer of 2003, an exposure investigation (EI) was conducted to address an indeterminate public health hazard. Exposures to cadmium, arsenic and lead among residents who currently live near a lead smelter in Herculaneum, Missouri were the primary objective. Two data gaps existed at this site: 1) past community exposures to cadmium via air, soil, and indoor dust contamination, and 2) current exposures to cadmium, arsenic and lead that may be present in home grown vegetables.

Urinary cadmium levels were measured to investigate the possibility of past exposure to cadmium. Urinary cadmium values primarily reflect body burden of cadmium, thus allowing insight into past exposures. The estimated half-life of cadmium in humans is between 10 to 30 years. Cadmium concentrations in urine samples from participants were not at levels that are associated with adverse health effects. Therefore, the measured exposures to cadmium pose no apparent public health hazard.

Current arsenic levels in air, soil, and dust do not pose a health hazard.
However, residents had expressed concerns about their current arsenic exposure. As a public service to the community, urinary arsenic testing was offered to alleviate community concerns. Arsenic concentrations in urine samples from participants were not at levels associated with adverse health effects. Therefore, the measured exposures to arsenic pose no apparent public health hazard.

Cadmium, arsenic and lead were measured in locally-grown vegetables to determine if they were safe for consumption. In this investigation, arsenic concentrations in vegetables were not likely to pose a health risk. Cadmium concentrations were not likely to pose a health risk if consumed occasionally for a few months each year. Lead concentrations in vegetables were not likely to pose a health risk to adults if consumed occasionally for a few times a week when in season. Children and pregnant women in the study area should avoid eating locally grown green leafy vegetables such as lettuce, polk wild greens and spinach.

Prudent and protective public health gardening practices are recommended to decrease future exposure to metals in Herculaneum. Detailed recommendations are listed in this document.

Background

The Herculaneum lead smelter is an active facility that has been operating since 1892. The Doe Run Company currently owns and operates the smelter. The facility is located in Herculaneum, Missouri, about 25 miles south of St. Louis, Missouri, on the Mississippi River. Doe Run processes a lead ore concentrate, consisting of approximately 80% lead sulfide, at the smelter. The ore is transported by truck from eight lead mines operated by the company near Viburnum, Missouri, approximately 75 miles south-southwest of Herculaneum. The 52-acre Herculaneum facility consists of a smelter plant, a 24-acre waste slag storage pile, and an on-site sulfuric acid plant (MDNR 1999).

According to the 2000 Census, Herculaneum has an estimated population of 2,805 people. Several homes are within 200 feet of the smelter facility, and at least four homes are located within 200 feet of the slag pile. Three schools are located in the city.

Environmental sampling has shown lead contamination throughout the community and high prevalence rates of elevated blood lead levels in children less than 6 years of age (ATSDR 2002a). Several exposure reduction activities have been implemented including smelter emission and fugitive dust reductions, soil yard replacements, and in-house dust removals (ATSDR 2002b).

In mid-2002, the U.S. Environmental Protection Agency (EPA) collected air, soil, and dust data in Herculaneum. In November 2002, the Missouri Department of Health and Senior Services (MDHSS) and the Agency for Toxic Substances and Disease Registry (ATSDR) reviewed this EPA data and issued a public health consultation in 2002. (ATSDR 2002c).

The community was concerned that past and current arsenic and cadmium emissions from the smelter would cause adverse health effects, especially to the kidney. Based on the analysis of EPA sampling data, we concluded that the current exposure to arsenic and cadmium in soil, air, and dust did not pose a health hazard, although past exposures were likely to have been higher (ATSDR 2002c). Because we lacked data for past exposures to

arsenic and cadmium, we classified those as an indeterminate health hazard. In this health consultation, exposure to cadmium, lead, and arsenic from garden vegetables was also classified as an indeterminate health hazard because of insufficient data.

To better characterize human exposure to site-related metals, ATSDR and MDHSS conducted an exposure investigation (EI) to address community concerns. ATSDR and MDHSS also wanted to help the community better understand the potential past and ongoing exposures to environmental contaminants. Therefore, this EI had three objectives:

1) Measure urinary cadmium levels to investigate the possibility of past exposure to cadmium. Urinary cadmium values primarily reflect body burden of cadmium, thus allowing insight about past exposures. The estimated half-life of cadmium in humans is 10–30 years.

2) Measure cadmium, arsenic, and lead in locally grown vegetables to determine if they are safe to eat.

3) Measure urinary arsenic (total and speciated) levels three separate times over a 2–3 month period in the same person. Serial arsenic testing would provide a public health service to the community and support the health consultation conclusion. Although current environmental arsenic levels do not pose a health hazard, residents have expressed concern about their current exposure to arsenic. This effort would assist in alleviating concerns and further reinforce that current arsenic levels do not pose a hazard.

Results of this EI will help local agencies decide whether public health actions are needed to reduce exposure and may assist in determining a focus for future studies. The results of this investigation are applicable only to the individuals tested and cannot be generalized to other populations. In addition, results of this exposure investigation cannot be used to predict the future occurrence of disease.

Target Population

This exposure investigation had specific eligibility criteria for each of the three objectives: urine cadmium testing, urine arsenic testing, and homegrown vegetable sampling for heavy metals (cadmium, arsenic and lead). We attempted to recruit 30 residents each for urine cadmium and arsenic testing. There was some overlap among these target populations. Therefore, one person could potentially qualify for both cadmium and arsenic urine testing.

Eligibility criteria were developed to select for the worst-case, nonoccupational exposure scenario. For example, participants who lived closest to the smelter were preferred, because they may have a significantly higher exposure to metals than those that lived farther away. People who had lifestyle factors that could contribute to an increased body burden of a contaminant were eliminated. For instance, smoking contributes to cadmium

body burden; therefore, only nonsmokers were eligible. If evidence of increased exposure was not found in this potentially highly exposed group of residents, those with lower exposure risk factors (e.g.: live far from smelter) are less likely to be affected.

(1) For urine cadmium testing, the participants met the following eligibility criteria:

- 10 years of age or older
- Nonsmoker or ex-smoker for more than 10 years
- No current kidney disease
- Lived in Herculaneum at least 10 years
- No history of occupational exposure to cadmium
- Live within a ½-mile radius of the smelter
- Nonremediated home (yard and interior of home)

(2) For arsenic urine testing, the participants met the following eligibility criteria:

- Live within a ½-mile radius of the smelter
- Lived in Herculaneum at least 1 year
- No history of current occupational exposure to arsenic
- Nonremediated home (yard and interior of home)
- Will remain at their current home for the next 6 months to assure availability for serial arsenic testing

(3) Homegrown vegetable sampling for cadmium, arsenic and lead occurred between June and August, the season when home-grown fruits and vegetables are available for testing. Fifteen vegetables samples were collected. For home-grown vegetable analysis, a participant had to meet the following criteria:

- Provide home-grown vegetables; green leafy vegetables and root vegetables were preferred.
- Live within ½ mile of the smelter

A few exceptions to the eligibility criteria for urine and vegetable sampling are discussed below. Limited quantities of homegrown fruit and vegetables were available in the area. Consequently, two vegetable samples from one garden (onions and turnips) were collected just outside a ½ mile of the smelter, but within 1 mile of the smelter. Several homes within ½ mile of the smelter had yard remediation; therefore additional participants were recruited if they lived just outside ½ mile of the smelter and still met all the other selection criteria. Four cadmium urine samples were obtained from individuals who lived just outside the ½-mile radius of the smelter, but within 1 mile of the smelter. Four arsenic urine samples were obtained from individuals who lived just outside the ½-mile radius of the smelter, but within 1 mile of the smelter.

Test Procedures

During the week of May 20, 2003, staff from ATSDR/MDHSS held a public meeting with area residents to inform them of the exposure investigation and participation criteria.

Community members were asked to sign up if they were interested in participating. The following week, staff from the MDHSS contacted interested residents by telephone to assure eligibility criteria and set up appointments for biological and environmental testing.

During the week of June 16, 2003, ATSDR/MDHSS met with eligible participants at their homes. Each adult participant completed a written informed consent form. Parents or guardians of children gave written permission to test eligible children or wards. Children, ages 7–17 years old, gave their written assent. In addition to completing consent/assent forms, each participant was asked a few questions to gather information on risk factors for exposure to cadmium and arsenic through food pathways, contact with contaminated soil, or use of other cadmium and arsenic containing products.

Biological Samples

Urine arsenic samples were collected three separate times from the same individual over 3 months. Serial urine arsenic samples were collected during the weeks of June 16, July 14, and August 18, 2003. Urine cadmium samples were only collected once from an eligible participant.

A 24-hour urine collection is considered an optimal sample because of fluctuations in excretion rates. However, exposure studies often use a first morning void or a random spot sample because it is easier to collect. To control for differences in urine output and dilution, urine creatinine is measured. Under steady state exposure conditions, as would be assumed for most residents of this community, random or spot urine results have correlated well with 24-hour results. Cadmium spot urine samples adjusted for creatinine have been shown to be comparable to a 24-hour urine collection for cadmium (Trevesian 1994). In addition, the Third National Health and Nutrition Examination Survey urine cadmium analysis collected “spot” or random urine samples from their subjects (Paschal 2000). In this investigation, arsenic urine samples were also collected as “spot” or random samples.

A 70-mL plastic urine cup was given to each participant and he/she was instructed to provide a mid-stream void urine sample. Urine samples were random urine samples, not first morning void urine samples. Parents of young children were given verbal instructions on how to assist their children in collecting a urine sample. ATSDR/MDHSS staff collected the urine sample from the participant, followed proper chain of custody procedures, and froze the urine sample with dry ice for shipment to the National Center for Environmental Health Laboratory (NCEH) in Atlanta, Georgia, for analysis.

The NCEH analyzed urine samples for total arsenic and cadmium using inductively coupled plasma-dynamic reaction cell-mass spectrometry (ICP-DRC-MS). Speciated arsenic was analyzed using high performance liquid chromatography (HPLC). Creatinine concentrations were measured with an automated clinical Kodak 250 analyzer using the

manufacturer's single slide, two-point enzymatic method. The laboratory followed method-specific quality assurance and quality control (QA/QC) procedures.

Environmental Samples

Eligible participants were asked to wash and prepare the fruit/vegetable as they would for eating or cooking. Green leafy vegetables and root vegetables were preferentially selected because they tend to take up metals.

ATSDR/MDHSS staff collected the vegetable samples and followed proper chain of custody procedures. Vegetable samples were placed and stored in doubled zip-lock bags and shipped on ice to the Food and Drug Administration (FDA) laboratory in Lenexa, Kansas, for analysis. The FDA analyzed the samples for lead, arsenic, and cadmium using graphite furnace atomic absorption spectroscopy (GFAAS). The laboratory followed method-specific QA/QC procedures.

Results and Discussion

Biological Samples

Cadmium

Cadmium urine levels are a good measure of cadmium body burden and of chronic exposure to cadmium. Urinary cadmium values primarily reflect the total body burden of cadmium and are proportional to the concentration in the kidney (Jarup 1998). The estimated biological half-life of cadmium in the body is 10–30 years (ACGIH 2001).

Thirty participants completed a consent/assent form and provided a urine sample for cadmium. The population consisted of 29 adults and one teenager, ranging in age from 17–86 years. The median age was 64.5 years and the mean age was 62.8 years. There were 22 females and 8 males. The mean and median length of residence in Herculaneum was 35 years. Twenty-one participants were ex-smokers and nine had never smoked in their life.

The individual test results for cadmium were compared to the 95th percentile value for the civilian U.S. population, as reported in the Centers for Disease Control and Prevention's (CDC) *Second National Report on Human Exposure to Environmental Chemicals* (CDC 2003). This report provides information on the body burden of urine cadmium in the general U.S. population, these levels are *not* levels at which health effects are likely.

Advances in laboratory methods allow for measurements of chemicals at very low levels in biological fluids. The presence of cadmium in a person's urine does not, by itself, signify that the chemical will cause disease. Separate research studies—not the CDC National Report—are needed to determine the urine levels of cadmium that are associated

with disease (CDC 2003). The CDC National Report merely states how much cadmium is in the urine of most people who are not *unusually* exposed to cadmium.

The comparisons were made using creatinine-normalized values to correct for differences in urine dilution. Because the percentile values vary by age, individual comparisons were made using an age-specific percentile value. For adults, gender specific comparison values were used. Table 1 shows the data summaries for the concentrations of cadmium detected in the urine samples. For statistical analyses, chemical concentrations below the analytical detection limit were assigned a value equal to the detection limit divided by the square root of 2. Cadmium was not detected in one of the urine samples.

Table 1. Urine cadmium concentrations for participants (n=30).

	µg/g creatinine
Mean	0.65
Median	0.59
Range	Not detected – 1.53

Most of the individual cadmium urine test results were within the 95th percentile of the U.S. population (95th percentile = 1.03 µg/g creatinine). However, the urine concentration of cadmium was elevated above the 95th percentile comparison value in seven participants. These seven participants were all older than 56 years of age (mean age = 74.8 years); six were females and one was male.

In this investigation, age strongly correlated with increasing cadmium urine levels (multiple regression: $r^2 = 0.7085$, $p = 0.014$). This finding is supported in the cadmium literature. In the general population, the concentration of cadmium in urine increases progressively with age (Lauwerys 1994). Cadmium has a long half-life in the body (10–30 years); therefore, this finding is also physiologically consistent because cadmium bioaccumulates in the body with age.

For workers with occupational exposure to cadmium, the American Conference of Governmental Industrial Hygienists (ACGIH) recommends that urine cadmium concentrations should be below 5 µg/g creatinine (ACGIH 2001). In the past decade, research has shown that a urine cadmium level of 2.5 µg/g creatinine is associated with a 4% excess prevalence of renal tubular damage in the general population (Wittman and Hu 2002). Therefore, cadmium levels should be kept below 2.5 µg/g creatinine in the urine to prevent tubular damage that can potentially progress to renal dysfunction (Wittman and Hu 2002). Evidence for environmentally exposed populations suggests that if exposure is stopped or kept very low, the progress to renal dysfunction is so slow that a person is unlikely to suffer from severe renal dysfunction (Jarup 1998; Hotz 1999). The maximum urine concentration of cadmium detected in this investigation (1.53 µg/g

creatinine) was detected in a person over the age of 75 and was well below these health-based levels and is not likely to result in cadmium-related adverse health effects.

For the general population, the two main sources of cadmium are diet and tobacco smoking (Buchet 1990). In Herculaneum, the potential community cadmium exposures from contaminated air and soil/dust deposition span decades.

With chronic exposure, cadmium accumulates in the body, primarily in the liver and kidney. The critical target organ is the kidney because it often exhibits the first adverse effects (Lauwerys 1994). Cadmium exerts its effect on the proximal tubule of the kidney. Renal damage associated with low-dose cadmium exposure in humans is similar to the age-related changes in glomerular and tubular function (Jarup 1998). The findings in this investigation do not reflect unusual cadmium exposure and therefore participants are not likely to develop cadmium-related kidney dysfunction. In addition, air emissions from the lead smelter have improved recently. This will help reduce further environmental cadmium exposure.

Arsenic

Urine arsenic is the most reliable method for measuring arsenic exposure, particularly for exposures occurring within a few days of the specimen collection. Speciated urinary arsenic is preferable to total urinary arsenic. The speciated forms can distinguish between exposure to inorganic arsenic and its metabolites and the relatively nontoxic forms of organic arsenic, commonly found in seafood. (Kallman 1990).

Because urine arsenic measurements reflect only very recent exposure (a few days), it only provides a small window of assessment to arsenic exposure. Periodic urine arsenic measurements in the same individual over a few months provide a more reasonable representation of exposure and may capture intermittent exposures.

Thirty participants completed a consent/assent form and provided a urine sample for arsenic in June, July, and August 2003. Some participants did not participate in all three urine arsenic testing events. The population consisted of 28 adults and two children, ages 12–81 years. The median age was 55 years and the mean age was 54.3 years. There were 18 females and 12 males. Table 2 presents data summaries for the arsenic urine concentrations. Figure 1 depicts total arsenic concentrations for all participants in June, July, and August 2003.

Table 2. Urine arsenic concentrations in participants in June, July, and August 2003.			
	June 2003	**July 2003**	**August 2003**
Number of Participants	25	29	27
Total Urine Arsenic (µg/L)			
Mean	7.31	11.22	35.78
Median	4.60	5.4	5.8
Range	0.3 – 47.6	1.0 – 132.1	0.8 – 431.7
Inorganic Arsenic (µg/L)			
Mean	3.86	4.11	4.44
Median	3	2.7	3
Range	ND – 16.7	ND – 13.9	ND – 23.9
ND = Not detected			

All urine arsenic samples were tested for inorganic arsenic, organic arsenic and total arsenic. Inorganic arsenic species include arsenous (III) acid and arsenic (V) acid, monomethylarsonic acid, and dimethylarsinic acid. Organic arsenic species include arsenobetaine, arsenocholine, tetramethylarsine oxide.

Inorganic arsenic is considered to be more toxic than organic arsenic. Organic arsenic is often found in seafood and shellfish. Consumption of seafood a few days before urine arsenic testing can elevate the total arsenic urine concentration.

Total Urine Arsenic

Arsenic levels in the participants varied among family members within a single household. The mean and median total arsenic urine concentrations differed among the three testing events, with a general increasing trend. Those individuals who had increases in total urine arsenic in a particular month had consumed seafood before urine collection, except for one person. The increasing trend for total arsenic levels could be attributed to increases in organic arsenic from seafood consumption. This is supported by a positive correlation with reported seafood consumption and total urine arsenic concentration (multiple regression: $r^2 = 0.3643$, $p = < 0.0001$).

Arsenic reference ranges are not included in the *Second National Report on Human Exposure to Environmental Chemicals*. However, the published literature reports urine arsenic concentrations in populations with background exposures. Normal total urine arsenic levels are thought to be less than 50 µg/L (Sullivan and Krieger 2001). No participants exceeded this concentration in June. One person exceeded this level twice, in July and August, and two people in August exceeded this level. In short, a total of three people exceeded 50 µg/L of total arsenic in the entire investigation.

The two August participants who exceeded the background level of 50 µg/L had total arsenic urine levels above 200 µg/L. Both of these individuals reported that they had consumed seafood before urine collection and their organic arsenic levels supported this assertion. Seafood consumption is likely to have contributed to this total urine arsenic level, based on the elevated *organic* arsenic levels. These individuals had organic arsenic concentrations in urine (193.1 µg/L and 326 µg/L, respectively) that support their report of seafood consumption prior to donating the urine sample. Inorganic arsenic was detected in these individuals (11.5 µg/L and 23.9 µg/L, respectively). Both individuals had normal total urine arsenic levels in June and July. Therefore, arsenic-related adverse health effects are not likely in these two people.

The individual who had a total urine arsenic concentration above 50 µg/L in July and August did not report seafood consumption a few days before each urine collection. In this individual, organic arsenic predominated in July and August (organic arsenic concentrations of 92.6 µg/L and 68 µg/L, respectively). *Inorganic* arsenic was detected in July and August in this participant and they were 9.8 µg/L and 7.4 µg/L, respectively. This individual may have been exposed to another source of organic arsenic that was not reported in our survey. This person's total urine arsenic was well below 50 µg/L in June. In short, the levels of inorganic arsenic and total arsenic detected in this person in July and August can be explained by organic (nontoxic form) arsenic levels. Therefore, arsenic-related adverse health effects are not likely to occur in this participant.

Inorganic Urine Arsenic

There is no national reference range for background concentrations of inorganic arsenic in urine samples from the general United States population. However, the results of several studies in the scientific literature indicate that the concentrations of inorganic arsenic in people with no unusual exposure to arsenic are usually less than 20 µg/L (Johnson 1989; Jensen 1991; and Andren 1988) and in one study less than 10 µg/L. (Kallman 1990). Inorganic arsenic is more toxic or harmful than organic arsenic. The ACGIH recommends that urine inorganic arsenic concentrations should remain below 35 µg/L (ACGIH 2001).

In this investigation, one person had an inorganic arsenic concentration (23.9 µg/L) in excess of 20 µg/L. This person reported eating seafood prior to urine collection and had a significant amount of organic arsenic (326 µg/L) in the urine. Organic arsenic predominates in seafood; however, seafood can still contain small amounts of inorganic arsenic. The seafood meal most likely contributed to the inorganic arsenic level in this person. In addition, this person did not report seafood consumption in the two previous testing periods and had inorganic arsenic levels below 10 µg/L. Therefore, this elevated inorganic arsenic concentration is not likely to result in arsenic related adverse health effects because it remains below the ACHIG level. In this investigation, no participant exceeded the ACGIH level (35 µg/L) for inorganic arsenic in the 3 months of testing. Thus, no adverse health effects from arsenic exposure are expected in participants.

In this investigation, serial urine arsenic testing was used to better represent arsenic exposure in an individual over time. No participant had urine *inorganic* (toxic form) arsenic levels in all three testing periods that were of health concern. In summary, the urine arsenic levels in this exposure investigation reflect low levels of exposure, consistent with what might be found in the general population. These levels would not be expected to cause arsenic-related health problems. A few individuals had elevated total arsenic levels in some months, but not all. These elevated total arsenic levels can be attributed mostly to seafood consumption and not to the toxic form of arsenic. Among these few individuals, arsenic-related health effects are not likely to occur, based on their urine measurements.

Environmental Samples

Fruits and Vegetables

The Herculaneum smelter has been active for more than 100 years. Accumulated airborne deposition of cadmium, lead, and arsenic onto area soils may have resulted in homegrown vegetables becoming a significant source of community exposure. Generally, there is not a strong relationship between levels of heavy metals in soils and plants (Vousta 1996). Soil levels of heavy metals cannot predict plant concentrations of heavy metals. Therefore, sampling home-grown vegetables for cadmium, arsenic, and lead provided valuable information for Herculaneum residents.

Fifteen home-grown vegetables were collected in June, July, and August 2003. Local residents provided vegetable samples for analysis. Fruit and vegetable samples were prepared and washed as they would be for eating or cooking. Fruits and vegetables were then analyzed for total arsenic, lead, and cadmium.

Individuals who provided the home-grown fruits and vegetables only ate them when ripe and in season. This reflects a few months of consumption each year. Table 3 presents data summaries of the metal concentrations detected in fruit and vegetable samples.

Table 3. Fruit and vegetable arsenic, lead, and cadmium concentrations, collected between June and August 2003, in Herculaneum, Missouri.				
Fruit / Vegetable	**Garden**	**Arsenic (ppm)**	**Cadmium (ppm)**	**Lead (ppm)**
Apples	A	0	0.004	0.100
Cucumbers	A	0.014	0.024	0.009
Green onions (2)	C, A	0	0.098, 0.031	0.364, 0.075
Green peppers	A	0	0.112	0.029
Lettuce (2)	B, C	0	0.046, 0.391	0.503, 1.737
Onions (2)	F, B	0	0.317, 0.026	0.080, 0.137
Polk wild greens	A	0	0.022	0.517
Potatoes	E	0	0.119	0.095
Tomatoes (2)	A, D	0	0.041, 0.228	0.047, 0.120
Turnips	F	0	0.019	0.132
Zucchini	A	0	0.010	0.008
( ) = number of samples collected ppm = parts per million; 1 ppm is equal to 1 ounce in 32 tons.				

Arsenic

Arsenic was not found in any of the fruits and vegetables tested, except for cucumbers. The risk posed by eating cucumbers would depend on the rate of consumption. For example, an adult (70 kg) could eat 1 cup of cucumbers per day which weighs 119 grams (US Dept. of Agriculture Database 2004).

The arsenic ingestion rate for eating 1 cup of cucumbers (119 gm) containing 0.014 ppm arsenic would be:

0.014 µg/g (ppm) x 119 grams ÷ 70 kg body weight = 0.0238 µg/kg/day

The result, 0.0238 µg/kg/day, is equivalent to 0.0000238 mg/kg/day.

This estimated arsenic dose is much less than ATSDR's chronic Minimal Risk Level (MRL) for arsenic of 0.0003 mg/kg/day (ATSDR, 2000). Eating these cucumbers is not likely to cause adverse health problems from arsenic exposure. To be protective of public health, ATSDR recommends that individuals follow general food safety guidelines. These include washing hands before preparing foods and washing foods before consumption.

Cadmium

Dietary exposure is a significant source of cadmium exposure in the general population. Grain and cereal products, potatoes, leafy vegetables, and root vegetables can contain high levels of cadmium (ATSDR 1999b). The cadmium levels in vegetables and fruits collected in this EI ranged from 0.004 - 0.391 ppm. If available, leafy vegetables and root

vegetables were preferentially selected for testing because they tend to take up more cadmium (ATSDR 1999b). The risk posed by eating cadmium-containing produce would depend on the rate of consumption. For example, an adult (70 kg) might eat 1 cup of lettuce (56 grams) per day, the vegetable with the highest concentration of cadmium (US Dept. of Agriculture Database 2004).

Using the highest cadmium concentration of 0.391 ppm, which was detected in lettuce, the maximum cadmium ingestion rate for this scenario would be:

0.391 µg/g (ppm) x 56 grams ÷ 70 kg body weight = 0.3128 µg/kg/day

The result, 0.3128 µg/kg/day, is equivalent to 0.00031 mg/kg/day.

This estimated maximum cadmium dose slightly exceeds the ATSDR chronic MRL for cadmium of 0.0002 mg/kg/day. Exposure to a level above the MRL does not mean that adverse health effects will occur. MRLs serve as a screening tool to help public health professionals decide where to look more closely for potential heath risks. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure.

The ATSDR chronic MRL for cadmium is based on exposure for 365 days a year for many years. However, the participants in this investigation ate home-grown vegetables occasionally, during a few months of the year, so their average daily dose would be considerably less than the value calculated above. Because of the large surface area of lettuce, a portion of the cadmium concentration may result from surface deposition. Peeling and thoroughly washing vegetables would reduce the amount of surface metal contamination.

The consumption of these fruits and vegetables is not likely to result in adverse health problems from cadmium exposure if consumed occasionally for a few months each year. To be protective of public health, ATSDR recommends that individuals follow general food safety guidelines. Those include washing hands before preparing foods and washing foods before consumption. Peeling and thoroughly washing vegetables would reduce the amount of surface metal contamination.

Lead

Many plants can take up lead from the soil (ATSDR 1999a). Edible plants acquire lead from the soil through their roots, by direct foliage uptake, and by surface deposition of particulate matter (ATSDR 1999a). Lead generally deposits superficially on leaves, with less leaf penetration than cadmium (Vousta 1996). At significantly high levels, lead is poisonous to plants and can stunt plant growth.

The lead levels in vegetables and fruits tested ranged from 0.008 – 1.737 ppm. Green leafy vegetables tend to accumulate lead on the plant surface. Root vegetables take up

lead into the plant tissue (ATSDR 1999a; Finster et al. 2003). The risk posed by eating lead-containing produce depends on the frequency and amount of consumption. An ATSDR MRL for lead is not available. Therefore, the U.S. Food and Drug Administration (FDA) provisional tolerable lead intake levels were used.

The FDA's recommended Provisional Total Tolerable Intake Level (PTTIL) for lead in children less than 6 years of age is 6 µg lead/day. For children 7 years and older, the PTTIL is 15 µg lead/day. It increases to 75 µg lead/day for adults (USFDA 1993).

The maximum concentration of lead detected in a plant in this investigation—1.737 ppm—was detected in lettuce. Plants with higher surface areas (green leafy vegetables, such as lettuce, collard greens, polk wild greens and swiss chard) tend to have higher lead levels (Finster et al. 2003). Contaminated soil and dust attaches to the plant surface more easily and tends to remain on vegetables that are not washed well.

The risk posed by eating lead-containing produce would depend on the rate of consumption. For instance, an adult might eat 1 cup of lettuce (56 grams) per day (USDA 2004)**.** Using the maximum lead level detected (1.737 ppm), an adult eating lettuce at that rate would ingest about 95 µg lead/day, which exceeds the adult PTTIL for lead. This ingestion estimate also exceeds the lead PTTIL for children. The other vegetables contained less than half the lead concentration found in lettuce. Most of the individuals who provided the home-grown fruits and vegetables only ate them when ripe and in season. This would result in a few months of occasional consumption each year.

Based on these assumptions, consumption of home-grown green leafy vegetables should be limited to a few times a week when in season. Lead content in lettuce, which has a large surface area, may partly result from deposition of lead-contaminated soil on the leaf surfaces.

To be protective of public health, ATSDR recommends that adults limit their consumption of green leafy vegetables to a few times a week when in season. Because young children and pregnant women are more sensitive to the effects of lead, they should avoid eating home-grown leafy vegetables. To reduce potential exposures to lead, home gardeners should preferentially plant fruiting vegetables instead of green leafy vegetables. Peeling and thoroughly washing vegetables can reduce the amount of lead contamination.

Reporting Results

In April 2004, individual test results and an explanation of their significance were provided to the participants of this investigation. An ATSDR physician was available to discuss participants' results.

Conclusions

These conclusions do not apply to arsenic and lead exposures that occurred in the past or to other residents in the area.

1. Cadmium concentrations in urine samples from participants were not at levels that are associated with adverse health effects. Therefore, the measured exposures to cadmium pose no apparent public health hazard.

2. Arsenic concentrations in urine samples from participants were not at levels associated with adverse health effects. Therefore, the measured exposures to arsenic pose no apparent public health hazard.

3. Home-grown fruits and vegetables were tested for arsenic, cadmium, and lead. Arsenic concentrations in vegetables are not likely to pose a health risk. Cadmium concentrations are not likely to pose a health risk if consumed occasionally for a few months each year.

4. Lead concentrations in vegetables are not likely to pose a health risk to adults if consumed occasionally for a few times a week when in season. Children and pregnant women in the study area should avoid eating locally grown green leafy vegetables such as lettuce, polk wild greens and spinach.

Recommendations

1. ATSDR recommends following general food safety guidelines. Those include washing hands before preparing or eating food and washing foods such as fruits and vegetables before consumption. Additional general food safety guidelines are outlined below for home-grown fruit and vegetable consumption to reduce potential exposure to metals:

- Wash all fruits and vegetables to remove soil.
- Peel fruits and vegetables to reduce surface contamination.
- Peel root crops (potatoes, carrots, beets, etc.).
- Discard older or outer leaves of leafy vegetables during food preparation.
- Do not compost unused plant parts, peelings, and parings for later use in a fruit and vegetable garden.

2. ATSDR recommends the following prudent, protective land use practices to decrease future exposure to metals from gardening in Herculaneum, Missouri:

- Avoid planting edible plants in soil with elevated heavy metal concentrations.
- Do not use CCA (arsenic containing) treated lumber to build raised beds.
- Plant fruit and vegetable gardens away from buildings, especially if peeling paint is evident.

3. To minimize absorption of lead by plants, a number of control measures may be taken:

- Maintain soil pH levels close to 7. Lead is relatively unavailable with a soil pH at this level.
- Lime the soil as recommended by soil testing to increase the pH of the soil.
- Add high phosphate fertilizer to the soil to decrease the plant uptake of soil lead.

4. Green leafy vegetables tend to accumulate lead on the plant surface and tend to have higher lead levels. Therefore, home gardeners should preferentially plant fruiting vegetables instead of green leafy vegetables. Some fruiting vegetables are corn, beans, squash, tomatoes, peppers, cucumbers, peas, apples, etc.

Authors

Prepared by:

Ketna Mistry, M.D., F.A.A.P.
Medical Officer
Exposure Investigations and Consultations Branch
Division of Health Assessment and Consultation
Agency for Toxic Substances and Disease Registry

Lori J. Harris-Franklin
Senior Epidemiology Specialist
Section for Environmental Public Health
Missouri Department of Health & Senior Services

Field assistance by:

Denise Jordan-Izaquirre
Regional Representative
Agency for Toxic Substances and Disease Registry

Rachelle Kuster
Environmental Specialist
Missouri Department of Health & Senior Services

Lorena Anderson, MEd., CHES
Health Educator
Missouri Department of Health & Senior Services

Carol Steinman
Health Program Representative
Missouri Department of Health & Senior Services

Gale M. Carlson, MPH
Chief, Hazardous Substances Assessment Unit
Missouri Department of Health & Senior Services

Reviewed by:

Susan Metcalf, M.D., M.P.H.
Team Leader
Exposure Investigations and Consultations Branch
Division of Health Assessment and Consultation

Susan Moore, M.S.
Acting Branch Chief
Exposure Investigations and Consultations Branch
Division of Health Assessment and Consultation

Acknowledgments

Michael Lewin—ATSDR, Division of Health Studies, for statistical analysis.

Duane Hughes—Food and Drug Administration, for vegetable processing and analysis.

Ken Orloff, Ph.D. and John Wheeler, Ph.D.—ATSDR, Division of Health Assessment and Consultation, for technical assistance with vegetable analysis.

Lynn Wilder, CIH—ATSDR, Division of Health Studies, for technical guidance on environmental sampling.

Kathleen Caldwell, Ph.D. and Robert Jones, Ph.D.—Centers for Disease Control and Prevention, for urine cadmium and arsenic laboratory analysis.

Kim Elmore, Ph.D.—ATSDR, for geographical information systems.

References

Agency for Toxic Substance and Disease Registry (ATSDR). 1999a. Toxicological profile for lead (update). Atlanta: US Department of Health and Human Services; 1999 Jul.

Agency for Toxic Substance and Disease Registry (ATSDR). 1999b. Toxicological profile for cadmium (update). Atlanta: US Department of Health and Human Services; 1999 Jul.

Agency for Toxic Substance and Disease Registry (ATSDR). 2000. Toxicological profile for arsenic (Update). Atlanta: US Department of Health and Human Services; 2000 Sep.

Agency for Toxic Substances and Disease Registry (ATSDR). 2002a. Health consultation on blood lead results for 2001 calendar year, Herculaneum lead smelter site, Herculaneum, Missouri. Atlanta: US Department of Health and Human Services; 2002 Feb 26.

Agency for Toxic Substances and Disease Registry (ATSDR). 2002b. Health consultation on determination if remedial actions are protective of public health, Herculaneum lead smelter site, Herculaneum, Missouri. Atlanta: US Department of Health and Human Services; 2002 Apr 16.

Agency for Toxic Substances and Disease Registry (ATSDR). 2002c. Health consultation on arsenic and cadmium levels in air and residential soils, Herculaneum Lead Smelter Site, Herculaneum, Missouri. Atlanta: US Department of Health and Human Services; 2002 Nov 12.

American College of Industrial Hygenists (ACIGH). 2001. Cadmium and inorganic compounds. In: Documentation of the biological exposure indices. 7th ed. Cincinnati: ACGIH Worldwide. p. 1–10.

Andren P, Schutz A., Vahter M., Attewell R., Johansson L., Willers S., Skerfving S. 1988. Environmental exposure to lead and arsenic among children living near a glassworks. Science of the Total Environment. 77(1):25-34.

Buchet JP, Lauwerys R, Roels H, Bernard A, Brauaux P, Claeys F, et al. 1990. Renal effects of cadmium body burden in the general population. Lancet 336:699–702.

Centers for Disease Control and Prevention. 2003 Jan 31. National Center for Environmental Health: Second National Report on Human Exposure to Environmental Chemicals. Available at URL: http://www.cdc.gov/exposurereport/pdf/SecondNER.pdf. Accessed April 2005.

Finster ME, Gray KA, Binns HJ. 2004. Lead levels of edibles grown in contaminated residential soils: a field survey. Sci Total Environ. 320(2-3):245–57.

Hotz P, Buchet JP, Bernard A, Lison D, Lauwerys R. 1999. Renal effects of low-level environmental cadmium exposure: 5-year follow-up of a subcohort from the Cadmibel study. Lancet. 354:1508–13.

Jarup L, Bergland M, Elidner CG, Nordberg G, Vahter M. 1998. Health effects of cadmium exposure: a review of the literature and a risk estimate. Scand J Work Environ Health 24(suppl 1):7-51.

Jensen GE, Christensen JM., Poulsen OM. 1991. Occupational and environmental exposure to arsenic--increased urinary arsenic level in children. Science of the Total Environment. 107:169-77.

Johnson LR and Farmer JG. 1989. Urinary arsenic concentrations and speciation in Cornwall residents. Environ Geochem Health 11:39-44.

Kallman, DA, Hughes J, Van Belle G, Burbacher T, Bolgiano D, Coble K, et al. 1990. The effect of variable environmental arsenic contamination on urinary concentrations of arsenic species. Environ Health Perspect 89:145–51.

Lauwerys, RR, Bernard A, Roels H, Buchet JP. 1994. Cadmium: exposure markers as predictors of nephrotoxic effects. Clin Chem 40(7):1391–4.

Missouri Department of Natural Resources (MDNR). 1999 Mar 30. Preliminary assessment: Herculaneum lead smelter site, Jefferson County, Missouri. Jefferson City, Missouri: Missouri Department of Natural Resources.

Paschal DC, Burt V, Caudill SP, Gunter EW, Pirkle JL, Sampson EJ, et al. 2000. Exposure of the US population aged 6 years and older to cadmium:1988–1994. Arch Environ Contam Toxicol 38:377–83.

Sullivan JB and Krieger GR. 2001. Clinical environmental health and toxic exposures. 2nd ed. Philadelphia: Lippincott Williams & Williams. Page 862.

Trevesian A, Nicoletto G, Maso S, Grandesso G, Odynets A, Secondin L. 1994. Biological monitoring of cadmium exposure: reliability of spot urine samples. Int Archives Occup Environ Health 65:373–5.

US Department of Agriculture, Agricultural Research Service. 2004. USDA National Nutrient Database for Standard Reference, Release 16-1; Nutrient Data Laboratory. Available at URL: http://www.nal.usda.gov/fnic/foodcomp. Accessed April 2005.

US Food and Drug Administration. 1993. Guidance document for lead in shellfish. Washington, DC: Center for Food Safety and Applied Nutrition. Available at URL: http://www.cfsan.fda.gov/~frf/guid-pb.html#sVI.

Vousta D, Grimanis A, Samara C. 1996. Trace elements in vegetables grown in an industrial area in relation to soil and air particulate matter. Environ Pollution 94(3):325–35.

Wittman R and Hu, H. 2002. Cadmium exposure and nephropathy in a 28-year-old female metals worker. Environ Health Perspect 110(12):1261–6.