

Appendix H—Information on Health Effects From Exposure to Arsenic and Lead

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Introduction

The risk of developing health problems from arsenic or lead depends on the amount of exposure and the concentrations to which a person is exposed. The greater the exposure is or the greater the concentrations are, the greater the risk is. Most information about the health effects of arsenic and lead comes from studies where exposures were greater than those expected from living and working in places with low to moderate levels of arsenic and lead in soil. Scientific studies to date have not found conclusive evidence that exposure to low to moderate levels of arsenic and lead contamination in soil has caused or is causing deleterious health effects in Washington residents. The number of pertinent studies is small, and their designs lack sufficient power to detect the presence of increased incidences of adverse health effects, if any do exist. Health monitoring and research studies have not been carried out to the extent necessary to understand and document whether exposure to low-to-moderate level arsenic and lead in soil is causing or contributing to long-term health problems in Washington.

Evaluating health effects at lower levels of exposure is difficult, and it is unlikely that conclusive scientific information to determine the health risks from exposure to area-wide soil contamination will be available in the foreseeable future. In light of this uncertainty, there is disagreement among scientists about how the information that is available should be interpreted and used to assess the risks of exposure to low to moderate level soil contamination. Some members of the scientific community argue that federal and state efforts to address low to moderate level soil contamination are not scientifically justified because there is no information demonstrating that health problems are being caused by exposure to such contamination. Other members of the scientific community argue that arsenic and lead in soil have the potential to cause health problems at low levels of exposure—especially for people who are particularly sensitive to the effects of these contaminants. In recent years, the majority of scientific review committees formed to evaluate the available scientific information on arsenic and lead have concluded that there is a sufficient scientific basis to justify efforts to reduce exposure to arsenic and lead.

Exposure to high levels of arsenic and lead can cause health problems in people. Arsenic can cause more than 30 distinct health effects, including nervous system damage, increased blood pressure, heart attack, stroke, and cancer of the bladder, lung, skin, and other organs. Lead can affect many parts of the body, causing health effects that include increased blood pressure, kidney damage, and brain damage. Although both children and adults can be adversely affected by lead poisoning, it is a particular concern for young children. Arsenic and lead are both considered persistent contaminants. This means that they bind strongly to soil and usually remain in the environment without breaking down or losing their toxicity, and thus can be a source of exposure for many decades.

Arsenic-Contaminated Soil and Public Health

A brief overview of how policy and science are used to evaluate the public health significance of area-wide contamination

Prepared for the Area-wide Soil Contamination Task Force, January 14, 2003

by

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Purpose of This Document

Three questions about public health aspects of area-wide soil arsenic contamination have been raised repeatedly by members of the Area-wide Soil Contamination Task Force:

1. Is area-wide soil arsenic contamination really a health hazard?
2. If so, how large is the hazard?
3. Where is the proof that people are being affected?

Brief answers to these questions are:

1. Scientific information about arsenic toxicity and people's exposure to soil indicates that area-wide soil arsenic contamination is a widespread public health hazard (as defined by state law) with potentially serious consequences for some individuals.
2. Depending on what it is being compared to, the health risk from area-wide soil arsenic contamination may be described as either large or small.
3. Scientific studies attempting to measure the effect of area-wide soil arsenic contamination on public health have been inconclusive. Specifically, available data are not sufficient to rule out, nor confirm, that the contamination is causing health problems.

This document attempts to explain these answers by providing insight into how public health agencies evaluate and address various hazards and briefly discussing scientific and risk considerations related to arsenic-contaminated soil.

The Roles of Policy and Science in Public Health

Addressing threats to public health often involves a mixture of scientific information and non-scientific policy choices (some of which are formalized as laws and regulations). As the Task Force continues its deliberations, it may be useful to have some information describing the roles of science and policy for public health agencies. In reality, policy and science in public health are not separate, but are deeply intertwined. However, one or the other is usually a driving force at each of the steps involved in addressing public health issues. Here is one way of listing the steps and the main driving force (science or policy) in addressing public health issues:

- **Policy choices** establish the importance of protecting public health.
- **Scientific information** is used to identify potential hazards.
- **Policy choices** define public health goals:
 - Which hazards should be addressed?
 - What is the desired degree of protection? (How safe is safe enough?)
- **Policy choices** guide how to choose, interpret, and use scientific data to meet public health goals. This may include guidance on dealing with uncertainty or lack of scientific information.
- **Scientific information** is used to evaluate the degree of risk posed to public health (within the framework established by the policy choices from the previous bullet).
- **Scientific information** can suggest ways to eliminate or reduce the hazard.
- **Policy choices informed by science** ultimately determine what actions are taken to protect public health.

Ultimately, policy choices that are informed by scientific information determine what actions are taken to protect public health.

The policies and scientific information used as the bases for public health decisions in Washington are generally similar to those used by other agencies across the country at the state and federal level. Both policy and science have played important roles in agency decisions about area-wide soil contamination. The following sections identify some of the important policy and scientific factors that have bearing on the evaluation of the public health significance of area-wide soil contamination.

General Public Health Policy

Good public health is a mandated goal. Policy, in the form of Washington state law, states that it is important to protect public health:

- RCW 43.70.005 – Intent. The legislature finds and declares that it is of importance to the people of Washington State to live in a healthy environment and to expect a minimum standard of quality in health care. (The first sentence authorizing the formation of the Department of Health.)
- RCW 70.105D.010 – Declaration of policy. (1) Each person has a fundamental and inalienable right to a healthful environment, and each person has a responsibility to preserve and enhance that right. (The first sentence of the Model Toxics Control Act (MTCA).)

Addressing health threats. To meet the goal of providing healthy, healthful, or safe conditions, policies determine which threats to health should be addressed. (Environmental chemicals, cigarette smoking, and communicable disease to name a few.) Many factors influence an agency’s choices, including:

- Who may be affected (Children? Adults?)?
- How many are affected?
- How serious is the health effect (Cancer? Skin irritation?)?
- Are resources available to address the hazard?
- Is it important relative to other public health hazards?
- Are there legal mandates that require the issue be addressed?
- Is there public and political interest?

As a general policy, preventing harm before it occurs is preferable to treating people after they have become ill. Because of limitations in scientific information about many hazards, it is sometimes deemed inappropriate to wait for *direct* scientific proof that harm is occurring before engaging in activities to reduce the hazard. Actions to prevent harm are often justified when there is sufficient *indirect* evidence that harm is likely to occur.

How safe? Policies define goals for safety. These goals are used to determine whether a hazard is sufficiently unsafe that it should be addressed. For environmental chemicals, safety goals depend on the chemical, where it is located, and the policies of the agency that oversees the hazard. For hazardous waste in the environment in Washington, “unsafe” typically means one of three things:

- the calculated excess risk of cancer from a chemical exceeds one in one million (WAC 173-340-700(5)(b)),
- the calculated excess risk of cancer from a chemical exceeds one in one hundred thousand (WAC 173-340-700(5)(c)), or
- the calculated risk of noncancer effects exceeds zero (WAC 173-340-700(5)(b) and (c)).

Science policy. Policy is used to determine how to choose, interpret, and use scientific information to evaluate hazards and meet public health goals. The choice, use, and interpretation of scientific data are often influenced by any special vulnerabilities of those we are trying to protect and by the degree of protection established for good health. Risk assessment calculations are widely accepted by public health agencies for evaluating environmental hazards when site-specific epidemiological information is unavailable or inconclusive (as is the case for area-wide soil contamination). Several sections of MTCA (WAC 173-340) discuss the use of risk assessment.

To summarize, public health policies recognize that:

- Protection of public health is important to society and mandated by law,
- The goal of a healthy and safe environment often involves protection against risks that some people might consider “small,”
- Prevention of problems before they occur is preferable to treating people after they have been harmed, and
- It may be appropriate to address some health threats even when strict scientific proof of harm is lacking.

Science Related to Arsenic-Contaminated Soil

Arsenic toxicity. The World Health Organization¹, the Environmental Protection Agency², the Agency for Toxic Substances and Disease Registry³, and two committees of scientists assembled by the National Research Council^{4,5} have performed comprehensive evaluations of the scientific literature related to arsenic toxicity and concluded that environmental arsenic can be a significant threat to human health. The scientific evidence that long-term exposure to arsenic can lead to many types of health problems is consistent and compelling. Principles of toxicology indicate that that some people could develop serious health problems, such as various forms of cancer, from exposure to arsenic at levels found in soil with area-wide contamination in Washington.

Soil exposure. Many studies⁶ show that the activities of people can lead to unintentional⁷ ingestion of soil and dust (and result in exposure⁸ to the chemicals they contain). The Environmental Protection Agency and the Agency for Toxic Substances and Disease Registry⁹ accept that unintentional ingestion of soil can be an important route of exposure.

Risk Assessment. Arsenic-contaminated soil has been evaluated as a public health hazard based on studies demonstrating that arsenic can harm people and that people can be exposed to chemicals by ingesting soil. Using information on arsenic toxicity and soil exposure, standard risk assessment calculations¹⁰ suggest that area-wide arsenic contamination presents risks that exceed Washington’s goals for public health protection.

Epidemiology. Because of several limitations, studies to date in Washington and elsewhere are scientifically inconclusive regarding the relationship between cancer rates and exposure to arsenic-contaminated soil. Scientific data offer no direct proof that people:

- have been harmed by arsenic in soil in Washington State, or
- have **not** been harmed by arsenic in soil in Washington State.

Cancer risk estimates derived through risk assessment calculations suggest that the increase in cancer rates from area-wide soil arsenic contamination is too low to be measured by practical epidemiological studies.

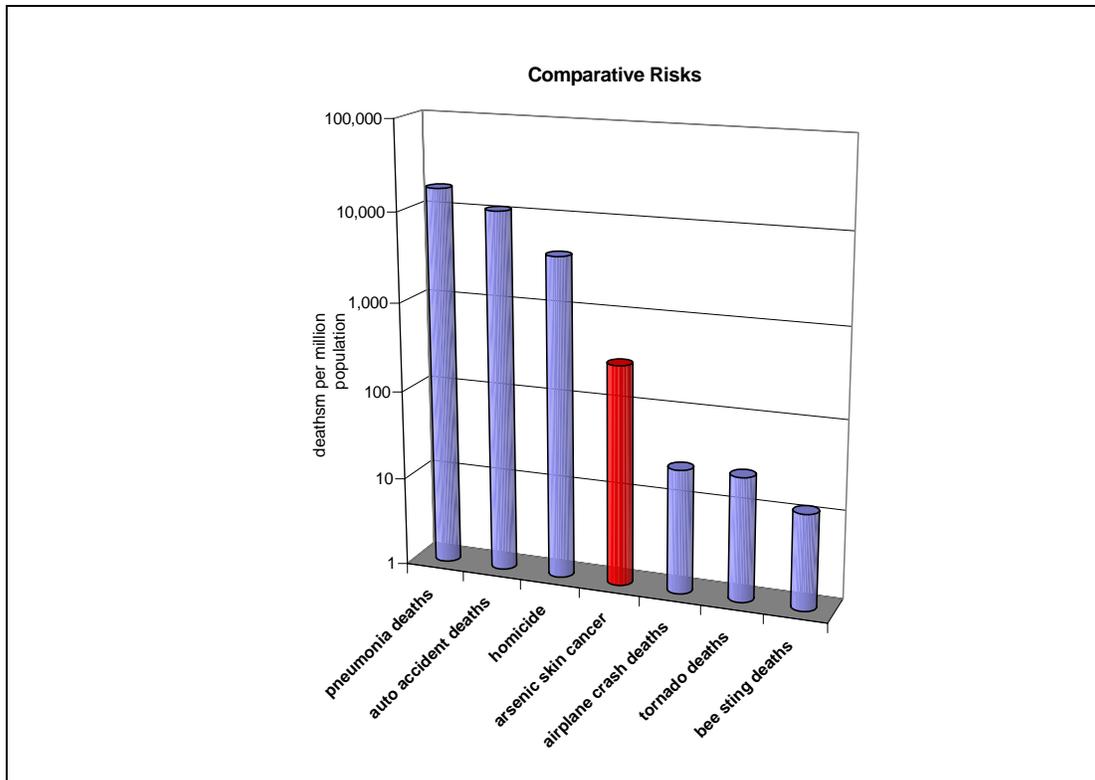
Risk of Harm from Arsenic-Contaminated Soil

Epidemiological studies to date do not provide adequate information about the hazards of area-wide arsenic-contaminated soil, but risk assessment calculations and the public health system in Washington suggest that the contamination problem should be addressed. Although the risk is different for each individual, general ballpark estimates of risk for a population can be developed for a few of the many possible health effects from exposure to arsenic associated with area-wide contamination.

As one example, using the standard Method B calculation in MTCA for arsenic in soil, a child exposed to 20 milligrams of arsenic per kilogram of soil for six years has an estimated increased risk of skin cancer of 30 per million. At 200 milligrams of arsenic per kilogram of soil, the estimated increased risk of skin cancer is 300 per million.

- This risk is **small** compared to the risk of dying of pneumonia (17,500 per million), in a motor vehicle accident (11,000 per million), or by homicide (4,000 per million).
- This risk is **large** compared to dying of a bee sting (12 per million), in a tornado (25 per million), or on a commercial airline (25 per million, pre-9/11/2001).
- This risk is **large** compared to the goal for maximum allowable cancer risk in most areas (1 per million) for cleanup of environmental contaminants, as established in cleanup laws in Washington and many other states.
- Because of the large number of people who are potentially exposed to area-wide soil contamination (including many future generations), and because of the toxicity of arsenic, the risk is **large** compared to risks from other sources of soil contamination (including most hazardous waste sites) where fewer people are likely to be exposed and the toxicity of the contaminants is likely to be less.
- The risk is **small** compared to our ability to measure it in a scientifically provable manner. *Note that the inability to directly measure the risk is a limitation of practical scientific techniques, which has little or no relationship to the public health significance of the risk.*

Different estimates of the risk of arsenic-contaminated soil could be developed. Adding the risk of skin cancer from the above calculation to the risk of lung and bladder cancer (not considered in the calculation) would result in a higher calculated risk. Alternatively, it could be assumed that arsenic in soil isn't fully absorbed by the body, leading to a lower calculated risk. In the long run, however, the soil arsenic risk calculation in MTCA uses widely-accepted input factors that are supported by scientific data. Reasonable changes to the above calculation are unlikely to result in substantially different risk estimates than those derived using the Method B calculation in MTCA for arsenic in soil.



A word of caution about comparing risks based on numbers alone. Level of concern can be influenced strongly by many other factors besides the calculated risk. For example, people are usually less concerned about risks that they control (driving a car), that provide a benefit (eating potentially contaminated fish), or that they have chosen (smoking), and more concerned about risks that they don't control (flying in an airplane), that don't provide a benefit (hazardous waste), or that they haven't chosen (second-hand smoke).

Summary of Responses to the Questions Asked By the Task Force

- A combination of policy and science influences the views about environmental hazards held by public health agencies.
- Scientific information about arsenic toxicity and people's exposure to soil indicates that area-wide soil arsenic contamination is a widespread public health hazard (as defined by state law) with potentially serious consequences for some individuals.
- Depending on what it is being compared to, the health risk from area-wide soil arsenic contamination may be described as either large or small.
- Scientific studies attempting to measure the effect of area-wide soil arsenic contamination on public health have been inconclusive. Specifically, available data are not sufficient to rule out, nor confirm, that the contamination is causing health problems.

Selected References

Arsenic Toxicity

National Research Council Subcommittee on Arsenic in Drinking Water. (1999). Arsenic in Drinking Water. National Academy Press, Washington, D.C. State-of-the-art review of information about arsenic toxicity by a group of scientists for the National Research Council, a private, non-profit society of scientists with a mandate by Congress to advise the federal government on scientific and technical matters. Available online at: <http://www.nap.edu/books/0309063337/html/index.html>

National Research Council Subcommittee to Update the 1999 Arsenic in Drinking Water Report. (2001). Arsenic in Drinking Water, 2001 Update. National Academy Press, Washington, D.C. Update to the 1999 report, by a (mostly) different group of scientists, reviewing studies on arsenic toxicity published between 1999 and 2001. Available online at: <http://www.nap.edu/books/0309076293/html>

Soil Ingestion

Scott Davis et al. (1994). Quantitative estimates of soil ingestion in normal children between the ages of 2 and 7 years: population-based estimates using aluminum, silicon, and titanium as soil tracer elements. Archives of Environmental Health, 45(2): 112-122. A study of soil ingestion in 104 children (ages 2 to 7) in Richland, Pasco, and Kennewick, Washington.

Edward J. Calabrese and Edward J. Stanek. (1994). Soil ingestion issues and recommendations. Journal of Environmental Science and Health, A29(3): 517-530. A review of published studies of soil ingestion (as of 1994) and a discussion of factors that might influence soil ingestion rates.

Studies of Orchardists

Paul A. Neal et al. (1941). A study of the effect of lead arsenate exposure on orchardists and consumers of sprayed fruit. U.S. Public Health Service Bulletin 267. No increased health effects found in orchardists.

Washington State Health Services Division. (1975). Cancer mortality in farmers and orchardists in Washington State, 1950 – 1971. E.P.A. Contract 68-02-0559 Annual Report 1975. Increased respiratory cancer found in orchardists.

Kristine Tollestrup et al. (1995). Mortality in a cohort of orchard workers exposed to lead arsenate pesticide spray. Archives of Environmental Health, 50(3): 221-229. Possible increased mortality from heart disease, but not cancer in orchardists.

Endnotes

¹ World Health Organization. 2001. Environmental Health Criteria 224: Arsenic and arsenic compounds. WHO (2001) states that "...[l]ong term exposure to arsenic in drinking water is causally related to increased risks of cancer in the skin, lungs, bladder and kidney, as well as other skin changes such as hyperkeratosis and pigmentation changes. These effects have been demonstrated in many studies using different study designs. Exposure response relationships and high risks have been observed for each of these endpoints. The effects have been most thoroughly studied in Taiwan but there is considerable evidence from studies on populations in other countries as well. Increased risks of lung and bladder cancer and of arsenic-associated skin lesions have been reported to be associated with ingestion of drinking water at concentrations [less than or equal to] 50 ug arsenic/litre." (p. 4)

² Environmental Protection Agency. 2000. Integrated Risk Information System (IRIS).

³ Agency for Toxic Substances and Disease Registry. 2000. Toxicological Profile for Arsenic (Update). Prepared by Syracuse Research Institute under contract to ATSDR. The 1986 amendments to the federal Superfund law directed ATSDR to prepare toxicological profiles for hazardous substances commonly found at hazardous waste sites. These profiles are designed to identify and review key scientific literature describing a substance's toxicological properties. Each profile is peer-reviewed by scientists from the Centers for Disease Control and other Federal agencies and non-governmental scientific review panel. With respect to carcinogenic effects, ATSDR concluded that "...[t]here is clear evidence from studies in humans that exposure to inorganic arsenic may increase the risk of cancer. In workers exposed by the inhalation route, the predominant carcinogenic effect is increased risk of lung cancer (e.g. Enterline et al. 1987a, 1987b; Jarup and Pershagen 1991; Jarup et al. 1989; Lee-Feldstein 1986; Welch et al. 1982), although a few reports have noted increased incidence of tumors at other sites (e.g. Lee-Feldstein 1983; Pinto et al. 1977; Tsuda et al. 1987). Based on the risk of lung cancer, EPA has assigned inorganic arsenic to Group A (known human carcinogen) by the inhalation route (IRIS 2000). This is supported by the U.S. Public Health Service, which has also classified inorganic arsenic as a known human carcinogen (NTP 1994, 2000). In general, most researchers observe that risk increases as a function of exposure level and duration (Axelson et al. 1978; Jarup et al. 1989; Lee-Feldstein 1983; Mabuchi et al. 1979; Pinto et al. 1978). Most cases are seen in workers with chronic exposures, although several studies suggest that even short (1 year) exposures may also increase risk (Lee-Feldstein; Sobel et al. 1988)...When exposure occurs by the oral route, the main carcinogenic effect is increased risk of skin cancer. This conclusion is based on a number of epidemiological studies of populations exposed to elevated levels of arsenic in drinking water (e.g. Chakroborty and Saha 1987; Hauptert et al. 1996; Luchtrath 1983; Tseng 1977; Tseng et al. 1968; Zaldivar 1974) and on numerous case reports of people exposed to Fowler's solution (Bickley and Papa 1989) Piontek et al. 1989) Sommers and McManus 1953). Based on these findings, the EPA has placed inorganic arsenic in Group A (known human carcinogen) for exposure by the oral route." (pp. 176 and 188)

⁴ National Research Council. 1999. Arsenic in Drinking Water. National Academy Press. Washington DC. In 1996, EPA's Office of Water requested that the National Research Council (NRC) independently review the arsenic toxicity data base and evaluate the scientific validity of EPA's 1988 risk assessment for arsenic in drinking water. The NRC convened the Subcommittee on Arsenic in Drinking Water, whose membership includes experts in toxicology, pharmacology, pathology, chemistry, nutrition, medicine, epidemiology, risk assessment and biostatistics. With respect to arsenic toxicity, the subcommittee concluded "...there is sufficient evidence from human epidemiological studies in Taiwan, Chile, and Argentina that chronic ingestion of inorganic arsenic causes bladder and lung cancer, as well as skin cancer..." (p. 2) The Subcommittee's conclusions were based on a thorough review of available human epidemiological studies and animal bioassays and considered studies that both demonstrated an

association between arsenic exposure and health effects and studies that found no association between elevated rates of cancer or other health effects.

⁵ National Research Council. 2001. *Arsenic in Drinking Water: 2001 Update*. National Academy Press. Washington DC. In January 2001, EPA lowered the federal drinking water standard from 50 ug/L to 10 ug/L. However, in March 2001, EPA announced it was delaying the effective date of the new standards pending additional scientific and economic analyses. To ensure that its decision reflected the most current scientific research, EPA requested that the NRC independently review studies on the health effects of arsenic published since the 1999 report. The NRC convened the Subcommittee to Update the 1999 Arsenic in Drinking Water Report. As with the earlier subcommittee, members selected had expertise in toxicology, pharmacology, pathology, chemistry, nutrition, medicine, epidemiology, risk assessment and biostatistics. With respect to cancer risks, the Subcommittee considered four studies that had been published since 1999. They found that three of those studies confirmed the association between lung and bladder cancers seen in earlier studies. They also reviewed a fourth study (conducted in Utah) that did not demonstrate an association between arsenic exposure and increased risks. However, the Subcommittee "...concluded that the limitations of the Utah study currently preclude its use in a quantitative risk assessment..." and that "...the other recent studies of arsenic in humans, taken together with the many studies discussed in the 1999 NRC report, provide a sound and sufficient database showing an association between bladder and lung cancers and chronic exposure in drinking water, and they provide a basis for quantitative risk assessment..." (p.5)

⁶ Environmental Protection Agency. 1997. *Exposure Factors Handbook*. Volumes I, II, III. U.S. Environmental Protection Agency, Office of Research and Development. EPA/600/P-95/002Fa. EPA (1997) states that "...[t]he ingestion of soil is a potential source of exposure to toxicants. The potential for exposure to contaminants via this source is greater for children because they are more likely to ingest more soil than adults as a result of behavioral patterns present during childhood. Inadvertent soil ingestion among children may occur through the mouthing of objects or hands. Mouthing behavior is considered to be a normal phase of childhood development..." EPA reviewed the available scientific literature and identified seven key studies that were used to prepare recommended guidelines for evaluating the amount of soil exposure. The mean values in these studies ranged from 39 mg/day to 271 mg/day with an average of 146 mg/day for soil ingestion and 191 mg/day for soil and dust ingestion. Based on these studies, EPA recommended that risk assessors use a value of 100 mg/day as a mean value, 200 mg/day as a conservative estimate of the mean, and a value of 400 mg/day as an upper bound value. Several researchers have evaluated potential exposures in areas with elevated soil arsenic levels. For example, the University of Washington (Polissar et al. 1990) found elevated urinary arsenic levels (a standard measure of arsenic exposure) were primarily due to hand-to-mouth activity. Hand arsenic levels were significantly correlated with length of time-spent in high- and medium-soil contact activities. In contrast, ATSDR (2000) summarized a German study (Gebel et al. 1998) conducted in a former mining area characterized by high levels of arsenic (2-605 ppm) in residential areas where arsenic levels in hair and urine samples from children in study area were similar to levels in children from a reference area in Germany.

⁷ Most of the available scientific studies have been designed to evaluate inadvertent or unintentional ingestion of soil. However, some children have been observed to deliberately ingest unusually large amounts of soil (pica behavior). Studies conducted in Boston, Baltimore, Cincinnati and Rochester estimated that 9.1 to 40.9 percent of the children in the various studies displayed some type of soil pica behavior (These studies are summarized in Chapter 3 of the *Risk Analysis to Support Standards for Lead in Paint, Dust and Soils* prepared by EPA). EPA has recommended using soil ingestion rates of 5000 to 10000 mg/day when evaluating health risks posed by pica behavior. However, this recommendation is primarily based on one study that observed an intake of 5000 to 8000 grams/day.

⁸ In order to cause an adverse response, arsenic that is ingested must be absorbed into the body. Studies in animals indicate soil bioavailability values ranging from less than 10% to 78 percent. The National Research Council (1999) stated that "...[w]hen ingested in dissolved form, inorganic arsenic is readily absorbed. About 80-90 % of a single dose of arsenite As (III) or arsenate As (V) was absorbed from the gastrointestinal tract of humans and experimental animals (Pomroy et al. 1980; Vahter and Norin 1980; Freeman et al. 1995). A much lower degree of gastrointestinal absorption was reported for arsenic-contaminated soil (Freeman et al. 1995), although the form of arsenic in the soil, as well as the type of soil, can be assumed to influence the degree of arsenic soils." (p. 150). ATSDR (2000) has also reviewed available studies and concluded that "...bioavailability of arsenic from soil is reduced by low solubility and inaccessibility due to the presence of secondary reaction products or insoluble matrix components (Davis et al. 1992). This is supported by studies conducted with in vitro simulations of the gastric and/or intestinal fluids (Hamel et al. 1998; Rodriguez et al. 1999; Ruby et al. 1996, 1999; Williams et al. 1998). When soils containing arsenic are incubated in simulated gastrointestinal fluids, only a fraction of the arsenic becomes soluble. Estimates of the soluble, or bioaccessible, arsenic fraction have ranged from 3 to 50% for various soils and mining and smelter waste materials (Rodriguez et al. 1999; Ruby et al. 1996); these estimates are similar to in vivo estimates of the relative bioavailability of arsenic in these same materials (Ruby et al. 1999)." (p. 136).

⁹ Agency for Toxic Substances and Disease Registry. 2000. Toxicological Profile for Arsenic. ATSDR (2000) reviewed a wide range of peer-reviewed studies and concluded that "...[a]rsenic exposure in communities near mining and smelting facilities or where arsenic has formerly been applied to agricultural land are a public health concern, especially for infants and children. Since arsenic remains in the surface soil indefinitely and long past land uses may be forgotten, people may not realize that they are living in areas where high levels of arsenic may occur in soil. Contaminated soils pose a particular hazard to children because of both hand-to-mouth behavior and intentional ingestion of soil (pica) that contain metals and other contaminants (Hamel et al. 1998). In these communities, arsenic may contaminate carpeting or may have been tracked in from outside. Children may be exposed to this arsenic while crawling around or playing on contaminated carpeting. Exposure may also result from dermal contact with the soil, or by inhaling the dust and swallowing it after mucociliary transport up and out of the lungs. Because much of the arsenic in soil is embedded in or adsorbed to soil particles or insoluble, it may not be in a form accessible for uptake by the body. Environment Canada (1993) estimated arsenic intakes from soil and dirt for infants and children in the general population and those living near point sources (See Table 5-4). Intake from soil and dirt in the exposed populations was 0.03-0.08 ug/kg body weight per day for infants 0-0.5 years old and 0.02-0.05 ug/kg body weight per day for children 0.5-4 years old. For those living near point sources, arsenic intake from soil and dirt reached highs of 3.0 ug/kg body weight per day for infants 0-0.5 years old and 1.9 ug/kg body weight per day for children 0.5-4 years old". (p. 287)

¹⁰ When evaluating the health risks associated with elevated levels of arsenic, most of the qualitative issues (e.g. arsenic's ability to increase cancer risks, soils as a potential source of exposure, etc) are no longer the subjects of serious scientific debate. However, the range of available scientific studies does result in wide ranges on several quantitative issues including (1) how much soil does a children ingest?; (2) how much of the arsenic bound to ingested soils is absorbed into the body (bioavailability); (3) what is the appropriate method for extrapolating results at high levels of exposure to estimate cancer risks at lower levels of exposure.



Lead Poisoning and Washington State's Children: A Condensed Overview

Purpose of this document

The purpose of this document is to provide the Area Wide Soil Contamination Task Force with information regarding the health aspects of lead toxicity and lead exposure through soil and dust. Toxicological and epidemiological staff at the Washington State Department of Health (DOH) have worked to provide answers to specific questions that the Task Force has brought forward at the meetings and during the working group conference calls. The goal has also been to provide more background information and range of perspectives on these issues. Six major questions have been used as the focus of this document. They include:

1. Does lead in soil pose a threat to health?
2. Is there evidence is that children take up lead from soil and dust?
3. Are there any Washington specific lead exposure studies in children?
4. Are there any specific data that demonstrate exposure from lead arsenate pesticides and exposure to humans (farm workers? children?)?
5. Is there evidence that associating exposure to lead in dirt with subsequent adverse neurobehavioral or other adverse health outcomes in children?
6. Do adverse health outcomes in children continue into adulthood?

Introduction

What are sources of lead exposure?

Lead is a naturally occurring metal found in the earth's crust that has no distinguishing taste or smell. Although it occurs naturally, almost all of the high levels found throughout our environment are due to human action. Until leaded gasoline was phased out during the 1980s, gasoline was the single greatest source of lead released into the environment in the United States. Sources of lead released into the air include burning fuel (e.g. oil and coal), industrial processes and burning solid waste. Once airborne, lead may travel hundreds to thousands of kilometers before being deposited on surface soil or in water by rain or airborne particulates. Presently, the release of lead to land from land exceeds that of lead released into the air from land. Today, landfills, municipal and hazardous waste dump sites, distribution of mining wastes, lead-based paints from older homes, windblown soil and soil contaminated with lead from historical automotive exhaust are most responsible for lead transfer into land.

The major route of human exposure to lead is from swallowing (food, drink and large particles), though little lead that is swallowed enters the blood stream. In general, if adults and children eat the same amount of lead, a larger proportion of the amount of lead swallowed will enter the blood of children than of adults. The major target of lead for producing toxic effects in humans is the nervous system. Exposure in workers to lead over a long period of time has resulted in decreased performance in tests that measure how the nervous system functions. Further, weakness in fingers wrists and/or ankles has been observed with such long-term exposure.

Who represents the population of concern?

Children are more susceptible to poisoning from lead than are adults. This is especially the case since children are exposed their whole lives: in the womb, possibly from breast feeding, from food and drink, from drinking water, and by swallowing or breathing lead in dirt, dust or sand. In the infant and toddler years, they have increased exposure to environmental lead through their mouthing and normal sucking behavior. Children are more sensitive to the effects of lead than adults, have higher absorption rates across the intestine and breathe in more air on a per kilogram bodyweight basis. Lead affects children in different ways, depending on how much lead is swallowed. Low levels of exposure to children can affect mental and physical growth.

How big of a problem is childhood lead poisoning?

The good news is that concentrations of lead in blood (often referred to as the blood lead level) of children in the United States are declining. This is mostly due to elimination of lead from gasoline, residential paint and solder used for food cans and water pipes. However, estimates from data obtained in the 1990's indicate that between 200,000 and 650,000 young children in the U.S. are considered to have blood lead levels at or above the level of concern, which is 10 micrograms lead per deciliter blood (10 µg/dL)(1). In addition, recent scientific findings have suggested that this level of concern should be lowered. If the level of concern is lowered, the number of children exceeding the level of concern would significantly increase.

What are the document's objectives?

This overview document presented with references, addresses the issue of low level exposure to children and how low level exposures can lead to serious and irreversible health effects. In addition, as present day blood lead concentrations are many times those of what are considered "normal," from pre-industrial times, we describe why we, as overseers of public health, need be troubled by possible exposures to children from lead-contaminated soil and dust. Further, a discussion of the re-evaluation of the level of concern regarding lead exposure is offered.

The intent of this document is not to provide a complete description of all available data but to indicate that, while there have been epidemiological studies that have not seen notable adverse health effects, the preponderance of scientific evidence during the last decade has provided an increase in our knowledge of the effects of low level lead exposure. The increased information supports our conclusion that public health efforts to reduce exposure to lead from all exposure routes to children are required and that further evaluations of the acceptable level of lead in blood should be conducted at the national level.

Synopsis of Lead Toxicity

To date, no single mechanism of lead toxicity has been described. However, effects on human brain function from exposure to lead are deleterious and irreversible, including effects from low-level exposure. Since the mechanisms of lead toxicity involve many basic biochemical processes, lead affects virtually every organ and system in the body from blood pressure elevations and decreased kidney filtration

processes in adults to adverse neurobehavioral outcomes in children (2). Lead is able to imitate the action of calcium and interacts with various proteins (3-7). These proteins along with calcium play a pivotal role in cell signaling systems. Through imitation of calcium and interaction with other proteins by lead, lead is thought to cause cardiovascular and neurological effects.

The Extent of Childhood Lead Poisoning

Due to restrictions in the United States on certain uses of lead, such as in gasoline and in lead-soldered food cans, the median blood lead level of young children has declined drastically from 15 µg/dL in the 1970s to approximately 2 µg/dL today (1, 8). One of three primary sources of data describing the extent, or the prevalence, of elevated lead concentrations in children in Washington state is “The Statewide Childhood Blood Lead Prevalence Survey” conducted by the Epidemiology Office of the Washington State Department of Health in 1999. These data indicate that approximately 1% of one- and two-year old children in Washington state have blood lead concentrations above 10 µg/dL and 3.5% have blood lead concentrations between 5 and 10 µg/dL. . Also, the results indicate that 3.8% of one- and two-year old Hispanic children in central Washington have blood lead concentrations above 10 µg/dL and 6.7% have concentrations between 5 and 10 µg/dL. Accordingly, several thousand children in Washington state have blood lead concentrations above the national average of approximately 2 µg/dL and above 5 µg/dL. This 5 µg/dL blood lead is taking on increasing significance as further data become available addressing lead’s effects on the cognition of children.

A study conducted by DOH between 1994 and 1997 investigated the prevalence of elevated childhood lead levels in neighborhood areas (according to Census data) of five cities in Washington where lead exposure would be predicted to be higher: individuals living in old housing, low-income residents and children under the age of three. Surveys of children in the cities of Bellingham, Seattle, Spokane, Tacoma and Yakima were conducted, environmental sampling for lead in the homes of eligible children was performed and blood samples for blood lead testing were obtained. The results of these studies suggest a low risk of lead poisoning in most of the state with a higher risk in Yakima than in the other cities (9).

In 1992 a study was conducted in Western Washington investigating blood lead concentrations in children (10). Although the study, which was performed by investigators from the Public Health Seattle and King County, Snohomish Health District and University of Washington was not published, the data provided insight into blood lead concentrations for the children residing in the targeted area; children that were living in neighborhoods with more than 50% of housing constructed prior to 1950 in two urban areas, Seattle and Everett and one rural area, Grey’s Harbor county. The study reported that 4.5% of the 465 children included in the sample had blood lead concentrations above 10 µg/dL and no children had a blood lead level above 20 µg/dL. Sources of lead exposure were thought to be primarily from lead paint in the older housing stock for the urban and rural areas, soil contaminated lead from gasoline emissions for the urban areas and both housing stock and gasoline emissions along with the nearby smelter site for Everett location. In 1992, this 4.5% prevalence rate of individuals with blood lead concentrations above 10 µg/dL appeared to be a very low figure and was 94.9% lower than the nationwide estimate of 87.8%. However, given our understanding of the adverse health effects from lead, this 4.5% figure could be deemed elevated. Presently, it is not known what the current prevalence of elevated blood leads in children in these areas are.

Lead Effects on Pregnancy Outcome

There have been numerous studies during the last several decades that have evaluated the adverse pregnancy outcomes that occur from exposure to lead. In populations who experienced lead exposure from sources such as emissions from automobile and smelters, battery plants, printing works, drinking water from leaded pipes, leaded pottery glaze and lead paint, adverse pregnancy outcomes were seen. Women with blood lead levels and babies born with cord blood levels approximating 15 go/dl were seen to have higher rates of intrauterine growth retardation (IUGR), still births, spontaneous abortions and mental retardation in their babies than those with lower blood lead levels (11-19). However, a recent study in the Czech Republic observed that women with low blood lead levels during pregnancy (average level 3.5ug/dl) did not experience adverse pregnancy outcomes (20).

Lead Effects on the Developing Fetus

The current definition of an elevated blood lead concentration for children is 10 µg/dL or greater and is based on adverse effects associated with prenatal exposure to lead (21). Children with cord blood lead concentrations above 10 µg/dL had significant cognitive delays at age two when compared to those with lower cord blood concentrations (22). Since this observation was made and the 10 µg/dL value was established in 1991 as the blood concentration at which lead poisoning occurs, several prospective studies have indicated that effects from prenatal exposure to lead are attenuated later in childhood (23-25) and that these children have not experienced long-term health effects from prenatal exposures to lead.

Impacts on neuropsychological function, measured most frequently through IQ and other psychometric tests, have been observed in children that were environmentally exposed to lead. Although many studies investigating mother-infant health relationships as well as early childhood development have come under scrutiny for the improper control of confounding variables, a consistent pattern of neuropsychological deficits have been observed among children exposed to elevated lead levels. As a result, the cumulative literature does indicate that cognitive deficits occur in children who are considered to be asymptomatic (without adverse health symptoms) (17, 26-36).

Lead Effects on the Young Child

Serious irreversible effects have been observed with postnatal exposures to lead. Presently, evidence indicates that cognitive function and academic achievement are impaired with blood lead concentrations below 10 µg/dL, and possibly even below 5 µg/dL, achieved through postnatal exposure (37- 40). This evidence has been the impetus for national agencies (i.e., the U.S. Health and Human Services Advisory Committee on Childhood Lead Poisoning Prevention and the National Academy of Sciences), to once again re-address the issue of defining elevated blood lead concentration.

Soil and Dust as Exposure Routes for Lead

Prior to the completion of many of the studies discussed in this document, the US Food and Drug Administration estimated in 1990 that toddlers (2-year olds) received 75% of their total lead exposure from dust, while only 1% of exposure came directly from soil (2). However, children engaging in pica behavior can be exposed to lead amounts from soil 100 to 250 times as great as an average child (41, 42). That is, soil containing 100 µg/g (Note: 100 µg/g = 100 ppm) of lead can result in an average child being directly exposed to 5 µg/day while a pica child can be exposed to amounts of 500 µg/day or greater. (Note: pica is an eating disorder manifested by a craving to ingest non-food items.)

In 1998, the U.S. Environmental Protection Agency addressed the issue of source apportionment with respect to lead in dust and soil (43). Their conclusions were that no single source could be regularly identified as being responsible for elevated lead concentrations in soil at a residence.

From their extensive literature review they indicate that many studies cite more than one source as commonly being responsible for elevated lead levels in soil. As expected, the three primary sources responsible for the elevated levels in soil were lead based paint, point source emitters and leaded gasoline emissions.

Environmental sources of lead were studied in a recently conducted cross sectional (point in time) epidemiology study in which the source of lead was associated with children's blood lead concentrations (44). Dust lead was determined to be the most important source responsible for increases in blood lead concentrations in children. An increase in dust lead loading from background to 200 $\mu\text{g}/\text{ft}^2$ provided for a statistically significant increase in blood lead levels in the children studied. It was concluded that the increased loading from background to 200 $\mu\text{g}/\text{ft}^2$ produced more than a 20% increase in the percentage of children estimated to have blood lead concentrations in excess of 10 $\mu\text{g}/\text{dL}$. Soil lead concentrations also played a significant role in increasing blood lead concentrations above 10 $\mu\text{g}/\text{dL}$. An increase from background soil lead levels to soil levels of 400 $\mu\text{g}/\text{g}$ (Note: 400 $\mu\text{g}/\text{g}$ = 400 ppm), was estimated to produce an increase of 11.6% in the percentage of children estimated to have blood lead concentrations above 10 $\mu\text{g}/\text{dL}$.

The experimental design of this study as described does not readily allow for a determination of percentage increases of children above 5 $\mu\text{g}/\text{dL}$ as it does for those above 10 $\mu\text{g}/\text{dL}$, based on changes in lead levels in environmental sources. However, one can anticipate that soil lead levels of 400 $\mu\text{g}/\text{g}$, which were considered to produce an increase of 11.6% in the percentage of children estimated to have blood lead concentrations above 10 $\mu\text{g}/\text{dL}$, will produce an even greater estimated increase in the percentage of children above 5 $\mu\text{g}/\text{dL}$.

Further results from this study suggest that soil lead levels well below 400 $\mu\text{g}/\text{g}$ (i.e. 50 $\mu\text{g}/\text{g}$) are sufficient to provide for estimated predicted blood lead concentrations above 5 $\mu\text{g}/\text{dL}$. Also, findings indicate that this 50 $\mu\text{g}/\text{g}$ soil concentration produced an increase of nearly 10% in the percentage of children estimated to have blood lead concentrations above 10 $\mu\text{g}/\text{dL}$.

It has been suggested when lead in soil is ingested, it is less bioaccessible and therefore less bioavailable (available to the body). This hypothesis was drawn from a study in which an explanation was sought to help clarify why young children living in a mining community did not have elevated blood lead concentrations (45, 46). This is not the only observation of lower blood lead concentrations in individuals living in what would be otherwise considered a possibly polluted or contaminated areas. A study comparing urban children to those living in a mining area found the urban children to have significantly higher blood lead concentrations than the children in the mining area (24). This observation was made despite the fact that there were higher concentrations of lead in soil in the mining area. Also, in a newly completed pilot study of pregnant women in an industrial and mining region with high levels of air pollution, maternal blood lead concentrations were not found to be elevated (20).

The preponderance of evidence however still suggests that soil and dust from soil represent an important pathway of human lead exposure (48). Lead levels of indoor dust and outdoor soil were shown to be strongly predictive of blood lead concentrations in a study of more than 200 urban and suburban infants followed from birth to 2 years of age (49). Further, in a recent study

involving children at day care centers, outdoor lead dust was found to be a more potent contaminant of children's hands than indoor lead dust (50, 51).

Remediation of Lead in Soil and Reduction in Blood Lead Levels

The US Environmental Protection Agency completed a study in the mid-90's to determine whether abatement of lead in soil could reduce blood lead levels of inner-city children (52). Their results suggested that when soil is a significant source of lead in a child's environment, abatement of lead in soil could result in reduced lead exposure and accordingly, blood lead concentrations. Although the study was criticized and the data re-analyzed with no statistically significant effect seen from remediation, results indicated that a number of factors are important in reducing exposure to lead through soil remediation (52, 53). These include, but are not limited to, the magnitude of remediation, magnitude of additional sources of lead exposure and the site-specific exposure scenario.

A more recent study involving the impacts of remediation was conducted in Idaho with children living near the Bunker Hill mining and lead smelter site in Silver Valley Idaho. This study found that removal of lead contaminated soil from residential yards was effective in reducing blood lead levels. The authors of this study note that soil remediation at this site might have been more effective in lowering blood lead concentrations in children because the soil lead levels were much higher than soil lead levels at other sites (54).

Additional Factors Associated with Elevated Blood Lead

Other factors that predispose children to lead include education status of the parents, income of the parents, race, geography and the time of exposure. Racial differences in urban children have been also been investigated. In a study conducted in Rochester, New York, a major contributor to blood lead concentration in urban black children was deemed to be interior lead exposure while in urban white children it was exterior lead exposure (55). This recent observation was made as an outgrowth of the fact that black children in the United States are much more likely to have elevated blood lead concentrations than white children. Differences in housing conditions and exposures to lead-contaminated house dust may contribute significantly to a racial disparity. In addition, the black children studied were more likely to use a bottle and put their mouths on window sills (which in poorer housing conditions would be a significant problem) while white children were more likely to put soil in their mouths and suck on their fingers.

Blood Lead as a Marker of Lead Exposure

Presently, children are screened or targeted for elevated exposure to lead by examination of blood lead concentrations. Unfortunately, this biomarker reflects only recent exposure from the past few months and not low-level exposure over prolonged periods of time (56, 57). Some studies have collected repeated blood samples over time so as to better identify cumulative lead exposure (58). Considering that physical growth is a time-integrated outcome, that the skeleton is the primary site of storage for approximately 95% of lead in the adult human body, and that bone lead has a half-life of years to decades, using the dentin of deciduous teeth or bone lead as a metric of exposure may provide greater insight into the association between exposure and effect (49). This is further supported by the fact that nearly all of lead in a whole blood specimen is bound to red cells; lead that is available to cross the placenta comes from lead in the free state which is found in the plasma (20, 59). Evidence suggests that plasma lead has a positive correlation with bone lead values; lead is mobilized from the bone into plasma (20, 59). As a result, target screening of children at high risk or universally screening in certain areas through the use of blood lead measurements as a biomarker may not include cumulative or chronic exposure. Chronic and

cumulative exposure may be better addressed through tooth lead or bone lead measurements. However, both measurements are costly and time consuming. For population surveillance studies, blood lead remains the main marker for identifying elevated blood leads in the populations, measuring trends in an individual or populations of exposed individuals and for developing public health action strategies.

Presently, sufficient concern warrants that the 10 µg/dL blood lead concentration be re-examined at the national level. Today's average blood lead concentrations should not be interpreted as physiologically "normal" levels. There is but a very small "safety margin", if any, between present day exposures that elicit a toxicological response and those exposure levels that can be considered tolerable. Estimated levels of lead concentrations in blood that are considered "normal" and are thus from the pre-industrial era, range from as low as 0.016 µg/dL up to 0.06 to 0.12 µg/dL (60). Present day national averages are 15 to 125 times higher than those considered "normal" levels. Further, to properly protect children from environmental exposures to lead, it is imperative that we determine empirical estimates of the contributions of the various environmental lead sources to children's intake. Available data indicate that lead contaminated dust is a major factor associated with elevated blood lead concentrations. Direct contact with soil or soil ingestion also plays a role, yet elevated surface soil concentrations leading to increased dust levels may play an even greater role in producing elevated blood lead concentrations.

As stated, the preponderance of data strengthen the contention that dust and also soil sources can be health hazards. These data can aid in establishing remediation standards and controls to prevent children's exposure to these environmental sources of lead. The majority of scientific evidence indicates that greater effort needs to be provided to identify and reduce exposure to environmental sources of lead, with elimination of exposure to lead as the goal.

While, there are no known studies addressing populations typical to Washington and exposure sources such as lead arsenate, using the practice of public health prevention to avoid having Washington's children exposure to a potent toxic substance that causes irreversible effects seems a reasonable approach. The Centers for Disease Control and Prevention, Environmental Protection Agency, Agency for Toxic Substances and Disease Registry have all devoted resources to reducing lead in the environment and educating the public and health practitioners about the health hazards of environmental lead exposure.

The available data, indicating that lead exposure is cumulative and its detrimental effects irreversible, are a challenge for present day public health protection approaches. In 1997, CDC determined that universal screening was not necessary for states that believed that they had a lower prevalence of childhood lead poisoning than the national average because of variations in populations and exposure sources. The new recommendations called for each state to engage an advisory group to develop lead screening guidelines. (61). The advisory group in Washington consisted of a broad based group of health care practitioners, community groups, parents and public health agencies. In Washington, as well as elsewhere, guidelines are focused on secondary prevention: targeted screening of high risk children for elevated blood lead concentrations, followed by controlling environmental sources through management of children with the elevated blood lead concentrations.

The goal of emphasizing primary prevention by preventing exposure to lead is an important and reachable one as evidence is available suggesting that low-level lead exposure may lead to irreversible effects on brain function such as lowered intelligence and diminished school performance. Environmental lead sources that cause overexposure to children require identification, intervention and remediation. Public health protection can be improved by advocating for the minimization of exposure to children so as to avoid any association, perceived or otherwise, that children represent sentinels of overexposure and provide evidence that lead contamination exists.

Summary

Responding to the original six questions that were the focus of this work:

1. Does lead in soil and dust pose a threat to health?
Yes, it can although it is difficult to separate exposure to lead from lead in paint as compared to lead in soil (such as lead arsenate contaminated soils).
2. Is there evidence is that children take up lead from soil and dust?
Yes, children have lead exposure from lead in soil and dust in and around where they live.
3. Are there any Washington specific lead exposure studies in children?
Beyond the three studies related to the blood lead program conducted by the Office of Epidemiology within DOH, there are no studies specifically aimed at lead exposure. There was a study that looked at the amount of soil that was ingested by children. This was done in the Tri-Cities area and suggested possibilities for additional research initiatives. It has been omitted from this review as it addressed soil ingestion only.
4. Are there any specific data that demonstrate exposure from lead arsenate pesticides and exposure to humans (farm workers? children?)?
An exposure study of lead arsenate has not been conducted. A study in the Wenatchee area evaluated the mortality (death) experience of adult farm workers exposed to lead arsenate spray and found only increased mortality ratios for heart disease in male intermediates. As this was not an exposure study, and thus has been omitted from this review. There is a study on occupational dermatoses (skin disorders) due to exposure to products used in agriculture. This was a case study of lead-arsenate toxicity in eight children published in 1981. The children inhaled and ingested 95% lead-arsenate pesticide; as this is an acute (short term or immediate exposure) case, it has been omitted from this review.
5. Is there evidence associating exposure to lead in dirt with subsequent adverse neurobehavioral or other adverse health outcomes in children?
From soil alone, no. There are data available in which soil lead levels are discussed as part of the total exposure leading to elevated blood lead concentrations. Some of these data are discussed. The evidence that has lead to many of the standards of public health practice are based on total exposure to lead, not just one source of lead.
6. Do adverse health outcomes in children continue into adulthood?
Yes, this is a major reason for why the National Academy of Science is being asked to re-evaluate the acceptable lead concentration.

In summary, it is hoped that this information has provided an increased understanding of the issues that the Task Force has to address. To conclude, this document has provided evidence that:

- Effects from low-level lead exposures are serious and irreversible,
- Present day childhood blood lead concentrations are many times those of what are considered “normal,” and childhood blood lead concentrations are elevated in certain areas of Washington state,
- Lead contaminated dust and soil (or dust from soil) can be a health threat.
- The 10 µg/dL blood lead concentration that is presently considered a “safe level” is being re-evaluated.

Included are a few of the relevant and applicable articles used to develop this short overview. Following this document you will find copies of several scientific (peer reviewed) articles that have been used in this document and that demonstrate the range of scientific findings. Also included are editorials from journals, newsletters or newspapers that are not peer reviewed. These represent opinions of individuals in the community at large and were not discussed in this overview. Also included are web sites where more information about lead exposure and toxicity can be found. If you need assistance in finding these materials – please contact DOH at 360-236-3192.

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WASHINGTON STATE CHILDHOOD BLOOD LEAD SCREENING RECOMMENDATIONS

November 2000



Washington State Childhood Blood Lead Level Screening Recommendations

November 2000



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Executive Summary

In 1997, the Centers for Disease Control and Prevention (CDC) withdrew their former recommendation for universal childhood lead screening and recommended that each state develop its own screening guidelines. In 1999, the Washington State Department of Health (DOH) formed an advisory committee to recommend screening guidelines for Washington State. The advisory committee included health care providers, representatives from the state and local health departments, Medicaid, managed care and private insurance organizations, and community action groups.

Only about 3 percent of children in Washington State ever have a blood lead test. Data collected by DOH indicate that the prevalence of elevated blood lead levels is very low among children in Washington State. The Childhood Blood Lead Registry has maintained a record of all blood lead test results on Washington children since May 1993. During this time, about 4 percent of the children tested have had an elevated blood lead level. Because the few children who are tested are probably selected for testing by their health care providers because they have risk factors that other children do not have, they are not necessarily a representative sample of children in Washington State. Between 1994 and 1997, DOH conducted residential lead surveys in Bellingham, Seattle, Spokane, Tacoma, and Yakima. These surveys were targeted to neighborhoods with high concentrations of old housing and poverty, where children were thought to be at high risk of lead poisoning. A high prevalence of elevated blood lead levels was found in Yakima (8.4 percent), but the prevalence of elevated blood lead levels in the other four cities combined was only 1.2 percent. In 1999, DOH conducted a representative statewide survey to determine the prevalence of elevated blood lead levels in one- and two-year old children. The survey also oversampled Hispanic children in Central Washington and was able to determine the prevalence of elevated blood lead levels among these children. The survey estimated the prevalence of elevated blood lead levels to be 0.9 percent (95 percent confidence interval [CI], 0—1.9 percent) among all one- and two-year old children, and 3.8 percent (95 percent CI, 0—7.8 percent) among one- and two-year old Hispanic children in Central Washington.

The advisory committee considered these data before making recommendations to DOH. Based on these recommendations, DOH has produced lead screening guidelines for health care providers in Washington State and produced a set of goals for monitoring lead levels in Washington children.

In summary, DOH does not recommend either universal or targeted screening of children for lead poisoning. DOH does not recommend the use of a risk factor questionnaire to select children for lead testing, because no risk factor questions have been shown to be effective for identifying asymptomatic children for whom blood lead testing is appropriate. Health care providers should continue to use clinical judgment to identify children who should be tested. DOH should continue to monitor blood lead levels in children statewide and should conduct studies in Central Washington to determine whether guidelines for targeted screening can be developed there.

Introduction

In 1991, the Centers for Disease Control and Prevention (CDC) called for universal blood lead screening among children age one- to six-years old in the United States. This recommendation was never fully implemented in many parts of the country, including Washington State, where only about 3 percent of children ever receive blood lead tests. Since 1991, the prevalence of elevated blood lead levels (EBLLs)¹ among children in the United States has declined substantially and the CDC has also recognized that the prevalence of EBLLs varies substantially across the different regions of the United States. In 1997, the CDC produced new recommendations² that call for each state to develop screening guidelines appropriate for the state.

The CDC recommendations provide guidance as to how each state should develop screening guidelines and the process of developing the guidelines is tied to CDC grants for surveillance and prevention of childhood lead poisoning. The recommendations call for each state to form an advisory committee consisting of health care providers and representatives from local health departments, managed care organizations, Medicaid, private insurance organizations, community groups, and concerned parents. A list of Washington State's advisory committee members is included in Appendix I. We attempted to recruit a parent of a lead-poisoned child, and representatives from environmental activist and Native American tribal organizations, but we were unsuccessful.

The committee was charged with deciding appropriate screening for different areas of the state. When a state has reliable data on the prevalence of childhood lead poisoning, the CDC recommends universal screening (blood lead tests for all children) in areas where the prevalence of lead poisoning is 12 percent or higher, targeted screening (blood testing after risk assessment with a risk factor questionnaire) in areas where the prevalence of lead poisoning is between 3 percent and 12 percent, and no routine screening in areas where the prevalence is below 3 percent. In the absence of routine screening, states should use other methods to monitor blood lead levels, such as periodic focused surveys and routine review of laboratory reports of blood lead level tests.

¹ For children, a blood lead level of 10µg/dL or higher is considered elevated.

² Centers for Disease Control and Prevention. *Screening young children for lead poisoning: guidance for state and local public health officials*. Atlanta: CDC, 1997.

Childhood Lead Poisoning Prevalence in Washington State

There are three primary sources of data concerning the prevalence of childhood lead poisoning in Washington State: The Childhood Blood Lead Registry (CBLR); the 1999 statewide Childhood Lead Prevalence Survey; and, the Five Cities surveys conducted between 1994 and 1997.

The Childhood Blood Lead Registry

The CBLR maintains a record of all blood lead tests performed on Washington children since May 1993. The percentage of children tested in Washington is low (about 3 percent), and we do not know how health care providers decide which children to test. Therefore, the registry data may not be representative of all children in Washington, but it does help to illustrate regional differences and trends over time. Table 1 shows the percentage of tested children who had elevated lead levels for each year from 1993 to 1998. The percentage of tested children who were found to have elevated lead levels is higher than was found in the statewide survey (0.9 percent in the survey versus 2.8 percent in the registry in 1998), suggesting that health care providers selectively test children who are at higher than average risk. The test results shown in Table 1 exclude tests reported from Madigan Army Medical Center, where all tests on military dependents in Washington State are done, because the military's screening practices are so different from that of other health care providers in the state. The military tests all children in their system when they are one year old and performs about one third of the tests done on children in Washington State. Test results performed at Madigan are shown separately in Table 2.

The registry allows us to look at regional differences in prevalence of elevated blood lead levels (see Table 3). The data suggest that children in Central Washington are at higher risk than children in other parts of the state.

Table 1. Children age 0 to 6, percent elevated by year.

Year	Number of children	Percent above 10 μ g/dL
1993	837	4.8
1994	1481	4.9
1995	1653	8.3
1996	3190	2.9
1997	2479	3.7
1998	2247	2.8
1993-1998	11887	4.2

excluding tests from Madigan Army Medical Center

Table 2. Madigan children age 0 to 6, percent elevated by year.

Year	Number of children	Percent above 10µg/dL
1994	1318	0.5
1995	1668	0.4
1996	1216	0.4
1997	1235	0.2
1998	1014	0.3
1994-1998	6453	0.4

Table 3. Percent of children with elevated blood lead levels by county, 1993—1998.

County	Number of children	Percent above 10µg/dL
Walla Walla	325	10.8
Chelan	1272	9.1
Yakima	1246	5.3
Kitsap	380	5.0
Pierce	740	4.7
Adams	72	4.2
King	1991	4.0
Lewis	52	3.8
Okanogan	54	3.7
Spokane	1567	3.4
Whatcom	251	2.8
Snohomish	428	2.8
Thurston	427	2.6
Unknown county	170	2.4
Skagit	141	2.1
Cowlitz	890	1.9
Island	58	1.7
Clark	120	1.7
Whitman	183	1.6
Grant	127	1.6
Benton	814	1.0
Franklin	369	0.8
Stevens	51	0.0
State Total	11887	4.2

excluding tests from Madigan Army Medical Center

The Five Cities Studies

The Environmental Health Division of the Department of Health conducted targeted residential surveys in Bellingham, Seattle, Spokane, Tacoma, and Yakima between 1994 and 1997. These surveys were targeted towards neighborhoods that had, according to Census data, old housing, low-income residents, and a high proportion of children under the age of three. The surveys included environmental sampling in the homes of eligible children, as well as blood lead testing. The overall prevalence of elevated blood lead tests is shown in Table 4. These studies suggest a low risk of lead poisoning in most of the state, with a higher risk in Yakima than in the other cities.

The Statewide Childhood Blood Lead Prevalence Survey

The Epidemiology Office of the Department of Health conducted a statewide survey in 1999 to estimate the prevalence of elevated blood lead levels in one- and two-year old children. The survey was designed to be representative of all one- and two-year old children in the state, and of one- and two-year old Hispanic children in nine counties in Central Washington (Adams, Benton, Chelan, Douglas, Franklin, Grant, Okanogan, Walla Walla, and Yakima). From the survey results, we estimate that 0.9 percent (95 percent CI, 0–1.9 percent) of one- and two-year old children in Washington have elevated blood lead levels, and that 3.8 percent (95 percent CI 0–7.8 percent) of one- and two-year old Hispanic children in Central Washington have elevated blood lead levels.

The survey results are consistent with the results from the CBLR and the Five Cities studies in suggesting a low prevalence of elevated blood lead levels in most of the state, with a somewhat higher prevalence in Central Washington.

Table 4. Blood lead test results from the Five Cities surveys.

City	Number of children	Number above 10µg/dL	Percent above 10µg/dL
Bellingham	126	1	0.8
Seattle	109	0	0
Spokane	86	0	0
Tacoma	106	4	3.9
Yakima	154	13	8.4
All five cities	581	18	3.1

Department of Health Recommendations

The following recommendations to health care providers and DOH are based on the recommendations of the advisory committee to DOH. The full text of the advisory committee recommendations is in Appendix II. The recommendations were developed after the advisory committee considered the prevalence data for Washington State.

Recommendations to Health Care Providers

1. Because of the low prevalence of elevated blood lead levels in children in Washington State, the Washington State Department of Health (DOH) does not recommend targeted or universal testing of asymptomatic children for lead poisoning. DOH does not recommend the use of a risk factor questionnaire to identify children who should have a blood lead test because no risk factor questions have been shown to be effective in Washington State for identifying asymptomatic children for whom blood lead testing is appropriate. DOH has found a higher risk for elevated blood lead levels in children in nine counties in Central and Eastern Washington (Adams, Benton, Chelan, Douglas, Franklin, Grant, Okanogan, Walla Walla, and Yakima), and DOH will conduct additional study in these counties to determine whether guidelines for targeted screening can be developed.
2. Health care providers should use clinical judgment to identify children who should be tested for blood lead levels. A blood lead test should be performed whenever a parent, guardian, or health care provider suspects that a child is at special risk for lead exposure or if a health care provider finds signs or symptoms consistent with lead overexposure (e.g., anemia, failure to thrive). Additional risk factors health care providers should consider include:
 - Age of housing
 - Renovation and remodeling in old homes
 - Parental occupations involving lead exposure
 - Children observed eating paint chips or showing symptoms of pica
 - Socioeconomic and educational status
 - Former residence outside Washington State

Recommendations to the Department of Health

1. DOH should conduct these activities to monitor childhood blood lead levels in Washington State:
 - Conduct periodic focused surveys to monitor or investigate suspected pockets of lead exposure
 - Routinely review blood lead level data from the Childhood Blood Lead Registry
 - Issue public health alerts about newly identified sources of lead exposure
2. In the nine counties in Central and Eastern Washington (Adams, Benton, Chelan, Douglas, Franklin, Grant, Okanogan, Walla Walla, and Yakima) where children are at higher risk for elevated blood lead levels, DOH should conduct additional studies to determine whether guidelines for targeted screening can be developed.

Appendix I

Advisory Committee on Childhood Lead Screening

Nancy Goodyear, PhD	University of Washington (representing the Clinical Laboratory Advisory Council)
Art Gordon	Urban League of Metropolitan Seattle
Beverly Green, MD	Group Health Cooperative of Puget Sound, Committee on Prevention
Joel Kaufman, MD, MPH	University of Washington (representing University of Washington School of Public Health & Community Medicine)
Teresa Marx	Head Start Program (representing Washington State Migrant Council)
Kimble McClung	Quality Improvement and Utilization Management (representing Community Health Plan of Washington)
William O. Robertson, MD	Washington Poison Center (also representing the Washington Chapter of the American Academy of Pediatrics)
Tina Saxton	Tacoma Urban League
Pat Wells	Spokane Regional Health District (representing Washington State Association of Local Public Health Officials)
Margaret Wilson, ARNP	Washington State Department of Social and Health Services, Family Services Section
Sharon Drozdowsky	Washington State Department of Labor and Industries, Occupational Lead Exposure Registry
Maxine Hayes, MD	State Health Officer, Washington State Department of Health
Juliet VanEenwyk, PhD, MS	State Epidemiologist, Washington State Department of Health
Lewey Kittle	Washington State Department of Health, Environmental Health Programs
Eric Ossiander (staff)	Washington State Department of Health
Marcia Mueller (staff)	Washington State Department of Health

Appendix II

Full text of the recommendations from the Advisory Committee on Childhood Lead Screening to the Washington State Department of Health:

1. Because of the low prevalence of elevated blood lead levels in children in Washington State, the Washington State Department of Health (DOH) does not recommend universal blood lead testing of children for lead poisoning. DOH also does not recommend the use of a risk factor questionnaire to identify children who should have a blood lead test. No risk factor questions have been shown to be effective in Washington State for identifying asymptomatic children for whom blood lead testing is appropriate.
2. DOH does not recommend targeted or universal testing of asymptomatic children for blood lead levels in most of the state, consisting of these 30 counties: Asotin, Clallam, Clark, Columbia, Cowlitz, Ferry, Garfield, Grays Harbor, Island, Jefferson, King, Kitsap, Kittitas, Klickitat, Lewis, Lincoln, Mason, Pacific, Pend Oreille, Pierce, San Juan, Skagit, Skamania, Snohomish, Spokane, Stevens, Thurston, Wahkiakum, Whatcom, and Whitman. However, in these 30 counties the following actions are appropriate:
 - DOH should conduct periodic focused surveys to monitor or investigate suspected pockets of lead exposure.
 - DOH should routinely review blood lead level data from the Childhood Blood Lead Registry.
 - DOH should issue public health alerts about newly identified sources of lead exposure.
3. DOH has found a higher risk for elevated blood lead levels in children in nine counties in Central and Eastern Washington (Adams, Benton, Chelan, Douglas, Franklin, Grant, Okanogan, Walla Walla, and Yakima). At the present time, DOH does not recommend targeted or universal testing of asymptomatic children for blood lead levels in these nine counties, however, additional study in these counties is warranted to determine whether guidelines for targeted screening can be developed. A blood lead test should be performed whenever a parent, guardian, or health care provider suspects that a child is at special risk for lead exposure, or if a health care provider finds signs or symptoms consistent with lead overexposure.
4. Health care providers should use clinical judgment to identify children who should be tested for blood lead levels. A blood lead test should be performed whenever a parent, guardian, or health care provider suspects that a child is at special risk for lead exposure, or if a health care provider finds signs or symptoms consistent with lead overexposure (e.g. anemia, failure to thrive). Additional risk factors health care providers should consider include:
 - Age of housing
 - Renovation and remodeling in old homes
 - Parental occupations involving lead exposure
 - Children observed eating paint chips or showing symptoms of pica.
 - Socioeconomic and educational status