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Case Studies in Environmental Medicine

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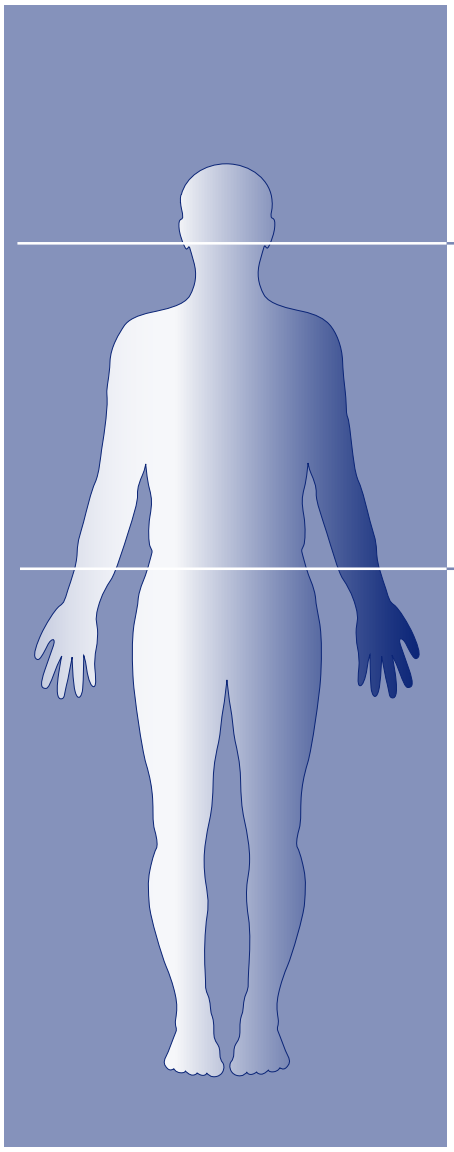

PEDIATRIC ENVIRONMENTAL HEALTH

Environmental Alert

- Childhood is a time of rapid growth and development, accompanied by changes in organ system functioning, metabolic capabilities, physical size, and behavior that can dramatically modify potential illness caused by a toxicant.
- Pediatricians and other child health care providers need to develop the expertise necessary to take an environmental history, deliver anticipatory guidance, and conduct appropriate risk-based laboratory tests for environmental illnesses.
- Pediatric Environmental Health Specialty Units are available for consultation and referral. Pediatricians and other child heatlh care providers should be aware of this resource.

This monograph is one in a series of self-instructional publications designed to increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients. This course is also available on the ATSDR Web site, www.atsdr.cdc.gov/HEC/CSEM/. See page 3 for more information about continuing medical education credits, continuing nursing education units, continuing education units, and continuing health education specialist credits.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Toxicology and Environmental Medicine

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Disclaimer

The state of knowledge regarding the treatment of patients potentially exposed to hazardous substances in the environment is constantly evolving and is often uncertain. In this monograph, ATSDR has made diligent effort to ensure the accuracy and currency of the information presented, but makes no claim that the document comprehensively addresses all possible situations related pediatrics and environmental health. This monograph is intended as a resource for pediatricians and other child health care providers in assessing the condition and managing the treatment of patients potentially exposed to hazardous substances. It is not, however, a substitute for the professional judgment of a health care provider. The document must be interpreted in light of specific information regarding the patient and in conjunction with other sources of authority.

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Each content expert for this case study indicated no conflict of interest to disclose with the case study subject matter.

Final responsibility for the contents and views expressed in this monograph resides with ATSDR.

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Case Studies in Environmental Medicine (CSEM): Pediatric Environmental Health

Goals and Objectives

The goals of this CSEM are to increase the knowledge of health care providers, especially pediatricians, of the special susceptibilities of children to hazardous substances in the environment and to aid in their evaluation of potentially exposed patients.

After completion of this educational activity, the reader should be able to describe how and why children differ from adults in their susceptibility to environmental hazards, apply the knowledge of environmental medicine in the evaluation of well and sick children, identify parental occupation and hobbies as a part of the environmental history, and identify additional sources of environmental health information.

Accreditation

Continuing Medical Education (CME)

The Centers for Disease Control and Prevention (CDC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. CDC designates this educational activity for a maximum of 2.0 hours in category 1 credit toward the American Medical Association (AMA) Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

Continuing Nursing Education (CNE)

This activity for 2.3 contact hours is provided by CDC, which is accredited as a provider of continuing education in nursing by the American Nurses Credentialing Center's Commission on Accreditation.

Continuing Education Units (CEU)

CDC has been approved as an Authorized Provider of continuing education and training programs by the International Association for Continuing Education and Training and awards 0.2 continuing education units (CEUs).

Continuing Health Education Specialist (CHES)

CDC is a designated provider of continuing education contact hours (CECH) in health education by the National Commission for Health Education Credentialing, Inc. This program is a designated event for the CHES to receive 2.0 category 1 contact hours in health education.

Instructions

See page 4

The questionnaire and posttest must be completed and returned electronically, by fax, or by mail for eligibility to receive continuing education credit.

Instructions for Completing CSEM Online

1. Read this CSEM, *Pediatric Environmental Health*; all answers are in the text.
2. Link to the MMWR/ATSDR Continuing Education General Information page (www.cdc.gov/atsdr/index.html).
3. Once you access this page, select the Continuing Education Opportunities link.
4. Once you access the MMWR/ATSDR site online system, select the electronic file and/or register and test for a particular ATSDR course.
   a. Under the heading "Register and Take Exam," click on the test type desired.
   b. If you have registered in this system before, please use the same login and password. This will ensure an accurate transcript.
   c. If you have not previously registered in this system, please provide the registration information requested. This allows accurate tracking for credit purposes. Please review the CDC Privacy Notice (www.cdc.gov/privacy.htm).
   d. Once you have logged in/registered, select the test and take the posttest.
5. Answer the questions presented. To receive continuing education credit, you must answer all of the questions. Some questions have more than one answer. Questions with more than one answer will instruct you to "indicate all that are true."
6. Complete the course evaluation and posttest no later than **July 30, 2008**.
7. You will be able to immediately print your continuing education certificate from your personal transcript.

Instructions for Completing CSEM on Paper

1. Read this CSEM, *Pediatric Environmental Health*; all answers are in the text.
2. Complete the evaluation questionnaire and posttest, including your name, mailing address, phone number, and e-mail address, if available.
3. Circle your answers to the questions. To receive your continuing education credit, you must answer all of the questions.
4. Sign and date the posttest.
5. Return the evaluation questionnaire and posttest, no later than **July 1, 2008,** to CDC by mail or fax:

   Mail
   Continuing Education Coordinator
   Division of Toxicology and
   Environmental Medicine, ATSDR
   4770 Buford Hwy, NE (Mail Stop F-32)
   Atlanta, GA 30341-3717

   or

   Fax
   770-488-4178
   ATTN: Continuing Education Coordinator

6. You will receive an award certificate within 90 days of submitting your credit forms. No fees are charged for participating in this continuing education activity.

Case Study

A 2½-year-old boy has progressive anorexia, weight loss, and a severe, itchy rash

A mother brings her 2½-year-old son to you for consultation. She explains that her family moved to your community about 7 months ago when her husband changed jobs. Over the past month and a half, the boy has developed progressive anorexia and weight loss. He has also suffered from an increasingly severe and itchy rash. Although normally very active and pleasant, he has become ill-tempered and, for the past couple of days, he refuses to walk around, preferring to lie in bed or be carried. He rubs his knees and cries periodically throughout the day. Neither the parents nor the child's grandmother, who lives with them, has been ill.

The boy's medical history has been unremarkable. His birth was at full-term by a normal spontaneous vaginal delivery without complications. His height and weight have been consistently in the 25th percentile for his age. He is on a regular toddler diet, and all developmental milestones have been appropriately met. His immunizations are up-to-date. He is not taking any medications. He had been taking a multivitamin with iron at the correct dosage as prescribed by his doctor. The mother denies any other family use of dietary supplements or herbal medicines. The family history is negative for blood transfusions and use of illicit drugs, human immunodeficiency virus (HIV) infection, and metabolic or genetic diseases. A review of systems and a brief assessment of how the family functions are noncontributory. No one in the family has been traveling in a foreign country.

Physical examination reveals an irritable, pale child with photophobia. His height is 35½ inches (90.17 centimeters [cm]) and his weight is 27.7 pounds (12.6 kilograms), both of which are in the 25th percentile for his age. (His mother remembers that he weighed 30 pounds the last time he was checked by his pediatrician, just before they relocated.) The child's head circumference is 15.2 inches (38.6 cm), also in the 25th percentile. His temperature is 98.3°F (36.8°C), blood pressure is 125/75 mmHg (90th percentile for his age is 105/69), heart rate is 96 breaths/min, and respiratory rate is 30 breaths/min. His skin and mucous membranes are dry. His trunk and face have an erythematous papulovesicular rash with signs of excoriation, but no petechia. His neck is supple without enlarged nodes, masses, or thyromegaly. No other adenopathy is noted. Head, eyes, ears, nose, and throat (HEENT) are within normal limits. Lungs are clear to auscultation. Heart rate is regular without murmurs. His abdomen is soft and is not distended or tender to palpation. No hepatosplenomegaly is noted. His joints have full range of motion and no signs of inflammation. His hands and feet are pink, sweaty, and scaling. Neurologic examination reveals a tongue tremor, diffuse muscle weakness, and unsteady gait, but no focal abnormalities.

During the day, the child stays at home with his mother or grandmother. The mother works part-time as a bookkeeper-clerk in a local dry cleaning

The Figure and Tables for this case study can be found in Appendix A (begins on p. 51).

Pretest

(a) What additional information should you gather by interview?

(b) What would you include in this patient's problem list?

(c) What is the differential diagnosis for this patient?

(d) What baseline laboratory tests would you order to support your differential diagnosis at this point?

(e) What laboratory test would you order to confirm your diagnosis?

facility. The father works as a production manager in a mercury thermometer factory. The mother states that both parents are concerned about environmental contaminants, specifically those that might be associated with their workplaces, and whether or not these contaminants can put their family at risk. The parents have heard neighbors' and co-workers' comments about ailments associated with mercury exposures. The parents also mention recent reports of a group of teenagers in the community taking elemental mercury over the past several months from the local junior high school chemistry lab and the resulting ongoing investigation by the environmental division of the state health department. One of the teenagers who reportedly took mercury from the lab helped with odd jobs around the parents' house, including indoor house-cleaning. The mother expresses the family's concern and asks for your help.

On further questioning, you learn that the family lives in a converted loft apartment in a building that was once part of a jewelry factory complex. No additional remodeling or interior painting has occurred since the conversion 2 years ago. Drinking water is supplied by the city. Each apartment has its own natural gas heating system. The bedrooms and living room are carpeted. The family does not have a garden, but the child often plays at a park and playground within walking distance. The family has no pets. The parents have no more information regarding the teenager, although they have been trying to contact her since the report came out this past week regarding the ongoing investigation conducted by the environmental division of the state heath department.

The child is hospitalized for further evaluation and workup. Baseline laboratory tests include

- white blood cell count with differential;
- blood smear;
- electrolytes, with blood urea nitrogen and creatinine;
- urinary mercury and blood lead levels;
- erythrocyte sedimentation rate, antinuclear antibody, antistreptolysin-O titer;
- urine analysis with specific gravity;
- radiograph of the chest, knees, and bilateral hips;
- computed tomography scan of the brain (to rule out degenerative changes or space-occupying lesion); and
- a spinal tap (after risk for herniation has been excluded).

Of these laboratory tests, only the urine mercury is elevated.

Direct Biologic Indicators

A 24-hour urine collection is obtained from the child and tested. It reveals a total mercury level of 321 micrograms per gram (µg/g) creatinine. Urine specimens are collected from the parents and grandmother and tested. The test results are expressed as urine mercury per gram creatinine, and are as follows: father, 18 µg/g creatinine; mother, 12 µg/g creatinine; and grandmother, 37 µg/g creatinine.

The World Health Organization's *upper limit of normal* for adults is 50 µg/g creatinine (World Health Organization 1991). The upper limit of normal for *unexposed adults* is 20 micrograms per liter (µg/L); most unexposed individuals have levels <5 µg/L (ATSDR 1999). However, the respective levels for children have not yet been established. More than likely, however, the upper limits are lower than those for adults. Urine mercury levels might be reported in different units of measure (e.g., micrograms per gram creatinine and micrograms per liter). A number of issues must be considered when interpreting results in children. These issues are discussed in more detail in Appendix B (p. 69).

Some pediatric experts would regard a urinary mercury level >50 µg/g creatinine as in the toxic range and blood mercury >7–10 µg/dL as elevated. More information on acceptable urine and blood mercury levels in children can be found in Paulson (2001). However, the diagnosis of mercury poisoning should never be based solely on the results of a blood or urine test—environmental history and physical findings must support the diagnosis.

Urine or blood measurements in both adults and children who have chronic mercury exposure might not correlate with signs and symptoms of mercury toxicity. This might be due to several factors, including the storage of mercury in a relatively unexchangeable tissue compartment (i.e., renal cortex) or clearance from past exposure after irreversible signs and symptoms are manifested.

Further information on elemental mercury direct biologic indicators and treatment is in Appendix B (p. 69).

Diagnosis

Chronic elemental mercury exposure in children can cause a severe form of poisoning called acrodynia (i.e., painful extremities; also called pink disease) weeks or months after exposure. This condition is *rare*. Acrodynia is characterized by pruritus; paresthesia; generalized pain; pink rash; and peeling hands, nose, and feet (Table 1; p. 54). Other cases of acrodynia in

the literature are discussed in *Environmental Health Perspectives* (2000) and Paulson (2001).

The most common manifestations of long-term exposure to mercury vapor are effects on the central and peripheral nervous systems. Early nonspecific signs of exposure include insomnia, forgetfulness, loss of appetite, and mild tremor, and symptoms might be misdiagnosed as psychiatric illness. Continued exposure leads to progressive tremor, erethism (which is characterized by red palms, emotional lability, and memory impairment), distal paresthesia, motor and sensory conduction delay, and limb weakness. Other accompanying signs include salivation, excessive sweating, and hemoconcentration. Mercury can also accumulate in kidney tissues, with resulting renal toxicity (including proteinuria or nephrotic syndrome), alone or in addition to other signs of mercury exposure. Differences between adult and childhood manifestations of mercury poisoning are discussed in Weiss (2000).

Case Study *(Continued)*

In the hospital, you make a working diagnosis of mercury poisoning and consult with a pediatric environmental medicine specialist. You determine that this is a case of acrodynia. The child is treated. In this case of acrodynia, succimer (2,3-dimercaptosuccinic acid [DMSA]) is administered orally in three daily dosages. DMSA is dispensed in 100-milligram (mg) capsules that can easily be opened, allowing the drug to be mixed with a food product (e.g., applesauce) if necessary. This treatment is continued for several days; urine mercury excretion peaks shortly after introducing the chelation treatment. Clinical reassessment and urine mercury excretion are measured on day 7 of treatment. Urine mercury levels are still elevated and treatment is resumed at a lower dosage for 5 more days, during which the child's mood and rash begin to improve. He is completely asymptomatic the next week, and has no elevation in urine mercury excretion.

Appendix B (p. 69) includes further information regarding the treatment of elemental mercury poisoning.

Note: Chelation has been used to reduce body burden of elemental mercury; however, whether it reduces toxic effects or speeds recovery in mercury-poisoned children remains unclear (Etzel 2001). Chelation should only be used for symptomatic patients with known mercury exposure, and only after consideration of the risk and benefits by a specialist experienced in the use of chelators and in consultation with the patient or family.

Challenge

(1) What is the likely explanation for this apparent pattern of mercury exposure?

(2) What agencies might help you assess the nature and extent of this problem, and what procedures can help confirm the source of the mercury exposure?

(3) You call local public health officials to discuss the case. You are asked your thoughts about the case. They ask whether you suspect that others have been exposed. What is your answer?

(4) How do you explain the finding of high urine mercury levels in the child, but normal levels in other household members?

Case Study *(Continued)*

An investigation reveals that the mercury thermometer factory employees (including the child's father) change their clothing after working with mercury, but that most do not change their shoes. You also find out that the teenagers who took the elemental mercury from the local high school chemistry lab were questioned and evaluated. The elemental mercury was retrieved and environmental testing of possible points of exposure was completed. One of the teenagers who had been doing odd jobs at the patient's home admitted spilling mercury in the child's room on the carpet. She tried to clean up what she could see with a paper towel, then flushed it down the toilet. In addition to mercury contamination in the patient's home, more than a dozen other homes, several cars, shops, schools, and recreational areas in the community have also been contaminated. A surveillance program finds that urine mercury levels in the thermometer factory workers, their spouses, and adults not associated with the thermometer factory do not exceed levels of public health concern, but that elevated levels were found in a few young children whose homes and family cars were contaminated. These children have been followed up with pediatric environmental medicine expert consultation. In addition, the state division of environmental protection has been addressing the environmental contamination issues.

The environment is remediated before the child returns to his home (the carpet is replaced [Goldman et al. 2001] and follow-up testing of elemental mercury levels in the home within acceptable limits). The father changes his work clothes and shoes before coming home from work. The state division of environmental protection is also addressing any possible source of mercury from when the building in which the family lives was used for commercial activities (as a jewelry factory complex) and whether mercury might be continuing to undergo subsequent volatilization. Whether the elevated urinary mercury level and acrodynia in this child is due to the

take-home exposures, the jewelry factory complex, or the spill in his room, all possible exposure pathways have been mitigated.

Challenge

(5) The wife of another thermometer factory worker is breastfeeding her newborn infant. She is concerned that her breast milk might be exposing the infant to mercury. How would you advise her?

(6) A neighbor's teenage son plans to work this summer as an errand boy and custodian at the thermometer factory. His father hears about your patient's recent mercury exposure and calls to ask for your advice. Are there any special dangers to an adolescent working on the production floor of the factory? How would you advise him?

This case study addresses a situation where a high index of suspicion for an environmental cause of disease is warranted. However, different types of office visits—a well child coming in for routine care, a child coming in for an illness that might be related to an environmental exposure, or a child with a known or suspected exposure (with or without symptoms)—would call for different evaluative approaches. In any of these scenarios, however, a pediatrician or other child health care provider can integrate environmental health issues into practice. This integration will be explored later in the text.

In addition to mercury, children might be exposed to a variety of environmental toxicants encountered in indoor and outdoor environments of the home, child care setting, school, or workplace (including take-home exposures). Children might also be exposed to out-of-home pollutants, including those found in hazardous waste sites. About 15,000 uncontrolled hazardous waste sites exist throughout the United States, with 1,508 sites proposed or listed on the National Priorities List (NPL). ATSDR has used geographic information systems to estimate the number of children living near NPL sites. On the basis of data from 1,255 sites, 1,127,563 children <6 years of age live within 1 mile of the sites (about 11% of the potentially affected population), although it is important to realize that proximity alone does not mean that exposure will occur.

The Exposure-Disease Model

To better conceptualize "exposure" and the steps necessary to effect disease, the exposure-disease model (Figure 1; p. 53) is often used.

No matter how toxic, no chemical can harm a person (a child or an adult) unless *exposure* occurs. After a sufficient level of exposure (dose) to the chemical, with subsequent biologic uptake and target organ contact, biologic change can occur, which might lead to disease (Figure 1; p. 53). This

process is the same for everyone, although some toxicants might be more hazardous to a child than to an adult or vice-versa. Special consideration of a child's exposure and consumption patterns combined with critical periods of target organ development is necessary to assess a child's risk from a particular toxicant exposure. This is discussed further in the Age-Dependent Toxicokinetic Changes section.

Application of the preceding case study to the exposure-disease model follows:

- Environmental contamination (potential exposure): Elemental mercury, whether spilled or tracked on the carpet from contaminated work boots (or both), volatized at room temperature in the child's room or aerosolized by vacuuming the carpet. Vapors accumulate near the floor where children play and breathe.
- Biologic uptake (exposure): In this case, exposure occurs primarily though the respiratory system via inhalation. The respiratory rate is considerably higher in a child than in an adult. In the case study, the 2½-year-old child's respiratory rate is 30 breaths/minute. In adults, it is about 16 breaths/minute. Indicators of exposure in this case include increased urinary mercury.
- Target organ contact: Target organs might include the skin, central nervous system (CNS), peripheral nervous system (PNS), renal system, and respiratory system.
- Biologic change: In this case study, biologic changes include CNS changes (e.g., irritability), dermal changes (e.g., erythema of palms, soles, and face; with the characteristic edema and desquamation of the skin of the hands and feet), ocular changes (e.g., photophobia), and PNS changes (e.g., limb weakness and tongue tremor).
- Clinical disease: acrodynia.

> **Note:** This condition is **rare** (see discussion under Diagnosis section). Not all children exposed to mercury vapors will have acrodynia.

The exposure-disease model (Figure 1; p. 53) depicts the relationship between an environmental contaminant and an adverse health effect. The model predicts that the harm caused by a contaminant depends on its toxicity, route of exposure, and host factors. (For chemical properties, personal risk, biologic fate, and other information about mercury poisoning, see *Case Studies in Environmental Medicine: Mercury Toxicity* [ATSDR 1992].)

The Child as Susceptible Host: A Developmental Approach to Pediatric Environmental Medicine

Childhood is a time of rapid growth and development, accompanied by changes in organ system functioning, metabolic capabilities, physical size, and behavior that can dramatically modify the potential effects and illness caused by exposure to a toxicant.

Research has not yet satisfactorily answered how host characteristics can affect the harm caused by a toxic substance. The federal government has begun to mobilize the scientific community to focus on the possible unique vulnerabilities of children. Although for some selected agents, children are no more susceptible (and are sometimes less susceptible) than adults to an adverse outcome, theory and empirical observations point to common overall themes of increased susceptibility to environmental hazards throughout childhood.

Differing Susceptibilities of Children

Factors That Affect Exposure and Are Unique to Children and Infants

Caregivers have a direct impact on the safety and health of children. Caregivers are entrusted to not only protect children from danger, but to consult child health care providers appropriately. A child relies on adults for protection from environmental tobacco smoke (ETS), excessive exposure to sunlight, pesticides in the home, take-home occupational exposure, and other environmental exposures including noise. Children's own behaviors, physical characteristics, and diet peculiar to each developmental phase (Table 2; p. 55) can put them at greater risk for exposure to environmental hazards.

Opportunities for exposure change as a child grows from total dependence on his or her parents or other caregivers to adolescent independence. Economic circumstances, environmental regulations, and legislation can restrict or reinforce pediatric exposures.

Multiple factors that enhance a child's opportunity for exposure (Tables 2 and 3; pages 55 and 61, respectively) include the following:

- Children breathe more air, drink more water, and eat more food per kilogram of body weight than adults do.
- An infant's respiratory rate is more than twice an adult's rate.

- In the first 6 months of life, children drink seven times as much water per kilogram of weight than an adult does.
- From 1 to 5 years old, children consume three to four times more food per kilogram of weight than an adult does.
- Restricted food choices in the dietary patterns of infants and toddlers lead to greater exposures to contaminants unique to certain foods that often dominate their diets. For example, because children consume about 15 times more apples and apple products per unit of body weight than adults do, risk assessments based on a typical adult diet might underestimate a child's risk of exposure to pesticide residues on apples.
- Deficiencies of dietary iron and calcium can increase lead absorption.
- Some toxicants more readily penetrate children's skin, especially in the newborn period when the skin is highly permeable (e.g., dermal exposure to lindane and hexachlorophene, with subsequent development of neurotoxicity).

Other factors influencing both exposure to and absorption of environmental agents include a child's

- home, play, or day care environment;
- physical stature;
- mobility;

  metabolic rate; and
- increased surface area to body mass ratio (in young children).

For example, in a home contaminated with mercury (e.g., caused by spillage or from mercury carried home on work shoes), a toddler's high respiratory rate, proximity to surfaces likely to be contaminated, and playful rolling around on the floor will increase his or her chance for mercury exposure. Other possible contaminants that settle near the floor are pesticides, formaldehyde (from new synthetic carpet), and radon.

Age-Dependent Toxicokinetic Changes

As children age, changes in their physiology and body composition affect the absorption, distribution, storage, metabolism, and excretion of chemicals (Behrman et al. 1996). Organ-system function changes with development. As muscle and bone mass increase, internal organs become a smaller part of the total body. As the size and function of organs change, so does the dose necessary to alter those target tissues. The kinetics and toxicity of a chemical cannot simply be predicted from data derived entirely from adults or even from children of different ages. For example, methemoglobinemia from nitrate exposure might occur in newborns more readily than in other age groups because during the first 4 months of life, newborns have low

concentrations of reduced nicotinamide adenine dinucleotide (NADH) methemoglobin reductase (i.e., erythrocyte cytochrome b5 reductase). This enzyme reduces methemoglobin, rendering the enzyme nonfunctional for its oxygen-transporting function.

No simple generalization can be made about age-dependent changes in the metabolism of xenobiotics (i.e., foreign organic chemicals). First, efficient metabolism of a substance does not necessarily decrease its toxicity. In some cases, metabolic by-products are more toxic than their parent compound. Methyl parathion, an organophosphate pesticide for use on outdoor crops, but with a history of misuse indoors, is metabolized to more toxic by-products once exposure has occurred. It is the toxic by-products that cause organ damage.

Second, enzymatic pathways do not mature at equal rates: some mature rapidly, others slowly. For example, caffeine has a half-life of about 4 days in the neonate, compared to about 4 hours in the adult. Infants achieve adult rates of metabolizing caffeine by 7 to 9 months of age. Metabolism of some substances, such as theophylline (which is metabolized by the P450 cytochrome system), begins slowly at birth, exceeds that of adults in early childhood, and then falls gradually to adult rates by late adolescence. Further, different enzymatic pathways might be used in the metabolism of a particular chemical at different ages. For all of these reasons, studies of the variation in toxicokinetics with age must be compound-specific.

Under some circumstances, the immaturity of certain metabolic pathways in children might result in a lower susceptibility to certain toxicants (e.g., acetaminophen). In the adult, high levels of acetaminophen can cause fatal hepatotoxicity. However, infants delivered by mothers with high levels of acetaminophen will also have elevated acetaminophen levels in their blood, but will not sustain liver damage. It is thought that the fetus' inability to metabolize the acetaminophen protects the fetus from end-organ damage. Therefore, the biotransformation of xenobiotics is developmentally regulated and can either protect or harm the individual.

Organ Susceptibilities

The rapid development of a child's organ systems during embryonic, fetal, and early newborn periods makes him or her more vulnerable when exposed to environmental toxicants. These critical periods of vulnerabilities vary according to each organ system. CNS development occurs over a protracted period of time. Neuronal cell division is thought to be complete by 6 months of gestational age. However, CNS development continues to involve timed sequences of cell migration, differentiation, and myelination until adolescence. Disruption of these processes or their coordination before completion can result in irreparable damage. Different toxicants affect

different aspects of these sequences of events (e.g., cell proliferation is affected by irradiation, cell migration by ethanol, and cell differentiation by hypothyroidism) (Rice and Barone 2000), each resulting in functional impairments. Notably, the myelination of the brain and alveolarization of the lungs continue to develop throughout adolescence. Also during adolescence, the reproductive organs undergo hyperplasia, as well as maturation of structure and function.

Because children are at the beginning of their lives, more opportunity exists for both exposure to and expression of harmful effects from exposure to toxicants—especially those diseases with a protracted latency period (cancer). For example, the 1986 Chernobyl radiation exposure in Belarus, Ukraine, and Russia resulted in substantial increases in reported cases of thyroid cancer. Alterations in immunologic and thyroid parameters were observed in the exposed children monitored in one study for health status and level of internal contamination (DeVita et al. 2000). The Ukraine Health Ministry announced in 1997 that 10 times as many people (i.e., 50) are being diagnosed with thyroid cancer each year, compared to 5 per year before the accident. The ministry also stated that the death rate among those who stayed in the contaminated area was 18.3% higher than the national average.

Variations in Susceptibility With Developmental Stages

Much of the information in this section as well as in Table 2 (p. 55) is adapted from the work of Cynthia Bearer, MD, PhD (Bearer 1995a, 1995b).

Developmental milestones mark phases of changing susceptibility ("windows of vulnerability") that can profoundly affect the consequences of chemical exposures. This section highlights critical aspects of each stage to form the basis of anticipatory guidance and clinical evaluation (Table 2; p. 55). Not only are children different from adults with regard to susceptibilities, they are different among themselves according to age. Various exposure scenarios, and issues important to each developmental stage, will be presented by route. Environmental exposures occur predominantly through three major routes: ingestion (oral), inhalation (respiratory), and dermal (skin). Specific examples of exposures through these major routes are included for newborns and toddlers.

Anticipatory guidance is the education provided to parents or caretakers during a routine prenatal or pediatric visit to prevent or reduce the risk that their fetuses or children will develop a particular health problem (CDC 1997).

Preconception

Because oogonia fully develop during fetal life, oocytes rest dormant, vulnerable to environmental insults until the time of ovulation. Ova forming within the fetus of the future mother are affected by exposure from both her grandmother and her mother.

Although injury to stem-cell spermatogonia can occur at any time and lead to infertility, male reproductive biology presents repeated, narrow windows of vulnerability in parallel with the continual postpubertal production of semen and regeneration of spermatozoa. Paternal exposures might also lead to adverse reproductive outcomes by transmission of toxicants in seminal fluid. (See ATSDR's *Case Studies in Environmental Medicine: Reproductive and Developmental Hazards* [ATSDR 1993].)

Parental exposures before conception can result in an array of adverse reproductive effects ranging from infertility to spontaneous abortion, as well as genetic damage that can lead to a viable, though defective, fetus. For example, a woman who has experienced a prepregnancy exposure to lead and who was inadequately treated for lead poisoning in childhood might give birth to an infant with congenital lead poisoning (Shannon and Graef 1992). The most logical explanation for this would be storage of the lead in bone with mobilization during pregnancy (Silbergeld 1991).

Environmental tobacco smoke and alcohol are known, preventable human growth retardants. Anticipatory guidance by the primary health care provider to prospective parents can help prevent adverse fetal outcomes by encouraging prospective parents to protect their health and that of their unborn infant. Preconception counseling is imperative in proactively addressing issues that can significantly impact the health of the unborn child.

The Fetus

The fetus cannot escape the transplacental transport of toxicants encountered by the mother; that is a fact of fetal life. Both historic and gestational maternal exposures can affect the fetus. During gestation, the placenta, which establishes its circulation by around day 17 after fertilization, acts as the most important route of exposure. The placenta is a semipermeable membrane that permits easy transport of low-molecular-weight (i.e., carbon monoxide) and fat-soluble (i.e., polycyclic aromatic hydrocarbons and ethanol) compounds, as well as certain other compounds such as lead. Some water-soluble and high-molecular-weight compounds might also cross the placenta, albeit more slowly. The placenta has limited detoxification ability that helps mitigate only very low concentrations of toxicants.

Contaminants in a pregnant woman's current and past diet can harm the fetus. Physiologic changes during pregnancy mobilize stored toxicants, such as lead from bone or polychlorinated biphenyls (PCBs) from fat cells, resulting in fetal exposure. Maternal alcohol ingestion can lead to fetal alcohol syndrome, and maternal smoking during pregnancy has been associated with lower mean birth weight, increased risk of infant mortality, and decrements in lung function noted later in the life of the exposed child.

Anticipatory guidance by the child health care provider can help stop the parental consumption of tobacco and alcohol.

Fetal exposures can also occur independently of the placenta. These exposures include heat, noise, and ionizing radiation (Paulson 2001). A mother's exposure to ionizing radiation can increase the likelihood of the occurrence of childhood leukemia and neurologic delays. Although the mechanism is uncertain, some parental exposures during gestation, including anesthetic gases and some solvents, might be associated with adverse reproductive outcomes (ATSDR 1993).

During critical periods of organogenesis (i.e., the 6-week period that follows the establishment of the placental circulation), exposures can cause profound systemic damage that is out of proportion with the usual dose response. The fetal brain is particularly vulnerable because it lacks a blood-brain barrier or detoxification capabilities. *In utero* exposure to lead during this stage causes more damage to the nervous system than does exposure at any other stage of development. In the fetal brain, neurons originate in a central location (germinal matrix) and later migrate to predetermined sites. Exposure to ethanol during this stage might interrupt migration and lead to brain malformation, as is sometimes seen in fetal alcohol syndrome. High levels of methylmercury exposure from maternal consumption of contaminated fish from Minimata Bay, Japan, caused cerebral palsy and severe mental retardation in children born in Minimata. Some studies suggest that lower concentrations of maternal dietary methylmercury also can lead to neurodevelopmental delays and mild retardation. The fetus is at an increased risk of acute toxicity from carbon monoxide; levels that are harmless to healthy children can create permanent deficits of cognitive and motor functions in a fetus.

Rapidly dividing fetal cells might have increased sensitivity to carcinogens. Epidemiologic evidence, however, is contradictory on the relationship between age of exposure and cancer risk. As previously noted, it appears that during childhood, sensitivity to carcinogens increases in some organs and decreases in others. The only two generally accepted carcinogenic *in utero* exposures proven to result in cancer later in life in the exposed offspring include diethylstilbestrol (DES) (via placenta) and ionizing radiation (acting directly on the fetus) (Anderson et al. 2000; DeBaun and Gurney 2001; Lemasters et al. 2000).

Newborns (Birth to 2 Months), Infants (2 Months to 1 Year of Age), and Toddlers (1 to 2 Years of Age)

The growth rate during the first few months of life is faster that than during the rest of life. Tissues with rapidly dividing cells might be especially vulnerable to carcinogens; those vulnerable include tissues in the hematopoietic cells, lungs, and epithelium. Children's growth velocity

smoothly decreases around 9 months, to about half the initial rate. Although resistance increases, toddlers exhibit similar vulnerabilities in absorption, detoxification, and organ development as do newborns and infants.

Exposure by Ingestion

The small intestine of a newborn responds to nutritional needs by increasing the absorption of specific nutrients. For example, calcium transport in newborns and infants is about five times the rate in adults. If lead exposure occurs, the lead will compete with the calcium for transport at this high rate.

Breastfeeding

Breastfeeding is considered the optimal form of infant nutrition in most circumstances. Research indicates that human milk and breastfeeding of infants provide advantages with regard to general health, growth, and development, while significantly decreasing the child's risk for a large number of acute and chronic diseases. The many benefits to the infant provided by breastfeeding greatly outweigh the risk from possible contaminants in breast milk. For more information regarding contaminants in breast milk, a good resource is the AAP *Handbook of Pediatric Environmental Health* (Etzel and Balk 1999) chapter on human milk (Schreiber 2001).

When breastfed, a baby remains vulnerable to both current and historic maternal exposures. Lactation mobilizes previously sequestered fat-soluble toxicants such as dioxins, other chlorinated pesticides, PCBs, and bone lead, which then contaminate breast milk. Maternal toxicokinetics, the solubility and binding properties of a toxicant, and the characteristics of breast milk determine the milk-maternal plasma (M/P) ratio. The higher the ratio, the more complete the transfer of the substance into the breast milk. Neutral, basic, low-molecular-weight, highly lipophilic substances transfer most readily into breast milk. M/P ratios have been published for a variety of xenobiotics (Schreiber 2001). The M/P ratio for lipophilic substances such as PCBs range from 4 to 10; the ratio for organic and inorganic mercury is 0.9.

Formula Feeding

On a daily basis, a newborn infant consumes a much larger amount of water (equivalent to 10%–15% of his or her body weight) compared to an adult (2%–4% of body weight). Formula-fed infants consume significant amounts of water; average daily consumption might be 180 mL/kg/day (6 fluid ounces/kg/day), which is the equivalent for an average adult male of thirty-five 360-mL (12 fluid ounces) cans of soft drink per day (Paulson 2001 and Table 3 [p. 61]). Contaminants such as heavy metals and nitrates are not eliminated by boiling water, and are concentrated when water is boiled away. Water from municipal water systems is usually low in lead content, but the water can acquire lead from soldered pipe joints and brass fixtures inside the home. The first-draw water (i.e., water that has stood in

pipes) should be discarded. Boiling before formula preparation need not exceed 1 minute. Water in municipal systems might also contain contaminants such as microbes and trace amounts of organic chemicals. Many families use private well water and consider it safe, perhaps safer than municipal water. However, private well water is largely unregulated and unmonitored and presents the potential for exposure to a spectrum of contaminants at high concentrations.

Nitrates are a well-recognized problem in private well water. Factors leading to increased risk of methemoglobinemia from nitrate exposure in infants younger than 6 months of age include the following:

- Gastric pH of infants is higher for the first 1–2 months of life and does not drop to adult levels until 3 years of age (Marino 1991), leading to excess bacterial colonization, which increases the conversion of nitrates to nitrites.
- NADH-dependent methemoglobin reductase activity in infants is 60% of that in adults. The relative lack of methemoglobin reductase enzyme necessary to convert methemoglobin back to functioning hemoglobin leads to methemoglobinemia. At about 6 months of age, infants begin to have adult levels of NADH-cytochrome b5 reductase, which converts methemoglobin back to hemoglobin (Avery 1999). Other causes of methemoglobinemia include genetic deficiency in methemoglobin-reducing enzymes; genetic abnormalities in the hemoglobin making the protein more susceptible to oxidation; GI infections and inflammation and the ensuing overproduction of nitric oxide; and exposure to oxidant drugs and chemicals, including nitrites.

Pica

The avid oral exploratory behavior of infants and toddlers makes ingestion an important exposure route to consider. Children who eat nonfood items are exhibiting pica behavior. Soil pica is the recurrent ingestion of unusually high amounts of soil (i.e., on the order of 1,000–5,000 mg per day). Groups at risk of soil-pica behavior include children age ≤6 years and individuals who are developmentally delayed (ATSDR 2001a). ATSDR uses 5,000 mg soil per day as an estimate of soil intake for children with soil-pica behavior. Accessible environments might be contaminated with lead paint, chips, or dust particles; pesticides; take-home contaminants (e.g., mercury); lawn chemicals; or floor-cleaning products.

Solid Foods

Because a typical toddler's diet is relatively rich in fruit, grains, and vegetables, the risk is higher for a toddler's exposure to food-borne pesticide residues than it is for adults, who routinely consume fewer of these foods. Some regulations now acknowledge children's different exposures and susceptibilities in an attempt to lessen children's exposures to toxic chemicals. For example, the Food Quality Protection Act of 1996

states that pesticide tolerances need to be set to protect the health of infants and children.

Exposure by Dermal Absorption

The ratio of the newborn's skin surface area to body weight is approximately three times greater than that of an adult (Table 3; p. 61). Therefore, covering a similar percentage of the body with a substance that can be dermally absorbed will lead to a larger dose on a weight basis in a child than in an adult. Other factors affecting dose include the surface area exposed and the vehicle (which may promote contact/residence time). In addition, characteristics of the skin of a newborn (birth to 2 months) enhance the absorption of xenobiotics. The thick keratin layer, which protects an adult's skin when in contact with a toxicant, does not form during the fetal stage. This keratin layer begins to develop in the first 3–5 days after birth; it remains more susceptible to absorption throughout the newborn period and is independent of gestational age. As a result, the newborn skin readily absorbs chemicals. Hexachlorophene-containing compounds were routinely used in the 1950s for the skin care of newborns as a prophylaxis against *Staphylococcus aureus* infection. By 1971, the use of hexachlorophene preparation as a skin cleanser for newborns was restricted because studies showed that it disrupted the cell walls and precipitated cellular protein, causing vacuolization in the CNS. Other examples include Betadine scrubs, which have caused hypothyroidism in infants, and dermal absorption of aniline dyes, which were used in a laundry service's advertisement printed on diapers and resulted in methemoglobinemia (Graubarth et al. 1945; Howarth 1951; Chai and Bearer 1999).

Exposure by Inhalation: Respiration

The younger the child, the higher the respiratory rate and the higher the weight-adjusted dose of an air pollutant (Table 3; p. 61). A baby's exposure to indoor and outdoor air pollution closely mirrors that of its parents or caregivers; however, the vulnerability of the infant's respiratory system increases the risk that early exposures to combustion air pollutants (e.g., ETS) will slow the rate of pulmonary growth. Acute clinical effects in infants exposed to ETS can include laryngitis, tracheitis, pneumonia, increased morbidity from respiratory syncytial virus (RSV) infection, and chronic middle ear effusions (Cook and Strachan 1999; Gitterman and Bearer 2001). Respiratory exposures to air contaminants (e.g., ETS, dust mites, and cockroach antigens) during the first year of life have a greater influence on the incidence and severity of asthma than do exposures later in life (Etzel 2001).

As infants and toddlers begin to explore the world away from the arms of parents or caregivers, they are often in the microenvironments of the floor and ground. Some toxic gases, including mercury vapor, are heavier than air and layer close to the floor in these microenvironments. A child's high

respiratory rate in breathing zones close to the floor results in higher inhaled doses of toxicants than an adult would receive in the same room. Mercury vapors can cause severe respiratory complications and other health effects.

Young Child (2 Years to 6 Years of Age)

Special circumstances increase susceptibility in this age range. With the newly acquired ability to run, climb, ride tricycles, and perform other mobile activities, the young child's environment expands and so does the risk of exposure. Exploratory behaviors also continue, making this age group's susceptibilities very different than those of their younger peers.

If a young child's diet is deficient in iron or calcium, as is possible with children in this age group, the small intestine will be able to avidly absorb lead. Pica is also a consideration for this age group. Children <6 years are at high risk for soil pica (ATSDR 2001a).

School-Aged Children (6 Years to 12 Years of Age)

School-aged children spend increasingly greater amounts of time outdoors and in school and after-school environments—each of which has its own hazards. Outdoor air pollution includes widespread air pollutants such as ozone, particulates, and oxides of nitrogen and sulfur, which result primarily from fossil fuel combustion. Although these pollutants concentrate in urban and industrial areas, they are wind-borne and distribute widely. Wood-burning and industry in rural towns can create local pockets of intense exposure. Toxic air and soil pollutants might result from local sources such as hazardous waste sites, leaking underground storage tanks, or local industry. Children exposed to high doses of lead released into the air from a lead smelter in Idaho showed reduced neurobehavioral and peripheral nerve function when tested 15 to 20 years later (ATSDR 1997, 2001b).

History of school and after-school environments should be included when assessing exposure to indoor and outdoor air pollutants and contaminated drinking water and soil. During play or normal activity, children might ingest or inhale dirt or dust contaminated with arsenic, mercury, or other environmental toxicants.

Adolescents (12 Years to 18 Years of Age)

Adolescent behavior leads to new categories of potential exposures. Risk-taking behaviors of adolescents might result in exploring off-limit industrial waste sites or abandoned buildings or experimenting with psychoactive substances (e.g., glue sniffing). Adolescents might take jobs or enter vocational schools where they are exposed to workplace hazards. For more information about labor issues and adolescents, see Goldman et al. (2001). Adolescents sustain more occupational injuries and suffer more illnesses than their elder co-workers. Hobbies and school activities, such as arts and crafts or chemistry, are also more likely to involve exposure to

hazards than are the activities of younger children. Few schools include basic training in industrial hygiene as a foundation for safety at work, at school, or while enjoying hobbies. For example, there have been reports of teenagers taking elemental mercury from an old industrial facility and playing with and spilling the elemental mercury in homes and cars (Nadakavukaren 2000).

During the adolescent period, the metabolism rate of some xenobiotics dependent on the cytochrome P450 (CYP enzyme) system decreases as a result of changes in cytochrome P450 expression (Nebert and Gonzalez 1987) (e.g., theophylline, which has a subsequent increase in blood) (Gitterman and Bearer 2001). Studies indicate that the metabolic rate of some xenobiotics is reduced in response to the increased secretion of growth hormone and/or steroids that occur during the adolescent years (steroids compete with theophylline metabolism) (Gitterman and Bearer 2001). The implications of these changes for environmental contaminants is an area of intense research. Pubertal changes lead to new tissues with the special vulnerabilities associated with rapidly growing, dividing, and differentiating cells. Profound scientific and public interest in endocrine disruptors reflects concerns about the impact of persistent synthetic organic chemicals on the developing reproductive system. Studies have shown that by the end of puberty, the metabolism of some xenobiotics have achieved adult levels.

Sources of Exposure

Exposure to environmental toxicants can occur through contact with contaminated soil, food, water, or air. Examples throughout the Variations in Susceptibility With Developmental Stages section reflect the special exposure susceptibilities by age group. Table 4 (p. 62) summarizes common sources of contamination for different environmental media, by route; however, it is not exhaustive. Although many potential sources of exposure to environmental toxicants exist, this section will focus on take-home sources of exposure because these sources are an often overlooked, yet important, source of exposure.

Take-Home Contamination

The transmission of potentially toxic quantities of industrial chemicals from occupational settings to homes and residences is referred to as take-home contamination. Sometimes thought of as paraoccupational exposure, take-home contamination has been more vividly called “fouling one’s own nest.” Unlike the types of environmental contamination that might impact many individuals and large geographic areas (e.g., air pollution, spills of industrial chemicals, and accumulations of toxic wastes), take-home contamination most often affects the immediate exposed families of the involved workers.

Take-home contamination can occur even when appropriate precautions seem to be in place. For example, it is not enough that contaminated workers change clothing and shoes before returning home. Because some exposure risks are associated with laundering contaminated work clothes, such clothing should not be brought home to be cleaned. Instead, these clothes should be professionally laundered, preferably as part of the employer's occupational safety program. The hazards of seemingly casual exposure should also be recognized. In addition to laundering clothing at work, showering at work might also be necessary in some work settings to ensure that contaminants are removed from hair and skin.

Industrial Chemicals

The most direct form of take-home contamination results when industrial chemicals are carried from the workplace to the home on clothing, tools, shoes, skin, and hair (Chisolm 1978). The nature of the chemical and individual variables (e.g., age and activities) determine which family members are most at risk for developing adverse health effects from exposure. Small children are often most susceptible. For example, numerous reports document lead contamination among the children of lead workers (Watson et al. 1978; Etzel and Balk 1999). In such cases, preschool children might have blood lead levels equal to or greater than those found in the parent or parents who work with or around lead. Similarly, take-home contamination by mercury-exposed workers involved in thermometer manufacturing has led to the greatest blood levels of mercury documented in young children (Schreiber 2001) and in elevated mercury levels in children whose parents worked in a mercury thermometer plant (Hudson 1987).

A less obvious form of take-home exposure results from industrial chemicals in breast milk. Because human milk contains high levels of fat (about 4%), lipophilic compounds are preferentially taken up into breast milk (Schreiber 2001). For some industrial chemicals, breast milk concentrations are threefold to tenfold greater than corresponding maternal blood levels. Very few instances of harm have occurred in a nursing baby because of the baby ingesting chemicals found in his or her mother's milk. The many benefits to the infant provided by breastfeeding greatly outweigh the risk from possible contaminants in breast milk. Good resources discussing breast milk contaminants and breastfeeding are the AAP *Handbook of Pediatric Environmental Health* (Etzel and Balk 1999) chapter on human milk and Schreiber (2001).

Fibrous Materials

Workplace asbestos has been linked to asbestosis and mesothelioma in family members of asbestos workers (Anderson et al. 1979; Etzel and Balk 1999). Among spouses of asbestos workers (who may have laundered contaminated clothing) and children at home, radiographic abnormalities

consistent with asbestos exposure were almost seven times more frequent than expected. Asbestos take-home contamination can also be persistent. It has been found in the homes of former asbestos workers 20 years after the workers stopped working at the plant.

Organic Compounds

Chloracne has occurred in the children of workers exposed to trichlorophenol, dioxins, and other polycyclic halogenated compounds (Jensen 1972; Yoshimura 1974; Mocarelli et al. 1991). Contact with the parent's or caregiver's contaminated work clothing was the likely cause of this chloracne. Gynecomastia and breast discomfort occurred in children of workers employed in the manufacture of synthetic estrogens (Budzynska et al. 1967). Children of agricultural workers might have increased exposure to pesticides. In another example, a toddler suffered status epilepticus-type seizures after chewing plastic pellets that had adhered to her mother's work boots (Woody et al. 1986). The pellets contained an explosive compound used in munitions and rockets that were manufactured at the mother's workplace.

Principles of Environmental Medical Evaluation

Pediatricians and other child health care providers should continue to expand their skills in taking an environmental history, delivering anticipatory guidance, and conducting appropriate risk-based laboratory tests for environmental illnesses (in consultation with pediatric environmental specialists as necessary). Table 5 (p. 64) is a basic environmental database form that can be used in an office setting to keep a baseline environmental record handy in the patient's chart and update it as necessary. Portions of this tool could be self-administered in the waiting room, as is done with medical history questionnaires. The practitioner can review the form with the patient as necessary. Appendix C (p. 72), taken from the work of Sophie Balk, MD (Balk 1996), provides a summary of environmental health questions for an environmental history. Appendix C also includes a table describing when to introduce specific environmental health questions appropriate to age. Other pediatric environmental health history tools are also available (e.g., Goldman et al. 1999).

To determine whether an environmental factor plays a role in a child's illness, a high index of suspicion should be maintained. Most investigations that require the help of a specialist in environmental medicine begin in the primary care provider's office. Further probing can be done when a clinical presentation warrants. (See ATSDR's *Case Studies in Environmental Medicine: Taking an Exposure History* [ATSDR 2001c].)

Clinicians rarely see a child with a disease pathognomonic for environmental exposure, such as fetal alcohol syndrome or acrodynia (a manifestation of chronic mercury poisoning). Instead, a child generally will have a complex of signs and symptoms for which there is an extensive differential diagnosis and the possibility of multiple causes. Some common conditions might be caused by one of several environmental contaminants; for example

- seizures can occur as the result of lead poisoning or carbon monoxide intoxication;
- learning disabilities can have multiple contributing environmental factors, such as intrauterine alcohol exposure and lead intoxication; or
- eczema and other preexisting diseases can be aggravated by environmental factors (e.g., if an adolescent begins working with solvents in an auto mechanics class at a trade school).

Preconception and Prenatal Counseling

Preconception and prenatal counseling present opportunities to prevent exposures that might have devastating and lifelong effects. The March of Dimes and the U.S. Surgeon General recommend that preconception counseling be done by all primary care physicians. When providing preconception and prenatal counseling, a primary health care provider should include a screening environmental exposure history to assess basic environmental information about the home, occupations, and hazardous hobbies of both parents and of other adults living in the home (Table 5; p. 64).

Child health care providers should

- Provide parents with an environmental hazards checklist to be used to prepare the home for the arrival of their baby (Table 6; p. 67).
- Discuss the hazards associated with remodeling (e.g., lead poisoning) and furnishing a nursery (e.g., what items are considered safe).
- Warn parents about the intake of certain potentially contaminated foods, such as fish that might be contaminated with mercury. Health care providers can use local public health advisories or those provided by the U.S. Food and Drug Administration, ATSDR, or the U.S. Environmental Protection Agency (EPA).
- Counsel parents and caregivers about the use of prescribed and over-the-counter medications (e.g., Tylenol, aspirin, and cough suppressants that contain alcohol), nutritional supplements, alternative remedies, and other "natural" treatments.
- Review and discuss at length the hazards of alcohol and controlled substance use and abuse while pregnant. It is important to emphasize that environmental tobacco smoke, marijuana smoke, and cocaine smoke can adversely affect fetal health (Etzel and Balk 1999)

(Appendix D, p. 74, provides resources for further information) and that these are all preventable causes of potential adverse fetal health effects.

Visiting the Doctor's Office

Pediatricians or child health care providers can integrate environmental health issues into their practices in three basic scenarios.

For the Well Child

For the *well child*, a developmentally appropriate environmental checklist should be used to identify the child's potential exposure risks. Age-appropriate, environmental, anticipatory guidance should be provided (Table 2; p. 55), and risk-based screening tests for lead poisoning should be performed (Centers for Disease Control and Prevention 1997 and Appendix E [p. 82]). This is another opportunity to provide parents and caregivers with educational materials on how to prevent exposure to hazardous substances and what to do if exposure occurs. The reality of a general pediatrician or primary health care provider's practice is that there is little time to do an extensive environmental exposure history. At a minimum, the following questions taken from the AAP *Handbook of Pediatric Environmental Health* (Etzel and Balk 1999) should be integrated into the well-child visit:

1. Where does the child live or spend time?
2. Does anyone in the home smoke?
3. Do you use well water? Tap water?
4. Is the child protected from excessive exposure to the sun?
5. What do parents/teenagers do for a living?

Appendix C (p. 72) and Table 5 (p. 64) include information about taking an environmental history; Appendix D (p. 74) includes additional information and resources for environmental health concerns. Responses to the questions in Appendix C and Table 5 can guide the child health care provider in providing anticipatory guidance about preventing or stopping harmful environmental exposures. Additional questions can be added as necessary when trying to determine if specific community environmental health risks might be a problem for the child.

For the Sick Child

For the *sick child* whose illness might be environmentally related, the physician should consider an environmental agent as potentially related to a child's current illness, particularly when the illness in question does not follow a usual pattern, or when more than one family member or schoolmate is affected.

For the Child With a History of Known or Suspected Specific Exposure

For the *child with a history of a known or suspected specific exposure (with or without symptoms),* concerned parents might visit a child's health care provider with worries that their child might become sick in the future as a result of a suspected exposure. The parents might inquire about signs and symptoms associated with such exposures. This inquiry will help raise suspicion for a possible environmental etiology and thus guide the history and subsequent differential diagnosis.

Evaluating the Exposed or Sick Child

Because most environmental or occupational illnesses manifest as common medical problems or have nonspecific symptoms, an environmental etiology might be missed. Therefore, it is important to take an exposure history, especially if an illness has been unresponsive to therapy or has an atypical presentation. In a practical sense, an extensive environmental exposure history is beyond the scope of a primary child health care provider's practice. However, asking a few screening questions that would alert the provider to a possible environmental cause would then allow the general provider to contact experts in pediatric environmental medicine for further guidance in the diagnosis, treatment, and management of such cases. Following is the evaluation process in its entirety, with emphasis on what is generally feasible within the clinical generalist's practice and what would probably be referred to a pediatric environmental specialist.

Identify Specific Health Concerns

Questions that might help in discerning whether an illness is related to the environment (in addition to the screening exposure history questions taken at the well child visit) follow. [Questions taken from the AAP *Handbook of Pediatric Environmental Health* (Etzel and Balk 1999).]

1. Do symptoms subside or worsen in a particular location (e.g., home, child care, school, or room)?
2. Do symptoms subside or worsen on weekdays or weekends? At a particular time of day?
3. Do symptoms worsen during hobby activities, such as working with arts and crafts?
4. Are other children that your child spends time with experiencing symptoms similar to your child's?

Establish a Problem List

Using the traditional tools of interviewing, physical examination, and problem-specific laboratory tests, the child health care provider should attempt to objectify complaints and establish a problem list and a differential diagnosis. The evaluation might identify a specific organ disorder such as

eczematous changes in the skin, asthma, or hepatitis, or broad abnormalities such as developmental delays. In other situations, the initial problem list might only include signs, symptoms, and laboratory test results. The child health care provider who has experience with environmental toxicants might be quick to suspect a disease or a syndrome that has been associated with hazardous environmental exposures, such as asthma or acute lead toxicity. However, the problem list should still be used to keep the differential diagnosis broad in the beginning. Any and all specific exposures identified by the child's parents or caregiver(s) or suspected by the child health care provider should also be listed. Clinicians should be trained to seek sophisticated environmental etiologies when dealing with possible hazardous environmental exposures. In most cases, these etiologies will involve consultation and/or referral to a pediatric environmental medicine specialist. Appendix D (p. 74) includes information on the Pediatric Environmental Health Specialty Units.

Identify Key Exposures and Routes of Exposure

Every clinical evaluation of a sick child should include an exposure history that is developmentally appropriate and relevant to the problem list (Table 5 [p. 64], Appendices C [p. 72] and D [p. 74], and ATSDR's *Case Studies in Environmental Medicine: Taking an Exposure History* [ATSDR 2001c]). If certain responses to a few screening questions point to a possible environmental etiology, a more detailed environmental history should be taken. In some cases, consultation with a specialist in pediatric environmental medicine might be indicated. The child health care provider should also be alert to clusters of cases that come into the office that would prompt further investigations. Augment the basic environmental history that might already be part of the patient's chart with problem-specific questions. Even if a parent is focused on a specific exposure, collect information about all possible sources of exposure to environmental hazards. For example, when assessing a 4-year-old child with asthma, focus questions on sources of allergens at home, at preschool, or at the child care center, as well as exposure to outdoor or indoor irritating pollutants (e.g., cat hair, mold, ETS, home pesticides, cockroaches, and periodic high ozone levels). Health care providers must specifically identify chemicals and the routes by which a child might be exposed.

No matter how toxic, no chemical will harm anyone unless exposure (biologic uptake) with subsequent target organ contact occurs, thus causing biologic changes that can lead to disease (Figure 1; p. 53).

When parental occupations might result in take-home exposures, the child health care provider should request copies of the material safety data sheets (MSDSs) from the parent's employer about hazardous substances at work (see shaded box on p. 29). MSDSs can also be obtained from other

sources. To obtain more reliable information on substance-specific health effects, see Appendix D (p. 74).

An MSDS describes harmful routes of exposure for specific hazardous substances. The particular route of exposure often determines whether an environmental contaminant will cause harm. For example, a child might bite and break a thermometer and swallow its liquid contents. Fortunately, elemental mercury is relatively nontoxic when ingested because it is not well absorbed by the intestinal route. However, because of its high absorption rate by the respiratory route, elemental mercury is highly toxic when it volatilizes and is inhaled.

Research the Properties of Toxicants

After identifying the relevant environmental contaminants, their properties need to be researched. If the primary child health care provider is not familiar with the contaminant or if the case is complex, consultation with a pediatric environmental specialist, poison control center, and/or toxicologist is indicated (Appendix D; p. 74). Physical and chemical properties of a contaminant help to determine the likelihood of exposure and absorption and how a chemical will be metabolized and excreted if exposure or absorption occur.

For example, knowing that metallic (elemental) mercury volatilizes at room temperature helps predict the occurrence of respiratory exposure if a rug is contaminated with mercury. Air monitoring can contribute to an understanding of the extent of exposure. Because mercury vapor layers close to the floor, this situation leads to greater concern for exposure of young children.

Details about a substance's metabolism and excretion (toxicokinetic) characteristics help to predict the type of biologic monitoring that would be useful in measuring exposure. With information about the half-life of a substance, the clinician can better interpret the results of biologic testing for exposure. Finally, information about animal and human toxicities helps focus laboratory testing on organs known to be affected.

Characterize Exposure

Dose response refers to the extent of a biologic effect in relation to the received dose of an agent. Although variations exist, generally, the higher the dose, the greater the effect. One exception, as discussed previously, is that low doses at critical periods of organ development might have a greater effect than higher doses at other times. An environmental medical evaluation must characterize the extent of exposure with the goal of estimating as closely as possible the absorbed dose (Figure 1; p. 53). This is usually done in consultation with or referral to a pediatric environmental medicine specialist. Exposure intensity, duration, and frequency all contribute to dose

An MSDS provides information about the hazardous ingredients of a product, its physical and chemical properties, relevant occupational standards, basic toxicologic and industrial hygiene data, and information about how to contact the manufacturer for additional details. **Although they are a good beginning, MSDSs might be incomplete, inaccurate, or unhelpful—particularly with respect to chronic exposures and their potential effect on children.** A health care provider only needs the name of a product to obtain its MSDS through the manufacturer or obtain reliable substance-specific medical information through the local poison control center or one of several Internet sites (Appendix D [p. 74]).

The Occupational Safety and Health Administration (OSHA) requires employers to maintain MSDSs on all chemical products used in their facilities. OSHA regulations require employers, if asked, to provide relevant MSDSs to their employees, their representatives, and their health care providers. The Superfund Reauthorization Act also requires businesses to provide MSDSs to concerned community members when the products in question might be released into the community.

considerations. The exposure assessment relies on three tools: the exposure history; the environmental monitoring performed on environmental samples; and the biologic monitoring performed on samples of blood, urine, or other body fluids or tissues from the exposed person.

Further Considerations

Even though more detailed information regarding environmental history, environmental monitoring, biologic monitoring, risk communication, and risk assessment goes beyond what a primary health care provider will realistically know and do in the midst of a busy practice setting, this information is provided in Appendix F (p. 83) to help with understanding the role of others and communication with others (e.g., staff at the state or local health department, poison control center, ATSDR, Association of Occupational and Environmental Clinics' Pediatric Environmental Health Specialty Units [PEHSUs; p. 79], and experts at other organizations). Appendix F also provides a better understanding of what is involved in doing a comprehensive pediatric environmental medical evaluation.

Pediatric Environmental Health Interventions

The Six Interventions for Clinical Management of an Environmental Medical Problem

1. Cessation or Minimization of Offending Exposures

Orchestrating the elimination or reduction of ongoing exposure of a child to an environmental contaminant deemed hazardous or potentially hazardous is one important role for the child health care provider. By hospitalizing a child poisoned by a heavy metal, the physician might initiate hazard reduction by removing the child from the offending environment. Before returning the child to his or her home, however, the environmental hazard must be eliminated or mitigated. Whenever possible, the offending agent should be entirely removed from the child's environment. If the agent serves an important function and it is possible to substitute a less toxic alternative, substitution should be made. For example, homeowners might replace lead paint with a nonlead alternative. However, because a toxicant becomes hazardous only to the extent exposure occurs, other measures can often accomplish the goal of hazard reduction more quickly and inexpensively. For example, measures could include (a) blocking pathways of exposure by encapsulating friable asbestos insulative lagging on pipes to reduce indoor air asbestos contamination or (b) putting household chemicals out of reach. Polluted tap water and poor indoor air quality can sometimes be managed through treatment technologies. Other measures for reducing hazards might include

careful home and personal hygiene, including weekly wet-wiping of lead-dust–contaminated windowsills and, for those children living in homes with lead paint, enforced handwashing before meals and at bedtime.

In many cases, specially trained workers and anticipatory guidance from child health care providers can provide appropriate direction to a family to make an environment safer for a child. Parents of children with asthma can be given information from the American Lung Association on reducing environmental asthma triggers. Preprinted information for a variety of other hazards such as medicines, pesticides, or other household chemicals can supplement age-appropriate anticipatory guidance. Appendix D (p. 74) lists books and organizations that provide detailed information for families about reducing a child's exposure to environmental hazards.

Improper attempts by untrained persons to mitigate environmental contaminants can lead to dramatic exposures. For example, an untrained individual who attempts to remove lead paint might acutely poison himself or herself and others (such as children). When in doubt, medical providers should collaborate with public health agencies and remediation specialists. In some cases of typically acute exposures, the exposure cessation involves medical as well as environmental interventions. For example, the first responder's treatment of a person who has been exposed to a hazardous pesticide begins with removing the individual from the contaminated environment, removing the individual's tainted clothing, and grossly decontaminating the individual's body (e.g., by giving the individual a shower). More refined decontamination then continues in the medical setting. Other medical interventions designed to stop the absorption of certain toxicants include the use of activated charcoal, gastric lavage, emetics, and cathartics for acute ingestion. However, it is important to remember that these measures are *not* recommended for all toxicants and might be contraindicated for some. Therefore, you must check with an up-to-date resource, such as the local poison control center, for current substance-specific treatment recommendations.

2. Standard Supportive Medical Therapy

Standard supportive medical protocols and pharmaceuticals are used to treat the majority of environmental illnesses. In most situations, the environmental contribution to an illness will not be immediately apparent. Respiratory failure, asthma, contact dermatitis, cancer, and other medical conditions call for standard therapies, pending determination of an environmental cause or trigger. Even then, medical treatment only rarely involves the use of medical therapies specific to a particular chemical agent. The *Medical Management Guidelines for Acute Chemical Exposures* (ATSDR 2001d) reviews the appropriate medical management of many of the most common acute chemical exposures. For many acute *known* exposures, when or if the child is very ill, or for *unknown* exposures, when

the child's signs and symptoms do not follow a usual pattern, consultation with hospital emergency room physicians, pediatric intensive care specialists, medical toxicologists, and/or environmental medicine specialists should be considered (e.g., PEHSUs [p. 79]).

3. Substance-Specific Medical Interventions

Although only relatively few substances have specific medical therapies, these therapies can enhance the elimination of an agent, block its absorption, reverse its effect, or otherwise render it less harmful. After identifying the offending agent, the child health care provider should consult texts, electronic databases, agencies, or experts to ascertain whether specific therapies exist for the exposure. Telephone hotlines through regional poison control centers, ATSDR, and PEHSUs, provide 24-hour support for clinical decision-making in cases of acute exposure (Appendix D; p. 74).

4. When to Refer

The primary health care provider's privileged position of trust with patients provides an early opportunity for more effective communication with parents and coordination of medical care in the event of an exposure. The pediatric generalist, however, will rarely have the specialized knowledge necessary for the management of less common environmental problems. The practitioner should work with specialized professionals to develop and support an appropriate therapeutic plan. Indications for referral to an environmental medicine specialist or government or private organization for assistance include the following:

- uncertainty about the extent and nature of relevant exposures,
- uncertainty about an environmental relationship to a specific health problem,
- uncertainty in risk characterization,
- the need for assistance with accurate and understandable risk communication information,
- presentation of similar problems from similar environments for several patients,
- the need for specialized diagnostic or therapeutic interventions,
- the need for expensive environmental mitigation management,
- consideration of a novel environmental diagnosis, and
- a hazardous exposure with public health implications.

5. Family Education and Risk Communication: Talking With Parents

Communication is essential in forming the necessary therapeutic alliance among the health care worker, the patient, and the patient's family. A

communication tool designed by Bernzweig et al. (1994) can enhance clinician-patient interaction.

6. Public Health Reporting

Many states require reporting of *specific* environmental illnesses such as lead or pesticide poisoning. Beyond these requirements, however, every case of environmental illness that the child health care provider identifies presents the opportunity for preventing further harm not only to the actual patient, but also to others. If one household member was exposed, presumably others in the household or community might also be exposed unless the physician initiates an appropriate environmental investigation with the help of those with special expertise. The physician has an obligation to take steps to prevent these additional exposures. In cases where public health reporting is not an issue (e.g., urging parents to eliminate exposure to ETS or remove animals from the home), anticipatory guidance could be important. In complex situations, the physician should report environmental exposures and illnesses to the appropriate public health authorities.

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Answers to Pretest and Challenge Questions

Pretest

(a) Relevant information should be obtained about the child's home environment and neighborhood. For example, it is important to find out the following information:

- type and age of the child's home,
- whether the home was recently remodeled,
- history of previous industrial use of the property,
- water source and heating system used in the home,
- any use of household chemicals (e.g., pesticides),
- occupations of adults in the home (e.g., the mother or father could bring hazardous materials home from work on clothes or shoes),
- hobbies of household members,
- herbal medicine consumption by anyone in the home, and

- whether any rituals that use mercury are conducted. (Some ethnic traditions encourage sprinkling of mercury around the house for religious reasons. Mercury or azogue is sold at shops or botanicas.)

Questions should also be asked about child care arrangements (e.g., type, condition, and age of the facility; location; habits of the caregiver [e.g., smoker or nonsmoker]; commonly used play areas; outdoor activities; and other potential sources of hazardous exposures to chemicals) (Tables 4 and 5 and Appendix C; pages 62, 64, and 72, respectively). In most cases, the interview alone will be sufficient to gather the information needed to assess the potential for hazardous exposures at home. Data collected by interview can then focus biologic testing to consider an environmental etiology of the presenting problem.

The interview and the results of clinical laboratory assessments will direct where focused evaluation of the child's environment is needed. If an environmental cause of a syndrome is strongly suspected, but no obvious source is identified by interview or clinical examination, a house call by an environmental assessment specialist might be indicated to determine the potential for hazardous environmental exposures.

(b) The patient's problem list is as follows: anorexia and weight loss; irritability and photophobia; weakness; pruritic rash on trunk and face; peeling, erythematous rash on hands and feet; and mild hypertension.

(c) The differential diagnoses for a 2½-year-old child who has new, but insidious, onset of behavioral change with irritability; photophobia; anorexia; and an excoriated papulovesicular rash on his trunk and face, with sweaty, pink and scaling skin on his hands and feet, include the following:

- heavy metal intoxication (e.g., lead or mercury),
- collagen vascular disease (e.g., juvenile rheumatoid arthritis [JRA]),
- brain tumor,
- acute rheumatic fever,
- meningitis,
- Fifth disease (*erythema infectiosum*),
- Kawasaki syndrome,
- nutritional deficiency,
- leukemia,
- immune deficiency disorder,
- metabolic disturbances,
- CNS degenerative disorders,
- pheochromocytoma, and
- psychosocial disturbances.

Because of the relatively long history (1½ months) of symptoms, the likelihood seems small for most infectious diseases. The history of normal growth, diet, and past use of multivitamins with iron makes the likelihood of a primary nutritional deficiency remote. CNS degenerative conditions do not show up with rash; then again, the rash on the trunk and face could be unrelated to the child's refusal to walk. Metabolic disturbances cannot yet be ruled out, but they do not include photophobia. Psychosocial disturbances could be a consequence rather than a causal

factor. Leukemia, collagen vascular diseases (e.g., JRA), acute rheumatic fever, vasculitis (e.g., Kawasaki syndrome), and intoxications also cannot yet be ruled out.

From an environmental/medical point of view, possible sources of intoxication include exposure to heavy metals such as lead and mercury. The distinctive dermatitis of hands and feet, along with CNS symptoms, suggest acrodynia, a form of childhood poisoning usually due to chronic elemental or inorganic mercury intoxication. Acrodynia develops after the mercury volatilizes or oxidizes, or both. In acrodynia, also known as "pink disease," the hands and feet are described as puffy, pink, paresthetic, perspiring, and painful.

Note: This is not a comprehensive listing of all differential diagnoses a health care provider would or could consider for this case scenario. The list should be used as a working guide only.

(d) Without prompting or experience with a recent evaluation of a case of pediatric mercury poisoning, a pediatrician or family physician is not likely to include environmental exposure as a primary or even secondary consideration when elaborating a differential diagnoses on the basis of the clinical picture presented. More than likely, the baseline tests ordered would include

- white blood cell count with differential;
- blood smear;
- electrolytes, with blood urea nitrogen and creatinine;
- erythrocyte sedimentation rate, antinuclear antibody, antistreptolysin-O titer;
- urine analysis with specific gravity;
- radiograph of chest, knees, and bilateral hips;
- a computed tomography scan of the brain to rule out degenerative changes or a space-occupying lesion; and
- a spinal tap (after risk for herniation has been excluded).

For those health care providers alert to environmental etiologies, screening for heavy metals (e.g., blood lead and urine mercury) in spot urine would be ordered.

(e) Urine tests provide the best estimates of the current body burden of chronic mercury poisoning. Elemental and inorganic mercury are mainly excreted in the urine. Laboratory confirmation of exposure to elemental and inorganic mercury can best be obtained by measuring the level of total mercury in a 24-hour urine collected in an acid-washed container. A first morning void can provide reasonable accuracy if the sample is adjusted for concentration of urine by using urine creatinine or specific gravity. Blood mercury levels reflect mainly recent elemental and inorganic mercury exposure (i.e., within 5 to 7 days) and correlate poorly with clinical effects. Appendix B (p. 69) has more information about lab testing and elemental mercury.

Also, for many acute *known* exposures, when or if the child is very ill, or for *unknown* exposures, when the child's signs and symptoms do not follow a usual pattern, consultation with hospital emergency room physicians, pediatric intensive care specialists, medical toxicologists, and/or environmental medicine specialists should be considered (e.g., PEHSUs; p. 79).

Challenge

(1) The child and grandmother have evidence of exposure to elemental and/or inorganic mercury at the following levels: 321 and 37 µg/g creatinine, respectively. The father and the mother have lower levels of exposure (18 and 12 µg/g creatinine, respectively). This pattern of mercury exposure suggests that the child's and grandmother's exposures are occurring at home, where both spend more time than either parent.

Three possible explanations should be considered. First, the father, who is almost certainly exposed to mercury in his work, might be bringing mercury home on his shoes or clothing, which has subsequently contaminated the rugs and volatilized at room temperature. Second, mercury might have been spilled recently in the family's home (a) by the teenager who cleans the home and who was involved with taking mercury from the chemistry lab, (b) by a broken thermometer or other mercury-containing instrument, or (c) as a result of a family hobby (e.g., cosmetic products or metallurgy); this mercury might have volatilized after exposure to room temperature. Third, mercury might have been spilled at some earlier time, when the building in which the family lives was used for commercial activities, and might be continuing to undergo subsequent volatilization. In any case, a 2½-year-old boy spends considerably more time playing closer to the floor than an adult does; thus, the boy will be exposed to the volatilized mercury.

About 30% of interior latex paints manufactured before 1990 contain mercury compounds that might volatilize at room temperatures. Although paint manufacturers voluntarily removed mercury from latex paints in August 1990, many people keep partially used cans of old paint for repainting. Therefore, pre-1990 paints might continue to be a source of mercury exposure. In this case, however, paint is an unlikely contributor to the mercury contamination because significant mercury exposures occur shortly after the application of mercury-containing paint (Aronow et al. 1990), and this home was not painted recently.

(2) Levels of ambient mercury should be measured in the home. Such testing and related assistance can usually be obtained through local or state public health officials. In some communities, poison control center professionals can facilitate appropriate testing of the home environment. Other sources of clinical toxicologic information and technical assistance include ATSDR and EPA (Appendix D; p. 74).

Information should also be gathered about possible take-home contamination from the father's workplace. The child health care provider and/or pediatric environmental medicine specialist might interview the father and also talk to a safety officer at the thermometer factory. MSDSs, listing hazardous agents used in the factory, should be requested by the child's father or the child health care provider. Because elemental mercury used in the manufacture of thermometers adheres easily to work clothing, work practices at the factory should be reviewed. The physician should ask whether factory workers wear appropriate protective clothing and whether contaminated shoes and clothing are left at the factory. In some cases, contaminated shoes and clothing might either be worn home or brought home for laundering, which allows take-home contamination. State and federal OSHA offices can provide information and assistance to reduce such workplace health concerns. The National Institute for Occupational Safety and Health (NIOSH) is another resource for workplace health information (Appendix D; p. 74).

Before discharging the child from the hospital, the health care provider must ensure no possible mercury exposure exists at home (i.e., remediation has taken place). Because of the high risk for increased mercury absorption after chelation, this is especially important if the child received chelation therapy. As part of discharge planning, health care providers should share usable information and materials or provide informational resources to parents and caregivers on how to properly store and discard medications, batteries, tools (e.g., thermometers), disinfectants, and cooking and garden products, among others. Parents and caregivers can conduct their own environmental surveillance in the home. A checklist of possible contaminants and steps to take to prevent accidents that result in

exposure is available for health care providers to distribute to parents and caregivers as part of anticipatory guidance practices (Table 6; p. 67). Health care providers should also encourage parents and caregivers to keep the local poison control center number close to all telephones in the home (Appendix D; p. 74).

(3) Mercury exposure might have affected others in the same apartment building or community. If exposures resulted from take-home contamination, the homes of other workers also might be contaminated. If exposure resulted from a spill of the mercury taken by teenagers from their school chemistry lab, others might also be at risk. If the exposure resulted from old contamination of the loft building, other residents of that building are at risk. Public health officials should conduct evaluations to determine if other groups have actually been exposed. In addition, the industrial hygiene practices at the thermometer factory should be reviewed to ensure that they are adequate.

(4) Young children are at particular risk from take-home as well as "in-home" contamination. A young child usually spends more time in the contaminated home compared to the parents and school-aged siblings. Also, mercury vapor is much heavier than air and tends to collect near the floor, where infants crawl, toddlers walk, and young children play, thus risking greater exposure to higher mercury air levels than are encountered by most adults.

(5) Mercury can be found in breast milk, but levels of concern regarding infant toxicity have mainly been associated with maternal exposure to organic mercury compounds (e.g., methylmercury), not elemental mercury vapor. If levels of mercury in the mother's urine are normal, breastfeeding will probably pose no exposure hazard to the infant. If the mother's urine mercury levels are high, however, mercury levels in breast milk should be measured to ensure that they pose no risk to the infant. Breast milk mercury levels >4 μg/L exceed the safe intake level for an infant. Because breastfeeding is the optimal infant nutrition, the child health care provider should evaluate each case individually, after a careful physical examination of the child (although with low-level exposures, overt symptoms are unlikely), to determine whether the risks of breastfeeding outweigh the benefits. For more information about breastfeeding issues, see the AAP *Handbook of Pediatric Environmental Health* (Etzel and Balk 1999) chapter on human milk.

(6) This teenage boy might be at increased risk of toxic mercury exposures for a number of reasons.

- First, his proposed cleaning activities might involve extended contact with mercury-contaminated waste materials.
- Second, part-time and temporary workers might not have an adequate opportunity to learn the proper use of personal protective equipment or might not fully understand or be aware of which hazardous substances to which they could be exposed at work and the health risks involved with such exposures.
- Third, the typical sense of invulnerability in adolescents might reduce the boy's vigilance in the use of protective equipment and other measures to minimize exposure. Fourth, the higher physical activity level of many teenagers might result in increased respiratory rate and volume and, therefore, greater inhalation exposures.
- Fifth, an adolescent might be fascinated with mercury as a toy or object to show off to his friends and he might be tempted to take some home.

As a result of increased risk of toxic mercury exposure, this teenager might exhibit neurologic effects similar to those seen in adults. In addition, he shares a similar risk of being the source of mercury take-home exposure to family members, who could include young children, by taking it home on his clothing, shoes, hair, and body.

Case Studies in Environmental Medicine:

Pediatric Environmental Health

Evaluation Questionnaire and Posttest, Course Number SS3098

Course Goal: To increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients.

Objectives

- Describe how and why children differ from adults in their susceptibility to environmental hazards.
- Apply the knowledge of environmental medicine in the evaluation of well and sick children.
- Identify parental occupation and hobbies as a part of the environmental history.
- Identify additional sources of environmental health information.

Tell Us About Yourself

Please carefully read the questions. Provide answers on the answer sheet (page 49). Your credit will be awarded based on the type of credit you select.

1. What type of continuing education credit do you wish to receive?
****Nurses should request CNE, not CEU. See note on page 48.**

A. CME (for physicians)
B. CME (for non-attending)
C. CNE (continuing nursing education)
D. CEU (continuing education units)
E. [Not used]
F. [Not used]
G. [Not used]
H. None of the above
I. CHES (certified health education specialist)

2. Are you a...

A. Nurse
B. Pharmacist
C. Physician
D. Veterinarian
E. None of the above

3. What is your highest level of education?

A. High school or equivalent
B. Associate, 2-year degree
C. Bachelor's degree
D. Master's degree
E. Doctorate
F. Other

4. Each year, approximately how many children do you see as patients?
A. None
B. 1–5
C. 6–10
D. 11–15
E. More than 15

5. Which of the following best describes your current occupation?
A. Environmental Health Professional
B. Epidemiologist
C. Health Educator
D. Laboratorian
E. Physician Assistant
F. Industrial Hygienist
G. Sanitarian
H. Toxicologist
I. Other patient care provider
J. Student
K. None of the above

6. Which of the following best describes your current work setting?
A. Academic (public and private)
B. Private health care organization
C. Public health organization
D. Environmental health organization
E. Non-profit organization
F. Other work setting

7. Which of the following best describes the organization in which you work?
A. Federal government
B. State government
C. County government
D. Local government
E. Non-governmental agency
F. Other type of organization

Tell Us About the Course

8. How did you obtain this course?
A. Downloaded or printed from Web site
B. Shared materials with colleague(s)
C. By mail from ATSDR
D. Not applicable

9. How did you first learn about this course?

A. State publication (or other state-sponsored communication)
B. *MMWR*
C. ATSDR Internet site or homepage
D. PHTN source (PHTN Web site, e-mail announcement)
E. Colleague
F. Other

10. What was the most important factor in your decision to obtain this course?

A. Content
B. Continuing education credit
C. Supervisor recommended
D. Previous participation in ATSDR training
E. Previous participation in CDC and PHTN training
F. Ability to take the course at my convenience
G. Other

11. How much time did you spend completing the course, evaluation, and posttest?

A. 1 to 1.5 hours
B. More than 1.5 hours but less than 2 hours
C. 2 to 2.5 hours
D. More than 2.5 hours but less than 3 hours
E. 3 hours or more

12. Please rate your level of knowledge before completing this course.

A. Great deal of knowledge about the content
B. Fair amount of knowledge about the content
C. Limited knowledge about the content
D. No prior knowledge about the content
E. No opinion

13. Please estimate your knowledge gain after completing this course.

A. Gained a great deal of knowledge about the content
B. Gained a fair amount of knowledge about the content
C. Gained a limited amount of knowledge about the content
D. Did not gain any knowledge about the content
E. No opinion

Please use the scale below to rate your level of agreement with the following statements (questions 14–23) about this course.

A. Agree
B. No opinion
C. Disagree
D. Not applicable

14. The objectives are relevant to the goal.

15. The tables and figures are an effective learning resource.

16. The content in this course was appropriate for my training needs.

17. Participation in this course enhanced my professional effectiveness.

18. I will recommend this course to my colleagues.

19. Overall, this course enhanced my ability to understand the content.

20. I am confident I can describe how and why children differ from adults in their susceptibility to environmental hazards.

21. I am confident I can apply the knowledge of environmental medicine in the evaluation of well and sick children.

22. I am confident I can identify parental occupation and hobbies as a part of the environmental history.

23. I am confident I can identify and identify additional sources of environmental health information.

Posttest

If you wish to receive continuing education credit for this program, you must complete this posttest. Each question below contains five suggested answers, of which one or more is correct. **Circle all correct answers on the answer sheet.**

24. The acrodynia (pink disease) syndrome includes all of the following except:

A. hypertension
B. tachycardia
C. hypotonia
D. dry mouth
E. desquamation of the skin of hands and feet.

25. Circumstances where a primary health care provider might refer to an environmental medicine specialist or government or private organization for assistance include

A. uncertainty about the extent and nature of relevant exposures
B. uncertainty in risk characterization
C. consideration of a novel environmental diagnosis
D. the need for specialized diagnostic or therapeutic intervention
E. well-child visit.

26. Likely sources of mercury poisoning in this case study include all of the following except:

A. take-home contamination from the father's workplace
B. spillage of elemental mercury in the child's carpeted bedroom
C. take-home contamination from the mother's workplace
D. mercury spilled in the building long before conversion to apartments
E. accidental ingestion of mercury from a broken thermometer.

27. Environmental toxicants other than mercury stored in fat and cleared from the body by breast milk include

A. lead
B. dioxin
C. polybrominated biphenyls
D. environmental tobacco smoke
E. polychlorinated biphenyls.

28. What are the special susceptibilities of newborn infants that place this age group at increased risk of exposure?

A. restricted diets
B. low respiratory rate compared to adults
C. thin keratin layer of their skin
D. sleeping patterns
E. larger skin surface-to-volume ratio.

29. Which of the following statements are true?

A. Efficient metabolism of the toxicants will always decrease their toxicity.
B. Metabolic by-products can be less or more toxic than the parent compound.
C. The toxicity of all compounds decreases with increasing age.
D. The study of the variation in toxicokinetics with age must be compound specific.
E. The placenta permits easy transport of high-molecular-weight and water-soluble compounds.

30. When taking an exposure history, it is essential to include which of the following?

A. Type of heating system in the home where the children live.
B. Location and year the house was built.
C. Parental occupation(s) and hobbies.
D. History of renovations and interior decoration and/or acquisition of new furniture in the last 3 years.
E. Environmental tobacco smoke.

31. The adolescent period leads to new categories of potential exposures because of

A. cell proliferation of the reproductive system
B. tutoring time needed by some youths
C. risk-taking behavior, disregard for warnings
D. accelerated growth
E. increased respiratory rate.

32. Which of the following are not part of the clinical management interventions in an environmental medical problem?

A. Family education and risk communication.
B. Substance-specific interventions.
C. Diet rich in proteins.
D. Cessation or minimization of offending exposure(s).
E. Public health reporting of parental environmental tobacco smoke.

33. Components of the Exposure-Disease Model necessary to arrive at clinical disease include which of the following?

A. biologic plausibility
B. biologic uptake
C. target organ contact
D. biologic change
E. all of the above.

Note to Nurses

CDC is accredited by the American Nurses Credentialing Center's (ANCC) Commission on Accreditation. ANCC credit is accepted by most State Boards of Nursing.

California nurses should write in "ANCC - Self-Study" for this course when applying for relicensure. A provider number is **not** needed.

Iowa nurses must be granted special approval from the Iowa Board of Nursing. Call 515-281-4823 or e-mail marmago@bon.state.ia.us to obtain the necessary application.

Case Studies in Environmental Medicine:

Pediatric Environmental Health

Answer Sheet, Course Number SS3098

Instructions for submitting hard-copy answer sheet: Circle your answers. To receive your certificate, you must answer **all** questions. Mail or fax your completed answer sheet to

Fax: 770-488-4178, ATTN: Continuing Education Coordinator

Mail: Continuing Education Coordinator
Agency for Toxic Substances and Disease Registry
Division of Toxicology and Environmental Medicine
4770 Buford Hwy, NE (Mail Stop F-32)
Atlanta, GA 30341-3717

Be sure to fill in your name and address on the back of this form.

Remember, you can access the case studies online at www.atsdr.cdc.gov/HEC/CSEM/ and complete the evaluation questionnaire and posttest online at www2.cdc.gov/atsdrce.

Online access allows you to receive your certificate as soon as you complete the posttest.

1. A B C D E F G H
2. A B C D E
3. A B C D E F
4. A B C D E
5. A B C D E F G H I J K
6. A B C D E F
7. A B C D E F
8. A B C D
9. A B C D E F
10. A B C D E F G
11. A B C D E
12. A B C D E
13. A B C D E
14 A B C D
15. A B C D
16. A B C D
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28. A B C D E
29. A B C D E
30. A B C D E
31. A B C D E
32. A B C D E
33. A B C D E

Name: E-mail (not required):

Address:

Zip code:

○ Check here to be placed on the list to pilot test new case studies

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Place Stamp Here

Continuing Education Coordinator

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Access the case studies online at www.atsdr.cdc.gov/HEC/CSEM/ and complete the evaluation questionnaire and posttest online at www2.cdc.gov/atsdrce.

Online access allows you to receive your certificate as soon as you complete the posttest.

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Appendices

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- Table 4. Sources of Common Environmental Hazards
- Table 5. Basic Environmental Database
- Table 6. Environmental Hazards Checklist for Home Assessment

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  - State and Local Health Departments

Appendix E: Lead Screening

Appendix F: Additional Information for Performing a Comprehensive Pediatric Environmental Medical Evaluation
- Exposure History
- Environmental Monitoring
- Biologic Monitoring
- Final Steps

Appendix A: Figure and Tables

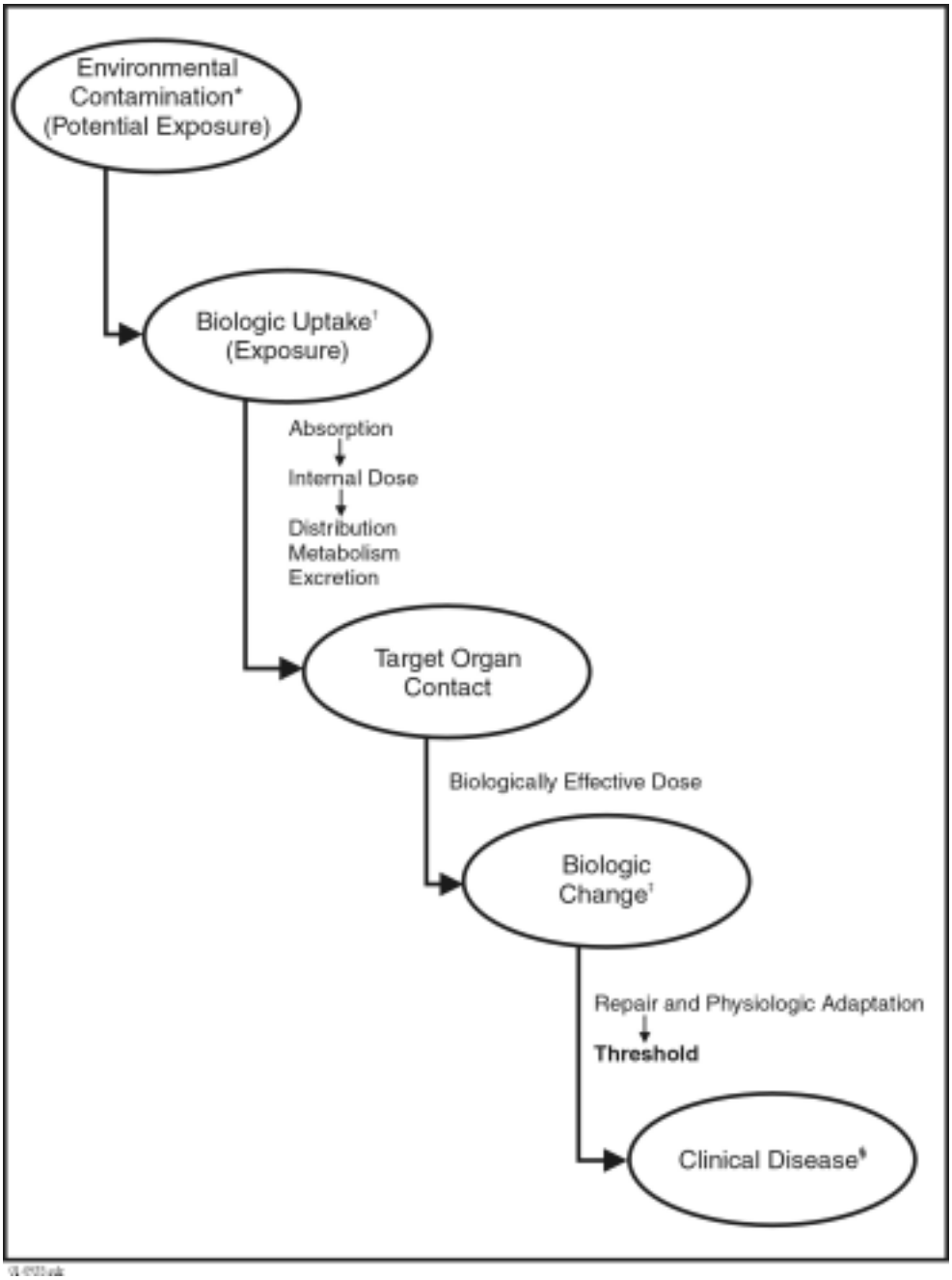

Figure 1. Exposure-Disease Model

*Contamination of environmental medium: air, water, soil, or food. Assessment tools: exposure history and environmental monitoring.

†Primary routes of exposure: respiratory, oral, and dermal. Secondary routes of exposure: breast milk, transplacental, nonplacental/intrauterine, and parenteral. Assessment tools: exposure history and biologic monitoring.

‡Assessment tools: biologic monitoring.

§Assessment tools: history, physical, biologic monitoring (to include advanced diagnostic testing of target organ), and specialty referral (if necessary).

Table 1. Clinical Characteristics of Acrodynia

System	Characteristic
Central nervous	Irritability Extreme photophobia (patient burrows head or covers eyes to block out light)
Cardiovascular	Hypertension Tachycardia
Gastrointestinal	Stomatitis with anorexia Colitis with diarrhea or constipation Salivation
Renal	Proteinuria Nephrotic syndrome progressing to renal failure in extreme cases
Dermal	Erythema of the palms, soles, and face Edema and desquamation of the skin of hands and feet Pruritus
Muscular/Skeletal	Hypotonia
Various	Gingivitis Diaphoresis Paresthesia Generalized pain

Table 2. Special Susceptibilities and Anticipatory Guidance About Opportunities for Hazardous Exposure by Developmental Stage

Developmental Stage	Developmental Characteristics	Vulnerabilities	Anticipatory Guidance
Preconception	—	Male and female parental reproductive systems Occupational, environmental, and vocational exposures Pharmaceuticals Substance abuse	Dietary advisories (mercury and PCBs)
Fetal	Rapid cell division Organogenesis Mother's internal environment	Dividing cells sensitive to transplacental carcinogens Developing reproductive system can lead to transgenerational effects Critical periods of organ development Immature blood-brain barrier Placenta as semipermeable membrane	Dietary advisories (mercury and PCBs) Occupational, environmental, and avocational exposures Take-home occupational exposures Pharmaceuticals and herbal and alternative remedies Substance abuse Topical insect repellents Baseline household environmental survey Maternal exposures during preparation of nursery and other remodeling (lead and volatile organic compounds)

Continued on page 56

The information in this table is adapted from Bearer (1995b). Anticipatory guidance is the education provided to parents or caretakers during a routine prenatal or pediatric visit to prevent or reduce the risk that their fetuses or children will develop a particular health problem (CDC 1997).

Table 2. Special Susceptibilities and Anticipatory Guidance About Opportunities for Hazardous Exposure by Developmental Stage *(Continued from page 55)*

Developmental Stage	Developmental Characteristics	Vulnerabilities	Anticipatory Guidance
Newborn (birth to 2 months)	Nonambulatory Restricted environment High calorie and water intake High air intake Highly permeable skin Alkaline gastric secretions (low gastric acidity)	Brain development - Immature blood-brain barrier - Synapse formation Lungs - alveolar development - lung fluid cleared by pulmonary lymphatic system High respiratory rate Skin very permeable with large surface-to-volume ratio Gastrointestinal tract: highly permeable, increased pH Immature detoxification capacity of liver, kidney, and digestive system Skin - Contaminants used or deposited on floor, especially household products, pesticides, and take-home occupational agents	Consider day care and home, indoor, and outdoor environments Ingestion - Breast milk - Infant formula (tap or well-water contaminants) Respiratory - Indoor air contaminants, especially those layering near the floor (e.g., mercury, pesticides, allergens, radon, asbestos, and take-home occupational agents). - Outdoor air pollutants, especially ozone and particulates

Continued on page 57

The information in this table is adapted from Bearer (1995b). Anticipatory guidance is the education provided to parents or caretakers during a routine prenatal or pediatric visit to prevent or reduce the risk that their fetuses or children will develop a particular health problem (CDC 1997).

Table 2. Special Susceptibilities and Anticipatory Guidance About Opportunities for Hazardous Exposure by Developmental Stage *(Continued from page 56)*

Developmental Stage	Developmental Characteristics	Vulnerabilities	Anticipatory Guidance
Infant/Toddler (2 months to 2 years)	Crawling and early walking Oral exploration Limited diet High intake of fruits and vegetables	Brain development - Immature blood-brain barrier - Synapse formation Lungs: alveolar development High respiratory rate Skin very permeable with large surface-to-volume ratio Small intestine avidly absorbs lead if diet deficient in iron and calcium Immature detoxification - capacity of liver, kidney, and digestive system	Consider day care and home, indoor, and outdoor environments Ingestion - Pesticides on fruit and vegetables - Tap water contaminants - Contaminants on floor and within easy reach, especially medicines, household products, pesticides, lead, and take-home occupational agents - Pica Respiratory - Indoor air contaminants, especially those layering near the floor (e.g., mercury, pesticides, allergens, radon, asbestos, and take-home occupational agents) - Outdoor air pollutants, especially ozone and particulates Skin - Contaminants on floor and within reach, especially household products, pesticides, and take-home occupational agents - Topical insect repellents

Continued on page 58

The information in this table is adapted from Bearer (1995b). Anticipatory guidance is the education provided to parents or caretakers during a routine prenatal or pediatric visit to prevent or reduce the risk that their fetuses or children will develop a particular health problem (CDC 1997).

Table 2. Special Susceptibilities and Anticipatory Guidance About Opportunities for Hazardous Exposure by Developmental Stage *(Continued from page 57)*

Developmental Stage	Developmental Characteristics	Vulnerabilities	Anticipatory Guidance
Young child (2 to 6 years of age)	Expanded environment, still includes significant time on floor Increased independence High intake of fruits and vegetables	Brain developing Lungs: alveolar development and increasing volume Small intestine avidly absorbs lead if diet deficient in iron or calcium Immature detoxification capacity of liver, kidney, and gastrointestinal system	Consider home, day care, preschool, and playmates' indoor and outdoor environments Ingestion - Pesticides on fruit and vegetables - Tap water contaminants - Contaminants on floor and within easy reach, especially medicines, household products, pesticides, lead, and take-home occupational agents - Pica Respiratory - Indoor air contaminants, especially those layering near the floor (e.g., mercury, pesticides, allergens, radon, asbestos, and take-home occupational agents) - Outdoor air pollutants, especially ozone and particulates Skin - Contaminants on floor and within reach, especially household products, pesticides, and take-home occupational agents - Topical insect repellents

Continued on page 59

The information in this table is adapted from Bearer (1995b). Anticipatory guidance is the education provided to parents or caretakers during a routine prenatal or pediatric visit to prevent or reduce the risk that their fetuses or children will develop a particular health problem (CDC 1997).

Table 2. Special Susceptibilities and Anticipatory Guidance About Opportunities for Hazardous Exposure by Developmental Stage *(Continued from page 58)*

Developmental Stage	Developmental Characteristics	Vulnerabilities	Anticipatory Guidance
School-aged child (6 to 12 years)	Increased number of environments and less supervised play: school, playground, friends' houses	Brain developing Lungs: increasing volume	Consider home, school, friends, and afterschool programs' indoor and outdoor environments Ingestion - Tap water - Food Respiratory - Indoor and outdoor air quality - Hazards associated with hobbies and school crafts - Take-home occupational hazards Skin - Hazards associated with hobbies and school crafts - Take-home occupational hazards - Topical insect repellents

Continued on page 60

The information in this table is adapted from Bearer (1995b). Anticipatory guidance is the education provided to parents or caretakers during a routine prenatal or pediatric visit to prevent or reduce the risk that their fetuses or children will develop a particular health problem (CDC 1997).

Table 2. Special Susceptibilities and Anticipatory Guidance About Opportunities for Hazardous Exposure by Developmental Stage *(Continued from page 59)*

Developmental Stage	Developmental Characteristics	Vulnerabilities	Anticipatory Guidance
Adolescent (12 to 18 years)	Puberty Accelerated growth Experimentation with controlled substances Independence and exposure to multiple environments Possible employment, work in family business, or training in hazardous trades	Brain and lungs continue to develop Muscles and bones grow rapidly Gonad maturation Breast development Ova and sperm maturation	Consider home, school, friends, occupational, and trade school environments Ingestion - Tap water - Food - Occupational hazards ingested because of poor poor hygiene - Substance abuse Respiratory - Indoor and outdoor air quality - Occupational and trade school hazards - Take-home occupational exposures - Hazards associated with hobbies and school crafts - Substance abuse Skin - Occupational and trade school hazards - Take-home occupational exposures - Hazards associated with hobbies and school crafts - Topical insect repellents

The information in this table is adapted from Bearer (1995b). Anticipatory guidance is the education provided to parents or caretakers during a routine prenatal or pediatric visit to prevent or reduce the risk that their fetuses or children will develop a particular health problem (CDC 1997).

Table 3. Differences in Children and Adults

	Infants	Children	Teens	Adults	Reference
Surface area: body mass ratio (m^2/kg)*	Newborn 0.067	Young child 0.047	Older child 0.033	Adult 0.025	Silvaggio and Mattison (1993)
Respiratory ventilation rates	Infant	–	–	Adult	Silvaggio and Mattison (1993)
Respiratory volume (mL/kg/breath)†	10	–	–	2	
Alveolar surface area (m^2)‡	3	–	–	10	
Respiration rate (breaths/min)§	40	–	–	75	
Respiratory minute ventilation	–	–	–	–	
rate¶	133		–	2	
Drinking water (tap)	<1 year	1–10 years	11–19 years	20–64 years	Snodgrass (1992)
Mean intake (mL/kg/day)**	43.5	35.5	18.2	19.9	
Fruit consumption (g/kg/day)††	<1 year	3–5 years	12–19 years	40–69 years	U.S. Environmental Protection Agency (1997a)
Citrus fruits	1.9	2.6	1.1	0.9	
Other fruits (including apples)	12.9	5.8	1.1	1.3	
Apples	5.0	3.0	0.4	0.4	
Soil ingestion (mg/day)‡‡	–	5,000§§	–	Adult	U.S. Environmental Protection Agency (1997b)
Pica child	–	2.5 years	–	–	
Outdoor	–	50	–	20¶¶	
Indoor	–	60	–	0.4	
Differences in gastrointestinal absorption of lead	0–2 years 42%–53%	2–6 years 30%–40% 6–7 years 18%–24%	– –	Adult 7%–15%	U.S. Environmental Protection Agency (1997c)

Adapted from Selevan et al. (2000).
*Square meters per kilogram.
†Milliliters per kilogram per breath.
‡Per square meter.
§Breaths per minute.
¶Milliliters per kilogram body weight per square meter lung surface area per minute.
**Milliliters per kilogram per day.
††Grams per kilogram per day.
‡‡Milligrams per day.
§§ATSDR (2001a).
¶¶Gardening for adults.

Table 4. Sources of Common Environmental Hazards

Hazards and Routes of Exposure*	Outdoor Air and Soil	Building Structure	Furnishings and Finishings	Mechanical Systems	Occupant Activities/ Source	Tap Water	Food
Asbestos (R, D, O)	Hazardous waste sites Abrasion of brake linings Building demolition	Sprayed-on fireproofing Roofing and siding Thermal insulation	Ceiling and floor tiles Textured wall and ceiling finishing	Gaskets Pipe and furnace insulation	Selected consumer products Take-home occupational exposures	Asbestos cement water pipes	—
Biologic (R, O)	Local flora Molds Animal droppings Insects Microorganisms Composting	Wet insulation Wet carpet Wet wallboard	Carpet Fleecy furnishings Bedding (All worse when damp)	Humidifiers Condensate pans in air conditioners and refrigerators Moist, dirty ductwork	Communicable occupational infections Respiratory droplet Body fluids	Contamination at source, distribution system, tap, storage containers	Contamination at source, transportation, processing, storage, preparation
Combustion products (R)	Combustion engines Incinerators Forest fires Residential and industrial furnaces	House fires	House fires	Malfunctioning and poorly vented heating and cooking devices	Tobacco smoking Environmental tobacco smoke	—	—
Lead (O, R)	Hazardous waste Industrial effluent Exterior paint Demolition and sandblasting Lead pipes	Lead plumbing fixtures and solder	Lead paint	—	Hobbies Folk remedies Remodeling Consumer products Take-home occupational exposures	Water service mains, plumbing before 1978	Imported canned food Pottery glazes
Mercury (R)	Hazardous waste Industrial emissions Food source (fish)	Old household paints before mercury ban Industrial or marine paints	Thermometers, thermostats, and medical instruments	Take-home occupational agents Folk remedies Hobbies	—	—	—
Pesticides (O, D)	Spray drift from foundation exterminations Lawn and agricultural residue Hazardous waste	Treated building materials	Carpets Wall coverings Shower curtains Paints	Contaminated ductwork Biocides in humidifiers and air conditioners	Consumer products, including aerosols, shampoos, pet collars, hanging strips, repellents Take-home occupational exposures	Ground and surface water contamination from agricultural and lawn chemicals	Residues from agricultural applications Bioconcentrated persistent organochlorines in fish, meat, and cow's and breast milk

Continued on page 63

Table 4. Sources of Common Indoor Environmental Hazards *(Continued from page 62)*

Hazards and Routes of Exposure	Outdoor Air and Soil	Building Structure	Furnishings and Finishings	Mechanical Systems	Occupant Activities/ Source	Tap Water	Food
Polychlorinated or brominated biphenyls	—	—	—	—	—	—	Bioconcentrated in food chain, including breast milk Occupational exposures leading to breast milk contamination
Radon (R)	Soil Well water Natural gas	Stone Brick Cement block	—	—	—	Well	—
Respirable particulates (R)	Wind-blown soil Industrial emissions Fossil fuel combustion Forest and brush fires Volcanic eruptions	Demolition of wall and internal structures	Fleecy furnishings, including shag carpets, upholstered furniture	Poorly ventilated and malfunctioning heating and cooling devices Humidifiers Degrading fiberglass ductwork	Tobacco smoke Remodeling Hobbies Cleaning	—	—
Volatile organic compounds (R, O, D)	Underground storage tanks Hazardous waste Industrial emissions Tap water pollution Inadequately aired dry cleaning	Composition board Urea formaldehyde insulation Adhesives Caulks Additives to fiberglass insulation Plastics	Spackling compound Paints and other surface coatings Cabinetry Furniture Carpets Plastics	Fugitive fossil fuel emissions Office machines Lubricants Duct sealants and cleaners	Use and storage of consumer products Cosmetics Hobbies Tobacco smoke Human metabolism	Hazardous waste Leaking underground storage tanks	Breast milk contaminated as the result of occupant activites

*R: respiratory; D: dermal; O: oral. Routes of exposure are listed for each contaminant in the usual order of importance.

Table 5. Basic Environmental Database

Name: _____ Date Completed: _____

Address of this home: _____

Date moved in: _____

Parents and other adults in the home: _____

_____

Current jobs of occupants (including how long in job): _____

_____

_____

1. Do you think you or a family member have a health problem caused by your home environment?
   _____ Yes _____ No
2. Building type:
   _____ Single-family, detached _____ Single-family, condo
   _____ Mobile home _____ Multifamily
3. Features: Single story _____ Multistory _____ Attached garage
4. Lowest level of home:
   _____ On-grade level _____ Below-grade basement _____ Crawl space
   _____ Dirt floor _____ Finished floor (material:)
5. Ownership: _____ Self _____ Other family member _____ Tenant
6. Year built:_____
   Location:
   _____ Industrial or agricultural pollution sources nearby (<1 mile) _____ Municipal landfills
   _____ Commercial orchards, fields _____ Livestock _____ Underground tanks
   _____ Hazardous waste site _____ Industry or business
7. Does anyone living in the household smoke tobacco products? Yes No
   If *yes*, how many smokers at home?
   Is there a child in your family exposed to smoke at day care or in cars? Yes No
8. Have there been renovations, interior decorating, or new furniture in the home in the last 3 years?
   Yes _____ No_____

If yes, please describe: _____

_____

Continued on page 65

Table 5. Basic Environmental Database *(Continued from page 64)*

9. How do you heat your home?
   a. Primary energy source:
   _____ Oil _____ Natural gas _____ Propane Coal
   _____ Wood _____ Electric heat pump _____ Solar
   b. Distribution of heat:
   _____ Forced air _____ Steam _____ Hot water Radiant
   c. Do you use another heat source? Yes No
   d. Secondary energy source(s):
   _____ Oil Natural gas _____ Propane Coal
   _____ Wood Electric Heat pump Solar
   _____ Kerosene
   e. Location of secondary heat source: _____
   f. If this heat source burns fuel, is it vented outdoors? Yes No
   g. If you use a wood stove or fireplace, how often do you use it?
   _____ Rarely Every week of winter Every day of winter

10. Do you have any of the following equipment or appliances?
   Air filter (Describe)
   Humidifier (Describe)
   Air conditioner (Describe)
   Gas appliances: Kitchen stove Hot water heater Dryer

11. Do you or a family member have a hobby or home business that might involve
   _____ biologic agents _____ chemicals _____ dusts _____ fibers _____ fumes
   _____ radiation _____ loud noise _____ vibration _____ metals _____ paints
   _____ extreme heat or cold
   For those that apply, please list and/or describe the hobby: _____
   _____
   _____

12. Is any part of your home damp or have you had a major leak or flood in your house?
   Yes No
   If *yes*, please describe _____
   _____

13. Have any pesticides or herbicides been used in or around your home within the last year (including on pets)?
   Yes No
   If *yes*, please describe _____
   _____

Continued on page 66

Table 5. Basic Environmental Database *(Continued from page 65)*

14. Have you ever tested your home for radon? Yes No

If *yes*, in which season of the year did you test your home?

Spring Summer Fall Winter

In which part of your home did you conduct the test? _____

What was the length of test? _____

What levels of radon, if any, were found? _____

Have any radon reduction measures been taken? Yes No

If *yes*, please describe _____

_____

If radon reduction measures have been taken, have the levels been rechecked?

Yes No

If *yes*, were the radon levels reduced?

Yes No

15. Where do you get your water supply?

Bottled Municipal Private well or spring

If you get your water from a well, was the well dug or drilled?

When was the well last tested for contaminants? _____

What were the results? _____

_____

_____

16. Do you use a water treatment device in your well (e.g., filter or softener)?

Yes No

If *yes*, please describe _____

_____

17. Do you have a septic system or do you use municipal sewers?

If you use a septic system, when was the tank last pumped? _____

18. Does any member of the household work at a job that might result in bringing chemicals home on his or her clothes or shoes? Yes No

If *yes*, please describe _____

_____

19. Have industrial chemicals been brought home from the workplace for domestic use?

Yes No

If *yes*, please describe _____

_____

_____

Table 6. Environmental Hazards Checklist for Home Assessment*

Family Name _____

Address _____

_____

Housing

Type of Housing? _____ Ownership?

How old? _____ Rental _____

Condition? _____ Owner-occupied _____

Public housing _____

Renovation/repairs occurring? Yes No Describe: _____

Existing rodents/insects? Yes No Describe: _____

Existence of molds/fungi? Yes No Describe: _____

What source of drinking water? Describe: _____

Heating Source

Uses gas stoves/ovens for heating? Yes No

Adequate ventilation? Yes No

Uses fireplaces/woodburning stoves? Yes No What is burned? _____

Wood smell indoors? Yes No

Evidence of smoke/soot? Yes No

Uses kerosene heaters? Yes No

Environmental Tobacco Smoke

Household members smoke? Yes No

Regular visitors smoke? Yes No

Smoking allowed in car? Yes No

Indoor Air Pollution—Formaldehyde and Asbestos

Sources of formaldehyde? Yes No Describe: _____

(particle board, urea in foam insulation, other)

Potential asbestos hazards? Yes No Describe: _____

(friable pipe/boiler insulation, old vinyl linoleum, wall board repair, home renovation or repairs)

Continued on page 68

Table 6. Environmental Hazards Checklist for Home Assessment *(Continued from page 67)*

Air Pollution—Toxic Organic Hydrocarbons			
Uses cleaners/polishers/air fresheners/disinfectants		Yes	No
Uses glues/solvents/varnishes/building materials?		Yes	No
Where are these materials stored? _____			
Pest/Mold/Fungi Control			
Home garden	Yes	No	
Use of pesticides outdoors	Yes	No	
Evidence of rodents/insects	Yes	No	
Use of pesticides indoors?	Yes	No	
Use of pesticides on children?	Yes	No	What type? _____
Use of pesticides on pets?	Yes	No	What type? _____
Is re-entry after pesticide use according to instructions?		Yes	No
Evidence of molds/fungi?	Yes	No	

*Adapted from Balk et al. (1999).

Appendix B: Important Issues Regarding Mercury

Direct Biologic Indicators and Treatment—Elemental Mercury

Urine mercury levels might be reported in different units of measure (e.g., micrograms per gram creatinine and micrograms per liter) that are not equivalent. This should be considered when interpreting results in children because creatinine levels in children differ by age due to developmental changes. In addition, fluid intake in children differs from fluid intake in adults, which can also affect the volume when converting values into different units. Other factors, such as chronic illness in both children and adults, might need to be considered when interpreting laboratory results. The method used by the laboratory to report values should be known. For example, the National Center for Environmental Health (NCEH) laboratory reports values in micrograms per gram creatinine, which is adjusted per sample by direct urine creatinine measurement. NCEH considers direct creatinine adjustment of urine mercury values imperative for proper interpretation. Age-specific reference ranges for urine volume and urine creatinine excretion are published if a conversion between the two units of measure is necessary (Behrman et al. 1996). However, interpretation should be done in consultation with a specialist who has experience managing cases of childhood mercury poisoning.

Treatment Considerations

For children exposed to mercury, no current standard medical guideline exists for treatment on the basis of mercury urine levels and clinical signs and symptoms. For this reason, the interpretation of laboratory values for urine mercury and choice of treatment regimen, if necessary, should be determined in consultation with a specialist in pediatric environmental medicine who has experience managing patients with mercury poisoning. In situations where a 24-hour urine mercury specimen is not easily attainable for a young child, such as in an outpatient clinic setting, a spot mercury level can be obtained for screening purposes. It is important to remember, however, that for this type of sample, the units of measure used in the reporting of laboratory results and the methods of adjustment for the concentration of the urine (e.g., using specific gravity versus amount of creatinine present) are not standardized across laboratories and, therefore, this should be considered when interpreting reported laboratory values.

Treatment

Removal from exposure is the first step, followed by an assessment of the child's clinical condition to ensure that the patient is stable. Chelation has been used to reduce body burden of elemental mercury, although whether it reduces toxic effects or speeds recovery in mercury-poisoned children remains unclear (Fullilove 2001). Chelation should only be used for symptomatic patients with known mercury exposure, and only after consideration of the risk and benefits by a specialist experienced in the use of chelators and in consultation with the patient or family. Mercury poisoning should be treated in consultation with experts in the field of environmental toxicology and pediatricians who have experience in management of children with this exposure.

Succimer, which has been used as an oral chelating agent in the treatment of lead poisoning, also increases urinary mercury excretion. However, its efficacy and long-term benefits are uncertain, thus classifying this treatment mode as experimental. Adverse side effects from succimer include abdominal distress, transient rash, increased liver function, and neutropenia. Other agents available for treatment, but not yet approved in the United States, might be more efficacious at removing mercury (e.g., 2,3-dimercaptopropan-1-sulfonate). Ethylenediamine tetra-acetate and

penicillamine are not very effective for mercury. When treating a patient with similar symptoms, consult with a pediatric toxicologist at your local poison control center or at a Pediatric Environmental Health Specialty Unit (Appendix D, page 74).

Different Forms of Mercury and Differing Health Effects

Mercury occurs naturally in the environment and exists in several forms, which can be organized under three headings: metallic mercury (i.e., elemental mercury—Hg^0, quicksilver), inorganic mercury (i.e., Hg^{+1} [mercurous salts] or Hg^{+2} [mercuric salts]), and organic mercury (i.e., methyl-, ethyl-, and phenylmercury). Because mercury's absorption and metabolism depend on its chemical and physical form, it is important to determine the form of mercury to which an individual is exposed. Different forms of mercury can have differing health effects (e.g., absorption and metabolism of different forms of mercury vary and, therefore, have different effects on the nervous system). When metallic mercury vapors are inhaled, they readily enter the bloodstream and cross the blood-brain barrier. Inhaling or ingesting large amounts of methylmercury also results in some of the mercury crossing the blood-brain barrier and affecting the nervous system. Inorganic mercury salts, such as mercuric chloride, do not cross the blood-brain barrier like methylmercury or metallic mercury vapor do. Mercury affects other systems in addition to the nervous system (ATSDR 1999, 1992).

Dental Amalgams

The ATSDR *Toxicological Profile for Mercury* (ATSDR 1999) states that

> One way in which people are routinely exposed to extremely small amounts of mercury is through the gradual (but extremely slow) wearing-away process of dental amalgam fillings, which contain approximately 50% mercury. The amount of mercury to which a person might be exposed from dental amalgams would depend on the number of amalgams present as well as other factors. The Centers for Disease Control and Prevention (CDC) has determined that dental amalgam fillings do not pose a health risk, although they do account for some mercury exposure in those having such fillings. People who frequently grind their teeth or often chew gum can add to the small amount of mercury normally released from those fillings over time.... The practice of having all your dental amalgam fillings replaced with nonmercury filling materials just to remove the possibility of mercury exposure is not recommended by ATSDR. In fact, the removal of the mercury amalgam fillings would actually expose the patient to a greater amount of mercury, at least for a while. There are other sources of mercury that may increase your overall exposure, such as the amount of fish consumed per week or an exposure to mercury from a nearby hazardous waste site or incinerator.

Thimerosal

Since the 1930s, some, but not all, of the vaccines routinely recommended for children have contained small amounts of thimerosal, a mercury-containing preservative. In July 1999, the U.S. government asked vaccine manufacturers to eliminate or reduce, as expeditiously as possible, the mercury content of their vaccines to avoid any possibility of infants who receive vaccines being exposed to more mercury than is recommended by federal guidelines. For additional information, see *Mercury and Vaccines (Thimerosal)* (CDC 2001), *Thimerosal in Vaccines: An Interim Report to Clinicians* (American Academy of Pediatrics 1999), and Goldman et al. (2001).

Fish Consumption

For those populations that rely on local fish for a major portion of their diet, a complete exposure history should include questions exploring subsistence fishing as a possible source of mercury exposure. Bioaccumulation occurs when mercury in lake and stream sediments is converted by bacteria into organic mercury compounds that accumulate in the food chain. Physicians living in active fishing areas with fish advisories related to mercury should ask women and children if their consumption of fish is in accordance with state-issued fish advisories. In general, freshwater fish have higher levels of contaminants than saltwater fish, but not always. Increased methylmercury content has been found in the larger ocean fish (e.g., tuna, swordfish, and shark) because of naturally occurring and manmade sources of mercury pollution. In the 1950s, when pregnant women in Minimata Bay, Japan, ingested fish with high levels of methylmercury, the result was at least 30 cases of infantile cerebral palsy (Klaassen 1996). As listed in the *Handbook of Pediatric Environmental Health* (Etzel and Balk 1999), to reduce hazards from fish consumption, individuals can be counseled to eat nonpredator fish rather than predator fish (e.g., shark, swordfish, and tuna); to eat small rather than large game fish; and to eat fewer fatty fish (e.g., carp, catfish, and lake trout), which accumulate higher levels of chemical toxicants. Emphasize to women of childbearing age, pregnant women, nursing mothers, and parents of young children the need to follow fish advisory guidelines. Fish advisories can be obtained from state health, environmental, and conservation departments (ATSDR 1999, 1992; EPA and ATSDR 2001).

Appendix C: Summary of Questions for an Environmental History

Issue: ***The child's home, school, or day-care center might expose him or her to potential toxicants.***

- Do you live in an apartment, house, or mobile home?
- On what level of your dwelling is the child's room located?
- What are the age and condition of your home?
- How is your home heated?
- Do you have a fireplace or a wood stove?
- Do you use pesticides inside or outside your home (including use on children and pets)?
- What are the hobbies of your child and other family members?
- Is your home (day-care center, etc.) near a polluted body of water, industrial plant, commercial business, or dump site?

Issue: ***Family members' jobs might involve exposure to contaminants.***

- What is your occupation?
- What is your spouse's occupation?
- Do other members of the family have jobs?
- If so, what are they?
- For teenagers:

  Do you work?

  What kind of job do you have and what hours do you work?

Issue: ***The child might be exposed to tobacco smoke.***

- Do you smoke tobacco products?

  If *yes*, do you smoke in your home?
- Does your spouse, other family member, or babysitter smoke?
- If you take your child to a babysitter, does he or she smoke at home?
- Do visitors smoke in your home?
- Does anyone smoke in your car?

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Issue: ***The child might eat food contaminated with environmental toxicants.***

- For breastfeeding mothers:

  Have you tested your water supply for lead?
- If not and you make the baby's formula with tap water, what procedure do you follow?

  Do you ever use hot tap water or water from instant hot taps or refrigerator taps to make the formula?
- Do you wash fruits and vegetables before giving them to your child?

  What do you wash them with?

  What kind of produce do you usually buy? Organic? Local? In season?
- Does the child live with an adult whose job or hobby involves exposure to lead?

Issue: ***The child might be at high risk for lead poisoning.***

- Is there a brother, sister, housemate, or playmate being followed or treated for lead poisoning (i.e., blood lead 15 µg/dL)?
- Does the child live with an adult whose job or hobby involves exposure to lead?
- Does the child live near an active lead smelter, battery recycling plant, or other industry likely to release lead?
- Do you use home remedies or pottery from another country?

Adapted from Balk (1996).

When To Introduce Environmental Questions

Topics	The Right Time
Home renovation, smoking, breast and bottle issues	Prenatal period
Environmental tobacco smoke	When child is 2 months old
Poison exposures, including household pesticides and lead poisoning	When child is 6 months old
Arts-and-crafts exposures	Preschool period
Occupational exposures, exposures from hobbies	When patient is a teenager
Lawn and garden products, lawn services, scheduled chemical applications	Spring and summer
Wood stoves and fireplaces, gas stoves	Fall and winter

Appendix D: Environmental Health Concerns: Resources and Sources of Information

General Resources

- General Resources for Parents and Caregivers
- General Resources for Clinicians

Consultation Sources

- Government Agencies
- Association of Occupational and Environmental Clinics
- Pediatric Environmental Health Specialty Units
- National Institute for Occupational Safety and Health's Education and Research Center Grants
- American Association of Poison Control Centers
- State and Local Health Departments

General Resources

General Resources for Parents and Caregivers

Agency for Toxic Substances and Disease Registry. 2002. Child health web page. Atlanta: US Department of Health and Human Services. Available from: URL: www.atsdr.cdc.gov/child/ochchildhlth.html.

Children's Environmental Health Network. 2000. The household detective primer: how to protect your children from toxins in the home. Princeton (NJ): Children's Environmental Health Network. Available from: URL: www.cehn.org.

Needleman HL, Landrigan PJ. 1994. Raising children toxic free: how to keep your child safe from lead, asbestos, pesticides, and other environmental hazards. New York: Farrar, Straus and Giroux.

Schoemaker JM, Vitale CY. 1991. Healthy homes, healthy kids—protecting your children from everyday environmental hazards. Washington (DC): Island Press.

Upton AC, Graber E. 1993. Staying healthy in a risky environment: the New York University Medical Center family guide. New York: Simon & Schuster.

General Resources for Clinicians

Children's Environmental Health Network. 1997. Resource guide on children's environmental health. Washington (DC): Children's Environmental Health Network.

Goldman R, Shannon M, Woolf A. 1999. Pediatric environmental health history [on CD-ROM]. Boston: Pediatric Environmental Health Unit Cambridge Hospital and Children's Hospital.

Textbooks

Behrman RE, Kliegman RM, Arvin AM, Nelson WE, editors. Nelson textbook of pediatrics, 15th edition. Philadelphia: W.B. Saunders Company; 1996.

Burgess WA. Recognition of health hazards in industries: a review of materials and processes, 2nd edition. New York: John Wiley & Sons, Inc.; 1995. *Contains work-site analyses that facilitate recognition of health problems in design and operation of industrial processes. In addition to identifying health issues, provides information on control measures.*

Encyclopedia of occupational health and safety, 3rd edition. International Labor Office/Boyd Printing; 1991. *Extensive information on all aspects of occupational health and safety, including occupational diseases, hazards, prevention, institutions. Two-volume set of more than 3,600 pages. 4th edition, with a CD-ROM version, 1997.*

Harris RL. Patty's Industrial Hygiene and Toxicology, 5th edition. New York: John Wiley & Sons Inc.; 2000. *Guide to principles and practice of industrial hygiene, hazard evaluation and control, toxic exposures, and similar topics.*

Hathaway GJ, Proctor NH, Hughes JP, Fischman ML. Proctor and Hughes' chemical hazards in the workplace, 4th edition. New York: John Wiley & Sons; 1996. *This is the classic text on more than 500 chemicals that may result in workplace exposure. Provides definitive information on effects of exposure and on treatment approaches.*

LaDou J, editor. Occupational and environmental medicine, 2nd edition. Stamford (CT): Appleton & Lange; 1997.

Manuele FA. On the practice of safety, 2nd edition. New York: John Wiley & Sons; 1997. *Discussion of basic principles and aspects of safety as an evolving discipline.*

Rogers B. Occupational health nursing—concepts and practice. Philadelphia: W.B. Saunders Co.; 1994. *Primary reference for occupational health nursing principles and practice. Provides clinical nursing guidelines for common occupational health problems.*

Rom WM, editor. Environmental and occupational medicine, 3rd edition. Philadelphia: Lippincott-Raven, 1998. *Another helpful reference that considers environmental as well as occupational health problems.*

Rosenstock L, Cullen MR, editors. Textbook of clinical occupational and environmental medicine. Philadelphia: Saunders, 1994. *Useful reference for both environmental and occupational medicine concerns.*

Vincoll J. Basic guide to accident investigation and loss control. New York: John Wiley & Sons; 1994. *Provides information on safety principles and techniques, including preparation of safety system applications.*

Wald P, Stave G. Physical and biological hazards in the workplace, 2nd edition. New York: John Wiley & Sons; 2001. *Focuses on physical and biological hazard in the workplace and serves as a companion to* Proctor and Hughes' Chemical Hazards in the Workplace. *Available on CD-ROM.*

Zenz C, Dickerson OB, Horvath EP, editors. Occupational medicine, 3rd edition. St. Louis (MO): Mosby, 1994. *Information on occupational medicine principles and practice as well as on specific hazards and agents; 1,336 pages.*

Web and Other Sources

Abelsohn A, Sanborn M. Environmental health in family medicine. Available at URL: www.ijc.org/boards/hptf/modules/content.html. *Set of six full-text modules (lead, outdoor air quality, indoor air quality, pesticides, clusters of disease [water quality], and persistent organic pollutants). The modules are based on clinical cases that can be used for self-learning, or for teaching residents or practicing physicians. Also available on CD-ROM.*

National Library of Medicine's Medline. Available via NLM's Health Information Web site at URL: www.nlm.nih.gov/hinfo.html. *Medline is the National Library of Medicine's premier bibliographic database covering the fields of medicine, nursing, dentistry, veterinary medicine, the health care system, and the preclinical sciences.*

TOMES Plus. Available from Micromedix, Inc. 6200 South Syracuse Way, Suite 300, Englewood, CO 80111-4740 (USA and Canada phone: 1-800-525-9083 or 303-486-6400; International Department phone: +1 303 486-6444); Web page: www.micromedex.com/products/tomesplus. *The TOMES System provides rapid, easy access to medical and hazard information needed for managing chemicals safely in the workplace, evaluating exposures, quick response to emergency situations, and regulatory compliance. The system includes MEDITEXT, medical managements; HAZARDTEXT, hazard managements with initial response information; and INFOTEXT, Documents with regulatory listings and other general information.*

Specific information, such as reports by the Hazard Evaluation and Technical Assistance Branch of the National Institute for Occupational Safety and Health, is available through the National Technical Information Service (NTIS) database at www.ntis.gov.

Professional Journals

Professional journals provide information on research, clinical practice, prevention techniques, new hazards, and similar topics. A few examples include the following:

- *American Industrial Hygiene Journal*, published by the American Industrial Hygiene Association.
- *American Journal of Industrial Medicine*, published by John Wiley & Sons, Inc.
- *Annals of Occupational Hygiene*, published by Elsevier Science Publishers.
- *Archives of Environmental Health*, published by Heldref Publications.
- *Environmental Epidemiology and Toxicology*, published by Stockton Press.
- *Environmental Health Perspectives*, published by the National Institute of Environmental Health Sciences.
- *International Archives of Occupational and Environmental Health*, published by Springer-Verlag.
- *Journal of Occupational and Environmental Medicine*, published by Williams & Wilkins.
- *Journal of Toxicology—Clinical Toxicology*, published by Marcel Dekker.
- *Occupational and Environmental Medicine* (formerly *British Journal of Industrial Medicine*), published by BMJ Publishing Group.
- *Scandinavian Journal of Work, Environment and Health*, published by the Finnish Institute of Occupational Health.

Professional Organizations

Web sites for a few professional organizations include the following:

- American Academy of Pediatrics: www.aap.org.
- American Association of Occupational Health Nurses: www.aaohn.org.
- American College of Medical Toxicology (ACMT): www.acmt.net.
- American College of Occupational and Environmental Medicine: www.acoem.org.
- American College of Preventive Medicine: www.acpm.org.
- American Industrial Hygiene Association: www.aiha.org.
- American Society of Safety Engineers: www.asse.org.
- Children's Environmental Health Network: www.cehn.org.
- Physicians for Social Responsibility: www.psr.org.

Government Agencies

Essentially every national agency with health and safety information or activities has an Internet Web site. These sites can be found by direct search using the agency's name or initials. A few examples include

- Agency for Toxic Substances and Disease Registry: www.atsdr.cdc.gov.
- Centers for Disease Control and Prevention: www.cdc.gov.
- National Institute of Environmental Health Sciences: www.niehs.nih.gov.
- National Institute for Occupational Safety and Health: www.cdc.gov/niosh.

- U.S. Department of Agriculture: www.usda.gov.
- U.S. Department of Housing and Urban Development: www.hud.gov.
- U.S. Environmental Protection Agency: www.epa.gov.
- U.S. Food and Drug Administration: www.fda.gov.
- U.S. Occupational Safety and Health Administration (OSHA): www.osha.gov.

Consultation Sources

Government Agencies

- Agency for Toxic Substances and Disease Registry: emergency response line 404-498-0120; 1-888-42-ATSDR (1-888-422-8737).
- Centers for Disease Control and Prevention Hotlines:

  National AIDS Hotline 1-800-342-2437.

  National HIV/AIDS Hotline (Spanish) 1-800-344-7432.

  National Immunization Hotline (English) 1-800-232-2522.

  National Immunization Hotline (Spanish) 1-800-232-0233.

  National STD Hotline 1-800-227-8922.

  SafeUSA Federal Safety 1-888-252-7751.

  Traveler's Health 1-877-394-8747.
- National Institute of Environmental Health Sciences: 919-541-3345.
- National Institute for Occupational Safety and Health: 1-800-35-NIOSH (1-800-356-4674).
- U.S. Environmental Protection Agency (National Response Center for environmental emergencies): 1-800-424-8802.
- U.S. Food and Drug Administration: 1-888-INFO-FDA (1-888-463-6332).
- U.S. Occupational Safety and Health Administration (OSHA): 1-800-321-OSHA (1-800-321-6742) TTY 1-877-889-5627.

Association of Occupational and Environmental Clinics

The Association of Occupational and Environmental Clinics (AOEC) is a network of more than 60 clinics and more than 250 individuals committed to improving the practice of occupational and environmental medicine through information sharing and collaborative research.

The primary goal of AOEC is to facilitate the prevention and treatment of occupational and environmental illnesses and injuries through collaborative reporting and investigation of health problems. AOEC members develop curriculum materials in occupational and environmental health and provide Education Activities (EA) programs for primary care practitioners and others.

For more information and a listing of AOEC clinics in your area, contact the AOEC office:

1010 Vermont Avenue, NW #513
Washington, DC 20005
Telephone: 202-347-4976
Fax: 202-347-4950
Web site: www.aoec.org.

Pediatric Environmental Health Specialty Units

The Pediatric Environmental Health Specialty Units (PEHSUs) are a resource for pediatricians, other health care providers, parents, teachers, the general public, and U.S. Environmental Protection Agency (EPA) and ATSDR staff members nationwide. PEHSUs provide medical education and training, consultation, and clinical specialty referrals. Health care providers can use this resource when responding to suspected clinical presentations, known exposures, and in community settings.

Ten PESHUs are now in operation across the United States; contact information for each unit is listed below. Check AOEC's Web site (www.aoec.org/pesu.htm) for the most up-to-date information on the units.

Massachusetts:

Pediatric Environmental Health Center, Children Hospital, Boston

Telephone: 1-888-Child14

Web site: www.childrenshospital.org (In the "find" box, enter the key word "environmental")

Washington State:

Pediatric Environmental Health Specialty Unit, Harborview Medical Center, Seattle

Telephone: 1-887-KID-CHEM (toll-free west of the Mississippi River) or 206-526-2121

Web site: www.depts.washington.edu/oemp/grants/PEHSU.html

New York:

Mt. Sinai Pediatric Environmental Health Unit/Mt. Sinai-Irving J. Selikoff Center for Occupational and Environmental Medicine, New York

Telephone: 212-241-6173

Web site: www.mssm.edu/cpm/peds_environ.shtml

Illinois:

Great Lakes Center for Children's Environmental Health, Cook County Hospital, Chicago

Telephone: 1-800-672-3113 (toll-free) or 312-633-5310

Web site: www.uic.edu/sph/glakes/kids

Georgia:

The Southeast Pediatric Environmental Health Specialty Unit at Emory University, Atlanta

Telephone: 1-877-337-3478 (1-877-33PEHSU)

Web site: www.sph.emory.edu/PEHSU

California:

University of California-San Francisco (UCSF)/University of California-Irvine (UCI) Pediatric Environmental Health Specialty Unit

Telephone: 1-415-206-4320 (for both sites)

Web site: www.ucsf.edu/ucpehsu

Texas:

Southwest Center for Pediatric Environmental Health, University of Texas Health Center at Tyler, Tyler, Texas

Telephone: 1-888-901-5665 (toll-free)

Web site: research.uthct.edu/swcpeh/

Washington, District of Columbia:

Mid-Atlantic Center for Children's Health and the Environment (MACCHE), George Washington University, Washington, District of Columbia

Telephone: 1-866-MACCHE1 (1-866-622-2431)
Web site: www.health-e-kids.org

Colorado:

Rocky Mountain Regional Pediatric Environmental Health Specialty Unit, National Jewish Medical and Research Center, Denver

Telephone: 1-877-800-5554 (toll-free)
Web site: rmrpehsu.org

Iowa:

Midwest Regional Pediatric Environmental Health Center, University of Iowa, Iowa City, Iowa

Telephone: 1-866-697-7342
Web site: www.uihealthcare.com/depts/pediatricenvironmentalhealth/index.html

National Institute for Occupational Safety and Health's Education and Research Center Grants

The National Institute for Occupational Safety and Health (NIOSH) has established centers throughout the United States for learning about occupational safety and health. The Education and Research Centers (ERCs) are in 33 universities in 14 states. The ERCs conduct training courses on occupational and environmental medicine topics (continuing medical education credits available).

Telephone: 1-800-35-NIOSH (1-800-356-4674)
Web site: www.cdc.gov/NIOSH/centers.html.

American Association of Poison Control Centers

Poison control centers were established in 1953 to help physicians and other clinicians deal with poisonings of adults and children in the United States. In 1983, the American Association of Poison Control Centers was established as the professional organization for poison control centers. The regional poison control centers can act as valuable resources in providing information about the toxicity and health effects of hazardous exposures involved in poisonings.

The local poison control center can specify the ingredients of common household products when labels do not provide adequate information.

Each certified poison control center is required to publicize its location and methods of contact. Typically, the contact telephone number can be found on the inside front cover of telephone books, where other emergency numbers are usually located.

The main emergency number across the country is 1-800-222-1222, although some states have other contact numbers as well as a number for the hearing impaired. For more information, contact the American Association of Poison Control Centers:

American Association of Poison Control Centers
3201 New Mexico Avenue, Suite 310
Washington, DC 20016
Telephone: (202) 362-7217
E-mail: aapcc@poison.org
Web site: www.aapcc.org.

For poisoning emergencies, call 1-800-222-1222. AAPCC does not manage poison exposure cases.

State and Local Health Departments

Both state and local health departments frequently have departments and staff with environmental health expertise. In some instances, a separate government agency addresses environmental concerns. In addition to providing assistance in evaluating individual patients, these organizations establish mandatory reporting requirements for selected environmental exposures or diseases related to environmental exposures. If necessary, department members can also help in obtaining assistance from federal agencies, such as the Agency for Toxic Substances and Disease Registry (ATSDR), Centers for Disease Control and Prevention (CDC), National Institute for Occupational Safety and Health (NIOSH), and Occupational Safety and Health Administration (OSHA). State health departments or the federal ATSDR both maintain inventory lists of chemicals at hazardous waste sites.

EPA and state health departments maintain the Toxic Release Inventory (TRI), which lists the amount of hazardous chemicals released by industry into the atmosphere at levels above the allowable threshold amount. These organizations can also help elucidate the history of former industrial or agricultural properties converted to residential use.

Appendix E: Lead Screening

The 1997 revised Centers for Disease Control and Prevention (CDC) guidelines are based on blood lead and housing age data (CDC 1997).

Universal screening is still the policy for communities with inadequate data on the prevalence of elevated blood lead level (BLL) and in communities with >27% of the housing built before 1950.

Targeted screening is recommended in communities where <12% of children have BLLs ≥10 micrograms per deciliter (µg/dL) or where ≥27% of houses were built before 1950. This recommendation is based on an analysis suggesting that the benefits of universal screening outweigh the cost only when the prevalence of elevated BLLs is in the range of 11% to 14% or higher.

Other candidates to be considered for *target* screening include children

- 1 to 2 years of age living in housing built before 1950 in an area not designated for universal screening (especially if the housing is not well maintained)
- of ethnic or racial minority groups who might be exposed to lead-containing folk remedies
- who have emigrated (or been adopted) from countries where lead poisoning is prevalent
- with iron deficiency
- exposed to contaminated dust or soil
- with developmental delay whose oral behaviors place them at significant risk for lead exposure
- who are victims of abuse or neglect
- whose parents are exposed to lead (vocationally, avocationally, or during home renovation)
- of low-income families who receive government assistance (Supplemental Feeding Program for Women, Infants, and children; Supplemental Security Income; welfare; Medicaid [note that blood lead screening for children on Medicaid is required by Federal law]; or subsidized child care).

According to CDC, children who receive government assistance and who live in areas where targeted screening is recommended do not require screening if they are at low risk based on the screening questionnaire (CDC 1997, section 5.2.3, p. 62) and if <12% of the children in that community have BLLs ≥10 µg/dL.

In addition to screening of children on the basis of risk questionnaires, screening for lead exposure should be considered in the differential diagnosis of children with unexplained illness such as severe anemia, seizures, lethargy, and abdominal pain.

Appendix F: Additional Information for Performing a Comprehensive Pediatric Environmental Medical Evaluation

Even though more detailed information regarding environmental history, environmental monitoring, biologic monitoring, risk communication, and risk assessment goes beyond what a primary health care provider will realistically know and do in the midst of a busy practice setting, this information is provided to help with understanding the role of others and communication with others (e.g., staff at the state or local health department, at the poison control center, at ATSDR, at the Association of Occupational and Environmental Clinics' Pediatric Environmental Health Specialty Units [PEHSUs], and experts at other organizations). It also gives a better understanding of what is involved in doing a comprehensive pediatric environmental medical evaluation.

Exposure History

The exposure history provides rough-estimate information about dose; the information rarely reflects the accurate quantitative value. Although the beliefs and concerns of the person providing the history can sometimes skew estimates of exposure, the interview usually successfully frames the boundaries of likely exposures. Reference to the scientific literature or the experience of specialists, such as industrial hygienists, might also provide reasonable estimates of dose based on monitoring done in similar situations. For example, knowing that a child lives in a house built in 1940 and that throughout the first 3 years of his life his father actively scraped, sanded, and repainted interior wood trim vividly points to potentially substantial lead exposure of the child. Similarly, if a father brings a pound of mercury home from work as a toy for his children, and it is known that this mercury spilled on a shag rug, an industrial hygienist can roughly predict the mercury air concentrations. This exposure information points the child health care provider directly to the appropriate biologic tests to measure the absorbed dose.

Environmental Monitoring

Environmental monitoring sharpens exposure estimates when the history is vague, when biologic tests of absorbed dose are not available, and when the effectiveness of environmental mitigation activities is being assessed. Monitoring allows a measurement of a contaminant's concentration in a medium (e.g., air, soil, water, or food). Some tests might serve as indicators of exposure to other agents of concern. For example, water might be tested for coliforms as an index of exposure to other pathogenic fecal contaminants. Alternatively, monitoring might focus directly on the substances of concern, such as lead concentration in paint. As with any laboratory test, the physician, with the help of those with special expertise (e.g., environmental medicine doctors and toxicologists), should be able to interpret measures of environmental contaminants with knowledge of the potential for misleading results. When doing environmental monitoring, consider the following basic considerations:

- Laboratory certification to perform the test. (**Note**: Both EPA and OSHA certify laboratories for specific contaminants. Contact the environmental division of your health department for further information.)
- Qualifications of the person performing the sampling. (Incorrect field techniques can invalidate the results.)
- Appropriateness of the test. (The child health care provider needs to have some idea of the chemical for which the test is being done [e.g., using a test to check for carbamate pesticide on a wipe sample in a house is meaningless if the pesticide used was a pyrethrin].)

- Inclusion of typical and worst-case exposure scenarios in the sampling. (A first-flush sample of tap water from lead pipes will contain higher amounts of lead than a sample taken after the tap has run for 60 seconds. Air monitoring done 1 month after a fuel oil spill in a basement will not accurately reflect the potential for exposure during the first few days after the spill.)
- Relevance of location of sample to the exposure area. (An elevated radon level in a crawl space might have little health significance as long as the radon level in the living area is low.)
- Level of concentration: average or peak. (Peak exposures to mold spores are more significant as a sensitizer than cumulative, average exposures.)

Using the monitoring results plus certain estimates (e.g., how much air children of a specific age breathe in a minute or how much soil they might ingest in a day), risk assessors in government agencies, including state health departments, can assist the child health care provider calculate dose estimates.

Biologic Monitoring

Biologic monitoring gets closest to the ultimate question of internal dose estimation: Has this patient absorbed sufficient amounts of a toxicant to cause harm? Knowledge of the metabolism, distribution, and excretion of a toxicant dictates the appropriate time and biologic fluid or tissue sample to obtain for testing. Most commonly, blood components and urine are tested. Biologic testing might do the following:

- Directly measure a toxicant such as blood lead or urine mercury.
- Measure a metabolite of a toxicant, such as urine hippuric acid, as an indicator of toluene absorption.
- Measure an effect of the interaction of a toxicant with the host's biochemistry, such as carboxyhemoglobin as a measure of carbon monoxide exposure.
- Indirectly measure the absorption of a toxicant by assessing the toxicity of a body fluid, such as testing carcinogen absorption in the urine. This is done with the Ames test, which detects compounds that are mutagenic. The test uses a strain of *Salmonella* that is auxotrophic for histidine (i.e., it cannot grow on a minimal media without added histidine).
- Measure effects on target organs, such as elevated liver enzymes in a child exposed to a hepatotoxin. [**Note**: To avoid incorrect conclusions, the physician must interpret biologic tests with attention to the toxicokinetics of the specific contaminant of concern, particularly the half-life and distribution of the toxicant into different body partitions, such as bone, fat, and blood. For example, a plasma acetylcholinesterase level reflects organophosphate exposures within the last few days, whereas a red blood cell acetylcholinesterase level might indicate exposures dating back several months.]
- Characterize the significance of exposure.

In the final phase of the evaluation, the clinician decides whether the identified environmental exposures are related to the presenting illness or are likely to cause a future health problem.

Final Steps

After a careful physical examination of the child, a conclusion is reached by answering several questions.

For the *well, but exposed child and/or worried parent:*

- Have the identified environmental exposures been associated with health effects in people? If so, how convincingly?

  How does the dose compare with known dose-response relationships?
- What are existing exposure standards? Note that occupational standards should not be considered protective of children. Whenever possible, environmental standards should be used as benchmarks.
- If standards do not exist, is there any available published information on human exposures?
- If no relevant human data exist, but animal data suggest a risk, consultation with a toxicologist or pediatric environmental medicine specialist should be obtained.
- What factors might increase or decrease the patient's susceptibility?
- What other community or home exposures might contribute to an adverse health effect?

For the *sick child:*

- How specifically can the medical problem be defined?
- Have any environmental factors been associated with the problem in others? If so, how convincingly?
- Could the identified hazards cause this problem?
- Has the dose been sufficient to result in an illness?
- Does the temporal relationship of exposure make sense?
- How does an environmental contribution to the problem compare in overall likelihood to other etiologies under consideration?

Rarely will a child health care provider have sufficient data or time, or the expertise, to conclude an environmental medical assessment with certainty. Rather, the data will usually permit only an estimate (≥0.5 or 50% chance) of the likelihood (risk) of a future illness or the probability of a causal connection to an existing disease.

Note: Hill (1965) defined five criteria that should be fulfilled to establish a causal relationship. These five criteria have been generally adopted as a test of causation. The criteria are

1. consistency of the association (i.e., different studies resulted in the same association);
2. strength of the association (i.e., size of the relative risk found increased if dose response can be established);
3. specificity of the association (i.e., measurability of the degree to which one particular exposure produces a specific disease);
4. temporal relationship of the association (i.e., exposure to the factor must have preceded development of the disease); and
5. coherence of the association (i.e., biologic plausibility).

Risk estimates can range from negligible (a lifetime excess cancer risk of 1 in 10^6) to levels of public health concern. In most forensic settings, the expression "more probable than not" (≥0.5 or 50% chance) describes the appropriate standard for decision-making. In most public policy decision-making, environmental agencies aim to

reduce risks to negligible levels. Doses relevant to risk assessments are expressed in milligrams/kilograms/day based on age/weight and physiologic differences.

In the exam room, no single probability threshold exists for recommending intervention. The more certain the hazard is as associated with an exposure, the stronger the indication for action. The clinician must consider the probability of adverse effects, as well as what interventions are available and their benefits.

On the basis of personal, social, and economic considerations, the risks that families consider acceptable vary. EPA has told the public that radon causes lung cancer, yet only a small percentage of people have measured and remediated radon problems in their homes. The clinician's responsibility is to blend knowledge of the medical significance of environmental exposure with an understanding of the other factors that families consider when deciding to take action.

For example: If a child has acrodynia clearly linked to chronic mercury contamination at home, removal of the child from the home pending remediation of the hazard is mandatory. On the other hand, the negligible risks of future illness from undisturbed, nonfriable asbestos insulative pipe covering (i.e., lagging to prevent heat loss) in a basement, compared to the high costs and difficulty of safe removal, point to the wisdom of encapsulating and/or leaving the asbestos alone.

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